

American Cancer Society Guideline for Diet and Physical Activity for Cancer Prevention

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Abstract: The American Cancer Society (ACS) publishes the Diet and Physical Activity Guideline to serve as a foundation for its communication, policy, and community strategies and, ultimately, to affect dietary and physical activity patterns among Americans. This guideline is developed by a national panel of experts in cancer research, prevention, epidemiology, public health, and policy, and they reflect the most current scientific evidence related to dietary and activity patterns and cancer risk. The ACS guideline focus on recommendations for individual choices regarding diet and physical activity patterns, but those choices occur within a community context that either facilitates or creates barriers to healthy behaviors. Therefore, this committee presents recommendations for community action to accompany the 4 recommendations for individual choices to reduce cancer risk. These recommendations for community action recognize that a supportive social and physical environment is indispensable if individuals at all levels of society are to have genuine opportunities to choose healthy behaviors. This 2020 ACS guideline is consistent with guidelines from the American Heart Association and the American Diabetes Association for the prevention of coronary heart disease and diabetes as well as for general health promotion, as defined by the 2015 to 2020 Dietary Guidelines for Americans and the 2018 Physical Activity Guidelines for Americans. *CA Cancer J Clin* 2020;0:1-27. © 2020 American Cancer Society.

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Introduction

Weight Control, Physical Activity, Diet, Alcohol, and the Cancer Burden

Cancer is the second leading cause of death, exceeded only by heart disease, in both men and women in the United States. It is the leading cause of death in many states, in Hispanic and Asian Americans, and in people aged younger than 80 years.¹ The burden of cancer extends beyond mortality. Individuals who are affected by a diagnosis of cancer experience physical suffering, distress, and diminished quality of life associated with disease-related symptoms, diagnostic procedures, cancer therapies, and long-term/late adverse effects of treatment. Moreover, quality of life can also be substantially reduced for family, caregivers, and friends of patients with cancer. Providing guidance, support, and evidenced-based strategies for individuals and populations to reduce cancer risk advances the mission of the American Cancer Society (ACS), which is to save lives, celebrate lives, and lead the fight for a world without cancer. This guideline provides specific recommendations for health care professionals, policy makers, and the general public regarding health behaviors related to maintaining a healthy body weight, being physically active, consuming a healthful diet, and avoiding or limiting alcohol intake to reduce cancer risk.

Indeed, in a recent analysis, the combination of these risk factors accounted for at least 18.2% of cancer cases and 15.8% of cancer deaths in the United States

TABLE 1. 2020 American Cancer Society Guideline on Diet and Physical Activity for Cancer Prevention

Recommendations for individuals
1. Achieve and maintain a healthy body weight throughout life. • Keep body weight within the healthy range and avoid weight gain in adult life. 2. Be physically active. • Adults should engage in 150-300 min of moderate-intensity physical activity per wk, or 75-150 min of vigorous-intensity physical activity, or an equivalent combination; achieving or exceeding the upper limit of 300 min is optimal. • Children and adolescents should engage in at least 1 hr of moderate- or vigorous-intensity activity each day. • Limit sedentary behavior, such as sitting, lying down, and watching television, and other forms of screen-based entertainment. 3. Follow a healthy eating pattern at all ages. • A healthy eating pattern includes: ◦ Foods that are high in nutrients in amounts that help achieve and maintain a healthy body weight; ◦ A variety of vegetables—dark green, red, and orange, fiber-rich legumes (beans and peas), and others; ◦ Fruits, especially whole fruits with a variety of colors; and ◦ Whole grains. • A healthy eating pattern limits or does not include: ◦ Red and processed meats; ◦ Sugar-sweetened beverages; or ◦ Highly processed foods and refined grain products. 4. It is best not to drink alcohol. • People who do choose to drink alcohol should limit their consumption to no more than 1 drink per day for women and 2 drinks per day for men. Recommendation for Community Action • Public, private, and community organizations should work collaboratively at national, state, and local levels to develop, advocate for, and implement policy and environmental changes that increase access to affordable, nutritious foods; provide safe, enjoyable, and accessible opportunities for physical activity; and limit alcohol for all individuals.

in 2014, the second highest percentages for any risk factor (after cigarette smoking) in both men and women.² These findings suggest that specific recommendations targeting these behaviors have tremendous potential to reduce the cancer burden.

Overview of the Guideline and Recommendations

Since the early 1980s, government and leading nonprofit health organizations, including the ACS and the World Cancer Research Fund/American Institute for Cancer Research (WCRF/AICR), have released cancer prevention guidelines and recommendations focused on weight management, physical activity, diet, and alcohol consumption. After the first update of the WCRF/AICR guidelines,³ the WCRF/AICR expanded their efforts and recommendations to include a Continuous Update Project, which reports comprehensively over a range of cancer types and is based

on rigorous systematic review protocols. The Third Expert Report from the WCRF/AICR, with updated cancer prevention recommendations, was released in 2018.⁴

The current ACS Diet and Physical Activity Guideline and recommendations (see Table 1) provide an update to the 2012 ACS guideline⁵ and are based largely on the WCRF/AICR systematic reviews and Continuous Update Project reports, supplemented with evidence from systematic reviews and large pooled analyses that have been published since the most recent WCRF/AICR reports. Table 2^{4,6-19} briefly summarizes the current epidemiologic evidence regarding select excess body weight, physical activity, diet, and alcohol-related exposures associated with specific types of cancer, which are described in more detail below.

Both the ACS and WCRF/AICR guidelines are based on the latest evidence, most of which is based on observational

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TABLE 2. Evidence for the Role of Weight Management, Physical Activity, and Diet for the Prevention of Cancer by Site^a

CANCER SITE	WEIGHT MANAGEMENT	PHYSICAL ACTIVITY	DIET	ALCOHOL
Breast	- Weight gain during adult life and/or excess body fatness increases risk after menopause (WCRF/AICR 2018⁴) - Weight loss may lower risk (Chlebowski 2019⁵)	Physical activity, especially moderate to vigorous, lowers risk for postmenopausal disease and also may lower risk for premenopausal disease; regular vigorous physical activity lowers risk for premenopausal disease (WCRF/AICR 2018,⁴ USDHSS 2019⁶)	- Dietary patterns rich in plant foods and low in animal products and refined carbohydrates lower risk (US Dietary Guidelines Advisory Committee 2015⁷); the Mediterranean diet pattern lowers risk (Toledo 2015⁸) - Consumption of nonstarchy vegetables and/or vegetables rich in carotenoids may lower risk for estrogen receptor–negative breast tumors (WCRF/AICR 2018⁴); diets higher in calcium/calcium-rich dairy may reduce risk (WCRF/AICR 2018⁴)	Alcohol consumption may increase risk of premenopausal breast cancer and increases risk of postmenopausal breast cancer (WCRF/AICR 2018⁴)
Colorectal	Excess body fatness increases risk (WCRF/AICR 2018⁴)	- Regular, moderate to vigorous physical activity lowers the risk of colon cancer, but not the risk of rectal cancer (WCRF/AICR 2018,⁴ USDHSS 2019⁶) - Reducing sedentary behavior may lower risk of colon cancer, but not the risk of rectal cancer. (USDHSS 2019⁶)	- A healthy eating pattern with whole grains, higher fiber, and less added sugar lowers risk (WCRF/AICR 2018,⁴ US Dietary Guidelines Advisory Committee 2015⁷); consuming nonstarchy vegetables and whole fruits probably lowers risk (WCRF/AICR 2018⁴) - Processed meat intake, even in small amounts, and red meat in moderate to high amounts, increases risk (WCRF/AICR 2018⁴) - Consuming nonstarchy vegetables and whole fruits probably lowers risk (WCRF/AICR 2018⁴) - Consume diets higher in calcium/calcium-rich dairy foods (WCRF/AICR 2018⁴); supplemental calcium may lower risk (WCRF/AICR 2018⁴) - Low circulating levels of vitamin D (<30 nmol/L) may increase risk (McCullough 2019¹⁰) - Eating a diet with low glycemic load (avoiding sweets, high-sugar/low-fiber foods, and sweetened beverages) may reduce risk (WCRF/AICR 2018⁴)	Alcohol consumption increases risk (WCRF/AICR 2018⁴)
Endometrial	Excess body fatness increases risk (WCRF/AICR 2018⁴)	- Regular, moderate to vigorous physical activity lowers risk (WCRF/AICR 2018,⁴ USDHSS 2019⁶) - Reducing sedentary time may lower risk (WCRF/AICR 2018,⁴ USDHSS 2019^{6,13})		
Gallbladder	- Weight loss may lower risk (WCRF/AICR 2018,⁴ Luo 2017¹²) - Excess body fatness may increase risk (WCRF/AICR 2018⁴) - Adult weight gain may increase risk (Campbell 2017¹⁴)			
Kidney	Excess body fatness increases risk (WCRF/AICR 2018⁴)	Regular, moderate to vigorous physical activity lowers risk (USDHSS 2019⁶)		
Liver	Excess body fatness increases risk (WCRF/AICR 2018⁴)	Regular physical activity may lower risk (WCRF/AICR 2018⁴)	Consumption of fish may lower risk (WCRF/AICR 2018⁴)	Alcohol consumption increases risk (WCRF/AICR 2018⁴)

TABLE 2. Continued

CANCER SITE	WEIGHT MANAGEMENT	PHYSICAL ACTIVITY	DIET	ALCOHOL
Lung		- Regular moderate to vigorous physical activity may lower risk (WCRF/AICR 2018,⁴ USDHSS 2019⁶) - Reducing sedentary behavior may lower risk (USDHSS 2019⁶)	- Consuming nonstarchy vegetables and whole fruits, including those high in vitamin C (especially for smokers), probably lowers risk (WCRF/AICR 2018⁴) - Processed and red meat may increase risk (WCRF/AICR 2018⁴) - High-dose β-carotene supplementation increases risk, particularly among smokers and those exposed to asbestos (WCRF/AICR 2018⁴)	
Ovary	- Excess body fatness may increase risk (WCRF/AICR 2018⁴) - Adult weight gain increases risk (Keum 2015¹⁵)	Regular moderate to vigorous physical activity may lower risk (USDHSS 2019⁶)		
Pancreas	- Excess body fatness increases risk (WCRF/AICR 2018⁴) - Adult weight gain increases risk (Genkinger 2015¹⁶)	Regular moderate to vigorous physical activity may lower risk (USDHSS 2019⁶)	- Processed and red meats as well as saturated fats in general may increase risk (WCRF/AICR 2018⁴) - Sugar-sweetened beverages may increase risk (WCRF/AICR 2018⁴)	
Prostate	Excess body fatness increases risk of advanced prostate cancer (WCRF/AICR 2018⁴)		Higher consumption of dairy products and calcium (>2000 mg/d) may increase risk (WCRF/AICR 2018,⁴ Wilson 2015¹⁷)	
Thyroid	- Excess body fatness may increase risk (Steele 2017¹⁸) - Adult weight gain increases risk (Kitahara 2016¹⁹)			
Stomach/ gastric	Excess body fat increases risk for gastric cardia cancer (WCRF/AICR 2018⁴)	Regular moderate to vigorous physical activity may lower risk (USDHSS 2019⁶)	- Regular intake of processed, grilled, or charcoaled meats increases risk for noncardia gastric cancer (WCRF/AICR 2018⁴) - Intake of nonstarchy vegetables and whole fruits, especially citrus fruits, probably lowers risk (WCRF/AICR 2018⁴)	Alcohol consumption may increase risk (WCRF/AICR 2018⁴)
Upper aerodigestive	Excess body fatness increases risk of esophageal adenocarcinoma (WCRF/AICR 2018⁴)	Regular moderate to vigorous physical activity may lower risk of esophageal adenocarcinoma (WCRF/AICR 2018,⁴ USDHSS 2019⁶)	Consumption of nonstarchy vegetables and whole fruits probably lowers risk (WCRF/AICR 2018⁴)	Alcohol consumption increases risk of oral cavity, pharynx, and larynx cancers, and squamous cell carcinoma of the esophagus (WCRF/AICR 2018⁴)

Abbreviations: BMI, body mass index; IARC, International Agency for Research on Cancer; WCIF/AICR, World Cancer Research Fund/American Institute for Cancer Research; USDHSS, US Department of Health and Human Services.

^aThis table provides a summary of the current epidemiologic evidence regarding associations of select diet- and activity-related exposures with specific types of cancer. This information illustrates where these associations and their strength of evidence vary by cancer type.

epidemiological studies, especially prospective cohort studies. Conducting and interpreting research on the diet-cancer relationship presents many challenges, including potential limitations of epidemiological and randomized control trial study designs, diet and alcohol intake estimation, and variable outcomes of intervention trials.^{20,21} Similarly, physical activity is inversely associated with risk of cancer, yet understanding the details of dose-response relationships and the critical time points in life at which benefits may be observed has been constrained by measurement challenges, confounding by obesity, and the limited number of intervention trials.²⁰

One major change in cancer prevention guidelines over time, which reflects the current and evolving scientific evidence, has been a shift from a reductionist or nutrient-centric approach to a more holistic concept of diet that is characterized as dietary patterns. A focus on dietary patterns, in contrast to individual nutrients and bioactive compounds, is more consistent with what and how people actually eat. People eat whole foods (not nutrients) that, in aggregate, represent an overall dietary pattern wherein dietary components often contribute additively or synergistically to modify cancer risk. Emerging evidence, largely epidemiological but also including a few controlled intervention trials, suggests that healthy (vs unhealthy) dietary patterns are associated with reduced risk for cancer, especially colon and breast cancer.²² Importantly, this ACS guideline and its recommendations are consistent with the WCRF/AICR guidelines, the US Department of Health and Human Services (USDHSS) Dietary Guidelines for Americans (DGA),⁷ the USDHSS Physical Activity Guidelines for Americans,²³ and the Centers for Disease Control and Prevention (CDC) cancer prevention²⁴ guidance (cdc.gov/cancer/dcpc/prevention), as well as dietary guidelines for the prevention and management of cardiovascular disease^{24–27} and diabetes.²⁸

Making healthy choices and translating guidance into feasible and consistent diet and physical activity behaviors can be a challenge for many individuals. A substantial amount of scientific evidence has accumulated to support the underlying behavioral theories and constructs and the specific strategies likely to promote healthful behavior change; a detailed review of this evidence is beyond the scope of this ACS report. It is critical to recognize that social, economic, and cultural factors, as well as policy, can influence diet and physical activity behaviors. Healthy choices are made by individuals, but these choices may be facilitated or impeded by the communities and environments in which people live. Community efforts to promote access to healthy food and resourced locations for physical activity are imperative to achieve individual adherence to the cancer prevention guidelines. Because the ACS advises policy makers and other groups that influence these community factors and efforts, relevant community and policy issues and recommendations are also addressed in this report.

Based on increased evidence since the last publication of this guideline, there are several recommendations that differ: increased emphasis on reducing the consumption of processed and red meat, in alignment with the World Health Organization's (WHO) International Agency for Research on Cancer (IARC) classification in 2015 of processed meats as a carcinogen and red meat as a probable carcinogen; increased emphasis on reducing the consumption of alcohol; and the addition of possible evidenced-based strategies to reduce barriers to healthy eating and active living and to reduce alcohol consumption.

Recommendations for Individual Choices

Overweight, Obesity, and Excess Body Fat

Recommendation: *Achieve and maintain a healthy body weight throughout life*

- Keep body weight within the healthy range, and avoid weight gain in adult life.

Excess body fat (overweight and obesity) occurs from energy imbalance as a result of excess energy intake (from both food and beverages) and low energy expenditure, although inherited genetic factors and changes in metabolism with aging also contribute to body fatness. The dietary factors most consistently associated with excess body fat include sugar-sweetened beverages, “fast-foods,” and a “Western”-type diet (ie, high in added sugars, meat, fat), whereas foods containing dietary fiber and a “Mediterranean” dietary pattern may reduce risk.⁴ In addition, aerobic physical activity, including walking, is associated with a lower risk of excess body fatness, whereas sedentary behaviors and greater screen time are associated with higher risk.⁴

Identifying more accurate approaches to measuring body composition is an important area of ongoing research, as is identifying the relative importance of fat and lean tissue in cancer prevention and control. Currently, the most accurate measures of excess body fatness include computed tomography, magnetic resonance imaging, and dual-energy x-ray absorptiometry; however, their application in large population studies and in many clinical settings is limited by high cost and logistical challenges, and thus are not typically used in clinical management. Body mass index (BMI) is a standard measurement of weight relative to height (kg/m^2) that correlates relatively well with dual-energy x-ray absorptiometry measures of body fatness among adults, with some attenuation of the correlation in older age groups.²⁹ The WHO classification for adults defines overweight as a BMI of 25.0 to 29.9 kg/m^2 and defines obesity as a BMI ≥ 30 kg/m^2 . Obesity can be categorized further into class 1 (BMI, 30.0–34.9 kg/m^2), class 2 (BMI, 35.0–39.9 kg/m^2), and class 3 (BMI, ≥ 40.0 kg/m^2).³⁰ Other easily obtained measures of

degree of body fatness include waist and hip circumferences (and the ratio of waist-to-hip).

In 1979, research based on the ACS Cancer Prevention Study-I provided robust epidemiologic evidence that excess body weight contributed to a higher risk of death from all-causes combined, coronary heart disease, diabetes, and some types of cancer.³¹ Since that time, associations of excess body fatness (assessed as BMI, waist circumference, and/or other measures) with a higher risk of being diagnosed with, or dying from, many specific types of cancer have been established. In 2000, an IARC expert panel report showed sufficient evidence that excess body fatness causes cancers of the female breast (postmenopausal), endometrium, kidney (renal cell), esophagus (adenocarcinoma), colon, and rectum.³² By 2016, this list was expanded to include cancers of the gastric cardia, liver, gallbladder, pancreas, ovary, and thyroid, as well as multiple myeloma and meningioma.³³ In addition, there is some evidence that excess body fatness probably increases the risk of advanced, high-grade, or fatal prostate cancer and cancers of the oral cavity, pharynx, and larynx.⁴ There is growing evidence from large pooled analyses and meta-analyses of prospective studies that adult weight gain also is associated with the risk of several types of cancer, including cancers of the gall bladder,¹³ thyroid,¹⁹ pancreas,¹⁶ postmenopausal ovary,¹⁵ postmenopausal endometrium,¹⁵ and postmenopausal breast,¹⁵ as well as multiple myeloma.³⁴ A recent study, using nationally representative, population-based data, reported that incidence rates increased for multiple obesity-related cancers (colorectum, corpus uteri, gallbladder, kidney, multiple myeloma, and pancreas) from 1995 to 2014 in the United States, particularly among young adults and in successively younger birth cohorts in contrast to the declining or stabilizing rates for smoking-related and HIV infection-related cancers. This finding suggests that the future burden of obesity-related cancers might be exacerbated as younger cohorts age, potentially halting or reversing the progress achieved in reducing cancer mortality over the past several decades.³⁵

Despite research from both observational epidemiologic or bariatric surgery studies suggesting that weight loss might be associated with a lower risk of some types of cancer, including postmenopausal breast and endometrial cancer, the 2016 IARC expert working group found that the evidence on weight loss and cancer risk was insufficient to evaluate.³⁴ More recent evidence from the Women's Health Initiative Observational Study supports an association between weight loss and a lower risk of breast⁹ and endometrial cancer,¹² although more research is needed to confirm this potential effect and to disentangle intentional from unintentional weight loss. Regardless, overweight and obese individuals should be encouraged

and supported to reduce their weight because of known beneficial effects of weight loss on risk of cardiovascular disease³⁶ and diabetes,³⁷ which has also been linked to numerous types of cancer.³⁸

Excess adiposity can contribute to a procarcinogenic environment through several carcinogenic pathways involved in inflammation, oxidative stress, cell proliferation and angiogenesis, inhibition of apoptosis/cell death, and metastases.³⁹ There is increasing research showing that the gut microbiome and secondary metabolites might play an important role in many obesity-related carcinogenic pathways.⁴⁰ Notably, emerging evidence suggests that metabolic dysregulation is highly correlated with central obesity, and may play a critical role in the risk of obesity-related cancers. In addition, results from large lifestyle and behavioral intervention studies have demonstrated that even modest weight loss improves insulin sensitivity and biochemical measures of hormone metabolism,⁴¹ which also play roles in cancer etiology.

The obesity epidemic is now well-recognized, and in 2015 and 2016, nearly 40% of American adults had obesity, with a slightly higher prevalence among women (41.1%) than men (37.9%).⁴² The prevalence of obesity varies considerably among racial/ethnic groups, being lowest among non-Hispanic Asian adults (12.7%), followed by non-Hispanic white (37.9%), Hispanic (46.8%), and non-Hispanic black (47.0%) adults.⁴² Moreover, in 2015 and 2016, 20.6% of adolescents aged 12 to 19 years, 18.4% of children aged 6 to 11 years, and 13.9% of children aged 2 to 5 years had obesity.⁴²

Approximately 10.9% of cancer cases diagnosed in the United States during 2014 among women and 4.8% of cancer cases among men were attributed to overweight or obesity; only cigarette smoking accounts for a higher percentage of cancer cases.² For some types of cancer, the fraction of cancer cases attributable to excess body fatness is extremely high; 60.3% of corpus uterine cancers and >30% of gallbladder, liver, and kidney/renal pelvis cancers and esophageal adenocarcinomas were attributed to obesity. Clearly, excess body fatness contributes substantially to cancer risk; however, the full impact of the obesity epidemic on the cancer burden, including the long-term effect of obesity that begins as early as in childhood, is not well understood.

Physical Activity

Recommendation: Be physically active

- Adults should engage in 150 to 300 minutes of moderate-intensity physical activity per week, or 75 to 150 minutes of vigorous-intensity physical activity, or an equivalent combination; achieving or exceeding the upper limit of 300 minutes is optimal.

- Children and adolescents should engage in at least 1 hour of moderate- or vigorous-intensity activity each day.
- Limit sedentary behaviors such as sitting, lying down, and watching television and other forms of screen-based entertainment.

The USDHSS Physical Activity Guidelines for Americans (2018) recommend that adults should “move more and sit less” because some activity is better than none. Specifically, adults should engage in 150 to 300 minutes of moderate-intensity aerobic activity or 75 to 150 minutes of vigorous-intensity activity, or an equivalent combination, each week and some muscle-strengthening activity at least 2 days each week.¹² Although muscle-strengthening activity is recommended for overall health, there is a paucity of evidence for this type of activity in relation to cancer; thus, the focus for cancer prevention guidance is largely on aerobic moderate-to-vigorous physical activity (MVPA). Despite the vast health benefits of engaging in regular MVPA, in 2018, nearly one-half of US adults (46.7%) did not meet the recommended amount of MVPA.⁴³

Children and adolescents should engage in at least 1 hour of moderate-intensity or vigorous-intensity activity each day that consists of daily aerobic physical activity, muscle-strengthening (at least 3 days a week) activities, and bone-strengthening activities (at least 3 days a week). Approximately one-fourth of 9th-grade to 12th-grade students met this recommendation for daily aerobic MVPA during 2017, and one-half met the recommendation for muscle-strengthening activity on 3 or more days.⁴⁴ Although the relationship is not as strong as for adults, it may be important for youth to instill physical activity as a daily behavior in their lives at an early age in order to help maintain activity as a lifestyle behavior in later age. Doing so may be one part of the equation contributing to maintaining weight and preventing weight gain with increasing age in later adulthood.

In 2018, both the WCRF/AICR and Physical Activity Guidelines Advisory Committee (PAGAC) reports concluded that there was sufficient and robust evidence establishing a link between physical activity and a lower risk of colon cancer. Beyond colon cancer, the strength of the evidence and/or the cancer types linked to physical inactivity is less consistent. The PAGAC concluded that there was strong evidence for 6 additional cancer types, including breast, kidney, endometrial, bladder, esophageal (adenocarcinoma), and stomach (cardia) cancers. The conclusion was that evidence for lung cancer was moderate and that evidence for hematologic, head and neck, pancreas, prostate, and ovarian cancers was limited.¹² In contrast, the WCRF/AICR⁴ concluded that the evidence was strong and probable for postmenopausal breast and endometrial cancers, respectively, and was limited but suggestive for esophageal (adenocarcinoma),

liver, premenopausal breast, and lung cancers. Although there is some disagreement regarding the strength of the evidence, it is clear that evidence is rapidly accumulating and supports an important role for MVPA in cancer prevention for a greater number of cancers than previously believed.

It is estimated that 1.5% of all cancers diagnosed in the United States during 2014 in men and 4.4% of all cancers diagnosed in women are attributable to physical inactivity, as are 1.4% of all cancer deaths in men and 3.0% of all cancer deaths in women.² These attributable fractions are based on earlier strong evidence linking physical inactivity with a higher risk of colon, female breast, and endometrial cancers.² However, recent consensus reports, including those from the WCRF/AICR⁴ and the Physical Activity Guidelines for Americans,¹² provide support for the role of physical activity in the prevention of many additional types of cancer, suggesting that the preventable fraction may be even larger.

Sedentary time has more recently been investigated as a behavior distinctly different from physical inactivity. With technological advancements, the amount of time spent sitting has increased significantly over the past few decades, and it has been estimated that more than one-half (53%) of nonoccupational time is spent on screen time (eg, computer, telephones, television).⁴⁵ During 2015 through 2016, approximately 60% of US children, adolescents, and adults spent at least 2 hours per day watching television, and approximately 50% reported at least an hour of computer use outside of school or work.⁴⁶ Prolonged sitting time has been associated with premature mortality, type 2 diabetes,⁴⁷ and cardiovascular disease,⁴⁸ and evidence is accumulating to support a role, separate from physical inactivity, in relation to cancer. The PAGAC concluded that there was moderate evidence linking prolonged sitting time with a higher risk of colon, endometrial, and lung cancers, whereas the WCRF/AICR concluded that there was limited, but suggestive, evidence only for endometrial cancer. Thus, there is a need for more research to address this emerging risk factor for cancer, but the early evidence suggests that reducing sitting time may be important for cancer prevention.

The role of physical activity in cancer prevention is supported by accumulating biological evidence. Physical activity has been shown to affect various systemic functions that would purportedly lower the risk of specific types of cancer, including its effects on insulin/glucose metabolism, immune function, inflammation, sex hormones, oxidative stress, genomic instability, and myokines.^{49–51} For example, physical activity has been associated with lower sex hormone levels in postmenopausal women,^{51,52} which can help explain the association between physical activity and a lower risk of postmenopausal breast cancer. Physical activity also aids in preventing weight gain and has been

associated with a lower risk of obesity; consequently, some of the benefit for cancer prevention may be mediated through the beneficial influence of physical activity on body weight.^{12,53} The biological mechanisms underlying the association between prolonged sitting time and cancer risk have not been studied extensively. However, studies are beginning to emerge to demonstrate that sitting time, independent of physical inactivity, affects several hormones and metabolic pathways.⁵²

Drawing clear conclusions regarding the dose and intensity of activity required for cancer risk reduction is challenging. The evidence supports that greater levels of physical activity may be required for cancer prevention than for the prevention of cardiovascular disease or type 2 diabetes. Studies broadly support the notion that there is a linear relationship between physical activity and cancer prevention (ie, the more MVPA in which one engages, the greater the cancer prevention benefits).⁴ Thus, although the PAGAC recommends that adults achieve 150 to 300 minutes of moderate-intensity physical activity per week (or 75-150 minutes of vigorous-intensity physical activity), or an equivalent combination of both, exceeding the upper limit of 300 minutes may be more optimal for cancer prevention. The evidence regarding whether breaks in prolonged bouts of sedentary time or an overall reduction in sedentary time might modify cancer risk is an important area of study, but evidence is too limited to draw clear conclusions at this time. The evidence from both physical activity and sitting time studies supports that the greatest risk reduction is consistently observed when an individual transitions from engaging in no MVPA to any amount; therefore, it is important to reinforce the message to “move more and sit less.”

Diet and Dietary Patterns

Recommendation: Follow a healthy eating pattern at all ages

- A healthy eating pattern includes:
 - Foods that are high in nutrients in amounts that help achieve and maintain a healthy body weight;
 - A variety of vegetables—dark green, red and orange, fiber-rich legumes (beans and peas), and others;
 - Fruits, especially whole fruits with a variety of colors; and
 - Whole grains.
- A healthy eating pattern limits or does not include:
 - Red and processed meats;
 - Sugar-sweetened beverages; or
 - Highly processed foods and refined grain products.

Diet and nutrition are important determinants of cancer risk, both through their contributions to energy balance and via biological mechanisms that alter risk independent

of body weight.⁷ Recent estimates attribute 4.2%-5.2%⁵⁴ of cancer cases per year directly to poor diet.² Investigating the role of diet in cancer prevention is challenging, because consumption patterns of humans are highly complex, the food supply is constantly changing, and relevant exposure periods are not always known. Moreover, the methods to measure long-term, usual diet in free-living populations necessarily contain some degree of error.⁵⁵ Randomized controlled trials (RCTs) of dietary interventions aimed at preventing cancer, conversely, are expensive and largely impractical. Therefore, most current evidence concerning diet and cancer prevention is derived from observational epidemiologic studies, in particular prospective cohort studies, mechanistic studies of food components in laboratory animals and cell culture, and RCTs when available.

Dietary patterns as a modern and more appropriate focus

Because of accumulating evidence on healthy dietary patterns in relation to chronic disease risk reduction, an emphasis on dietary patterns is now highlighted in the 2015 to 2020 US DGA.⁵⁶ This is particularly relevant because, although the associations of individual nutrients and foods with cancer may be small, additive and interactive effects could be important.⁴ Several comprehensive reviews support recommendations to follow healthy dietary patterns. The 2015 Dietary Guidelines Scientific Report concluded that there is moderate evidence that dietary patterns rich in plant foods and lower in animal products and refined carbohydrates are associated with a lower risk of postmenopausal breast cancer, and plant-based patterns low in red and processed meat and added sugars are associated with a lower risk of colorectal cancer.⁷ In addition, the WCRF/AICR concluded that a Mediterranean diet is “convincingly” associated with a lower risk of weight gain, overweight, or obesity, whereas a “Western”-type dietary pattern is “probably” associated with an increased risk of these outcomes.⁴ The Diet Patterns Methods Project,⁵⁷ a multicenter study of dietary patterns and cause-specific mortality, reported an 8% to 17% lower risk of cancer mortality among women and a 17% to 24% lower risk among men whose diets were most (vs least) concordant with 4 healthy dietary pattern scores. The dietary patterns examined included the Mediterranean Diet,^{58,59} the Dietary Approaches to Stop Hypertension diet,^{60,61} the US Department of Agriculture (USDA) Healthy Eating Index,⁶² and the Harvard Alternate Healthy Eating Index.⁶³ Although these and other healthful dietary patterns have unique features, they share a foundation of mostly plant foods (including nonstarchy vegetables, whole fruits, whole grains, legumes, and nuts/seeds) and healthy protein sources (higher in legumes and/or fish and/or poultry, and lower in processed meats and red meat), and include unsaturated fats (eg, monosaturated and/or polyunsaturated fat); these

patterns are also lower in added sugar, saturated and/or trans fats, and excess calories. These healthy dietary pattern scores have also been associated with a lower risk of colorectal cancer^{22,64} and total cancer incidence^{65,66} in meta-analyses of observational studies. Two randomized clinical trials found lower overall cancer or breast cancer⁸ risk among those randomized to follow the Mediterranean diet. Thus, these studies provide consistent and compelling evidence that healthy dietary patterns are associated with a decreased risk of cancer, all-cause mortality, and other chronic disease endpoints.

These healthy dietary patterns are associated not only with improved health but also with a lower environmental impact, such as reduced greenhouse gas emissions and energy, land, and water use compared with the average US diet.⁶⁷ Environmentally sustainable diets emphasize fruits and vegetables, whole grains, plant-sourced protein foods, unsaturated plant oils, and more limited (optional) quantities of animal-based protein foods, dairy products, and sugar.⁶⁸ Attention to complex issues of food production practices and distribution will be needed to identify approaches to further reduce the carbon footprint and other environmental impacts of dietary sources. Regardless, achieving recommended dietary patterns, such as those recommended herein, may lead to improved food security and environmental sustainability for future generations.

Healthy dietary patterns may reduce the risk of cancer and other diseases through multiple mechanisms. For example, plant-based diets are associated with lower levels of inflammation, improved insulin response, and less oxidative DNA damage.⁶⁹ Plant-based diets are also associated with higher concentrations of beneficial gut bacteria compared with mostly animal-based diets high in saturated fat and sugar.^{70,71} Additional research on the relationship of dietary factors with these metabolic and microbial biomarkers and with health outcomes will continue to help in elucidating the role that diet plays in carcinogenesis.

Vegetables and fruit

Several food and nutrient components of healthy dietary patterns are also independently associated with cancer risk. Although the relationship between vegetables and fruit intake with lowering cancer risk is weaker than previously believed, the 2018 WCRF/AICR report concluded that consuming nonstarchy vegetables and/or whole fruit “probably” protects against several aerodigestive cancers, including mouth, pharynx, larynx, nasopharynx, esophagus, lung, stomach and colorectal cancers.⁴ Promising research on molecularly defined tumor subtypes has shown that carotenoid-rich vegetables and fruit, and biomarkers of their consumption, are associated with a lower risk of more aggressive breast tumors, including estrogen receptor–negative breast tumors.^{72,73}

Vegetables (including beans) and fruits are complex foods, each containing numerous vitamins, minerals, fiber, carotenoids, flavonoids, and other bioactive substances, such as sterols, indoles, and phenols, that may help prevent cancer.⁴ There is ongoing research on the potential cancer chemopreventive properties of particular vegetables and fruits, or groups of these, including dark-green and orange vegetables, cruciferous vegetables (eg, cabbage, broccoli, cauliflower, brussels sprouts), soy products, legumes, allium vegetables (onions and garlic), and tomato products. Vegetables and fruits may also indirectly influence cancer risk through effects on energy intake or body weight.⁷⁴ Many vegetables and fruits are low in energy, high in fiber, and have a high water content, which may increase satiety and decrease overall energy intake,⁷⁴ and thus should contribute to weight loss and maintenance of that loss.

Vegetable and fruit consumption has also been associated with a reduced risk of other chronic diseases, particularly cardiovascular disease, an important contributor to overall morbidity and mortality in the United States.^{60,75–79} For cancer risk reduction, the ACS advises consistency with the DGA, which recommends consuming at least 2.5 to 3 cups of vegetables and 1.5 to 2 cups of fruit each day, depending on energy requirements.

Legumes are rich in protein, dietary fiber, iron, zinc, potassium, and folate, are low in saturated fat, and have a low glycemic index. This makes legumes a generally healthy addition to the diet, and good alternatives for those looking to reduce their consumption of red and processed meats. Legumes also are gluten-free, making them appropriate for people with celiac disease or gluten sensitivity.⁵⁶ Legumes include kidney beans, pinto beans, black beans, white beans, garbanzo beans (chickpeas), lima beans (mature, dried), lentils, edamame (green soybeans) and other soy foods.

Whole grains

The evidence that whole grains, in which 100% of the original kernel is retained, lower colorectal cancer risk is considered “probable” by the WCRF/AICR.⁴ Each 30 g per day consumption of whole grains was estimated to lower the risk of colorectal cancer by 5%.⁸⁰ In a separate meta-analysis, total cancer mortality risk was 6% lower with each 3 servings of whole grains daily.⁸¹ Rich in phytochemicals and dietary fiber, whole grains may lower colorectal cancer risk through modification of fatty acid production, lowered levels of proinflammatory bacterial species,⁷⁰ and by accelerating gut transit time, thus reducing duration of exposure of the gut to carcinogens. In addition, the WCRF/AICR considers the evidence “probable” that whole grains and foods high in dietary fiber are associated with lower risk of weight gain, overweight, or obesity.⁴ The 2015 DGA recommends consuming at least one-half of grains as whole grains⁵⁶ based on “moderate”

evidence that dietary patterns rich in whole grains are associated with lower BMI, waist circumference, percentage body fat, and/or obesity.⁷ The ACS guideline recommendation to choose whole grains is consistent with these guidelines.

Dietary fiber

Dietary fiber, which is found in plant foods, including legumes, whole grains, fruits and vegetables, and nuts and seeds, is considered “probably” associated with a lower risk of colorectal cancer as well as a lower likelihood of weight gain, overweight, and obesity.⁴ Dietary fiber has potent effects on bacterial species in the gut⁸²; and the relationship between gut microbial dysbiosis, body weight, and cancer risk is an active area of investigation.⁸³ In RCTs of fiber supplements, including isphaghula husk (psyllium fiber) and wheat bran fiber, the supplements did not reduce the risk of recurrent adenomatous polyps.^{84,85} Thus, the ACS recommendation is to obtain most dietary fiber from whole plant foods, such as vegetables, fruits, whole grains, nuts, and seeds.

Red and processed meats

Red meat refers to unprocessed mammalian muscle meat—for example, beef, veal, pork, lamb, mutton, horse, or goat meat—including minced or frozen meat, whereas processed meat is meat that has been transformed through curing, smoking, salting, fermentation, or other processes to improve preservation or enhance flavor, such as bacon, sausage, ham, bologna, hot dogs, and deli meats.⁸⁶ Most processed meats contain pork or beef but may also contain other red meats, poultry, or meat byproducts.

Evidence that red and processed meat increases cancer risk has existed for decades, and health organizations recommend limiting or avoiding consumption of these foods.^{4,5} The 2015 DGA noted moderate evidence that eating patterns lower in red and processed meats were associated with lower risk of obesity, type 2 diabetes, and some types of cancer in adults.⁵⁶ In 2015, the IARC expert panel concluded that processed meat is a group I carcinogen and red meat a “probable” (group 2A) human carcinogen based on evidence for increased risks of colorectal cancer in addition to evidence of biologically plausible mechanisms.^{86,87} The most recent WCRF/AICR report⁴ concluded that processed meat is “convincingly” related to colorectal cancer and that red meat “probably” increases colorectal cancer risk. Recent studies suggest a possible role of red and/or processed meats in increasing the risk of breast cancer^{88,89} and certain forms of prostate cancer,⁹⁰ although more research is needed.

In contrast with these systematic reviews and guidelines, a 2019 review of prospective cohort studies considered the effects of red and processed meat intake on cancer mortality and incidence to be small, with certainty of evidence that is

“low to very low certainty” based on review criteria that prioritized evidence from RCTs while downgrading evidence from observational studies. Therefore, the authors recommended that individuals continue current meat intake.⁹¹ However, the results of this group’s meta-analyses found significant reductions in risk of cancer death with lower intake of red and processed meat as well as a lower risk of prostate cancer death and of incident colorectal, esophageal, and breast cancers with a reduction in processed meat intake,⁹² entirely consistent with the systematic evidence reviews from the WCRF/AICR⁴ and other groups. Although imperfect, prospective cohort studies provide consistent evidence that individuals who consume higher amounts of red meat, and especially processed meat, are at higher risk of colorectal cancer.⁴ An RCT of red or processed meat and cancer outcomes is unlikely to take place for practical and ethical reasons. Even so, the authors point to the Women’s Health Initiative dietary modification trial as evidence that does not support an association between decreased red meat intake and reduce risk of cancer, although that trial was focused on decreasing total fat intake and not on reducing red meat intake.⁹³ The best available evidence continues to support recommendations to limit intake of red and processed meats for cancer prevention.

Potential biologic mechanisms underlying these associations include consumption of nitrates and nitrites in processed meats, with oxidative DNA damage from the formation of nitrosamines in the gut catalyzed by heme iron⁹⁴ and the formation of heterocyclic aromatic amines and polycyclic aromatic hydrocarbons during high-heat cooking of meat,^{95,96} such as cooking meat over flames or grilling. It is not known whether there is a safe level of consumption for either class of meat products, since the risk of colon cancer increases 23% with each additional serving (almost 2 ounces) of processed meat and 22% per 3 ounces serving of red meat.⁴ In the absence of such knowledge, while recognizing that the magnitude of increased risk has some uncertainty, the ACS recommends choosing protein foods such as fish, poultry, and beans more so than red (unprocessed) meat, and, for individuals who consume processed meat products, to do so sparingly, if at all.

Added sugars

White (processed) sugar, raw and brown sugar, corn sweetener, high-fructose corn syrup, and other added sugars in sugar-sweetened beverages and energy-dense foods (eg, traditional “fast food” or heavily processed foods) are associated with risk of weight gain, overweight, or obesity,⁴ which itself is considered a cause of 13 types of cancers.⁹⁷ In addition, the WCRF/AICR notes that diets with high “glycemic load”—reflecting their blood sugar-raising potential—are probably associated with higher endometrial cancer risk.⁴ Energy-dense and highly processed foods are often higher

in caloric sweeteners, refined grains, saturated fat, and sodium.⁵⁶ The 2015 DGA recommends limiting calories from added sugars and saturated fat⁵⁶ and specifically consuming <10% of energy per day from added sugars. Likewise, global health organizations note that limiting sugar-sweetened beverages should be a high priority,⁴ and recommend instead choosing water and unsweetened beverages.

Processed foods

The health impact of highly processed foods has become an area of heightened public health interest. Some types of processing, such as peeling, cutting, and freezing fresh vegetables and fruit for later consumption, have important health benefits that increase the safety, convenience, and palatability of foods. It is useful to consider the spectrum of food processing, from less processed foods such as whole grain flour and pasta to highly processed foods that include industrially produced grain-based desserts, ready-to-eat or ready-to-heat foods, snack foods, sugar-sweetened beverages, candy, and other highly palatable foods that often do not resemble their original plant or animal sources.⁹⁸ Highly processed foods tend to be higher in fat, added sugars, refined grains, and/or sodium and have been associated with adverse health outcomes, including cancer, in a small number of studies.⁹⁹ It is notable that up to 60% of energy consumed per day in US households is from highly processed foods and beverages.⁹⁸ The 2018 WCRF/AICR report recommends limiting consumption of “fast foods” and other processed foods high in saturated fat, starches, or added sugars⁴ because of their association with body weight.

Calcium, vitamin D, and dairy products

In addition to dietary patterns and foods, certain nutrients may modify cancer risk. The WCRF/AICR considers the evidence “probable” that diets high in calcium and dairy products lower colorectal cancer risk.⁴ The evidence that diets high in calcium may lower breast cancer risk is considered “limited/suggestive.” Also “limited/suggestive” according to the WCRF/AICR is evidence that calcium and dairy products increase prostate cancer risk.⁴ For each 400 grams of dairy intake (equivalent to almost 2 cups of milk per day), prostate cancer risk was 11% higher,¹⁰⁰ and a long-term diet that included higher doses of calcium (>2000 mg calcium) was associated with a greater risk of prostate cancer, including lethal, advanced, and high-grade cancers.¹⁷ The Recommended Dietary Allowance for calcium for adults ranges from 1000 to 1200 mg daily.¹⁰¹ Because the intake of dairy foods may decrease the risk of some cancers and possibly increase the risk of others, the ACS does not make specific recommendations regarding dairy food consumption for cancer prevention.

Vitamin D, which is synthesized in the skin with exposure to ultraviolet radiation, is recognized for its role in

maintaining bone health.¹⁰¹ Dietary sources include a few foods (eg, fatty fish, some mushrooms) in which this vitamin is found naturally, as well as fortified foods (milk, some orange juice and cereals) and supplements. Laboratory and observational studies indicate a potential role of vitamin D in the prevention of cancer.¹⁰² To date, the most consistent evidence for a cancer risk-lowering effect of vitamin D is for colorectal cancer.¹⁰ However, evidence from RCTs for the prevention of colorectal adenomas¹⁰³ or cancer¹⁰⁴ have not supported an association. The Vitamin D and Omega-3 Trial (VITAL) supplement trial¹⁰⁵ of 2000 IU of vitamin D per day found no association of vitamin D supplementation with all incident cancers combined; however, the trial reported overall lower cancer mortality from vitamin D supplementation. No association was seen for colorectal cancer specifically, but the study was not powered to test colorectal cancer outcomes. The study reported no adverse events with taking 2000 IU daily over the 6-year trial.¹⁰⁵ Based on current evidence, the US Preventive Services Task Force does not recommend widespread screening of vitamin D levels. However, most Americans have inadequate vitamin D intake, and, despite recent improvements, >25% of US teens and adults have insufficient (<50 nmol/L) vitamin D blood concentrations.¹⁰⁶ Although the role of vitamin D in cancer prevention remains an area of research interest and debate, avoiding deficient levels is recommended. People at higher risk of vitamin D insufficiency include individuals with dark skin, those living in Northern latitudes, and those who stay indoors and who do not consume sources of vitamin D.

Dietary supplements

Dietary supplements are a heterogeneous group of products defined under current US laws and regulations as containing vitamins and minerals as well as amino acids, herbs/botanicals, and other kinds of ingredients. Vitamin and/or mineral supplements are truly “dietary” because they contain micronutrients that are also present in foods. They are also “supplemental” because they have important health benefits for people whose intake of these micronutrients from foods is not sufficient or for those with malabsorption disorders. In contrast, many other products that are marketed as dietary supplements are not truly “dietary” because many come from sources other than foods and contain substances not found in foods, and they are not “supplemental” because they do not increase intake of micronutrients that have been scientifically shown to be important for human health. Furthermore, current laws and regulations do not guarantee that products sold as dietary supplements actually contain substances in the quantities claimed on their labels or that they are free from undeclared substances that can be harmful to human health.

For reasons other than cancer prevention, some vitamin and/or mineral supplements may be beneficial for

some people to prevent nutrient deficiency, such as in pregnant women, women of childbearing age, and people with restricted dietary intakes. Dietary supplementation may also be indicated to correct a documented clinical deficiency or insufficiency, such as supplementation with vitamin D in those with low circulating concentrations or vitamin B12 supplementation in those with vitamin B12-associated anemias.

Although a diet rich in vegetables, fruits, and other plant-based foods may reduce the risk of cancer, there is limited and inconsistent evidence that dietary supplements can reduce cancer risk.⁴ Whereas 2 RCTs showed reductions in cancer risk among men taking low-dose antioxidants or low-dose multiple micronutrients, evidence for women is lacking.^{107,108} Furthermore, evidence exists that some high-dose supplements containing nutrients such as β -carotene and vitamins A and E can increase the risk of some cancers.⁴ For individual nutrients, an exception may be calcium, in which supplemental calcium may reduce the risk of colorectal cancer. However, people who have excessive calcium intake (mostly from supplements) may have a higher risk of death from all cancer types combined compared with those who have a recommended level of dietary calcium.¹⁰⁹ The same study also reported no overall benefit to longevity from all dietary supplements considered together.¹⁰⁹ Nonetheless, more than one-half of US adults use one or more dietary supplement(s).¹¹⁰

Many healthful compounds are found in vegetables and fruits, and it is likely that these compounds work synergistically to exert their beneficial effect. There are likely to be important, but as yet unidentified, components of whole food that are not included in dietary supplements. Some supplements are described as containing the nutritional equivalent of vegetables and fruits. However, the small amount of dried powder in such pills frequently contains only a small fraction of the levels contained in the whole foods, and there is a lack of evidence supporting a role of these products in cancer prevention. Food is the best source of vitamins, minerals, and other bioactive food components. If a dietary supplement is used for general health purposes, the best choice is a balanced multivitamin/mineral supplement containing no more than 100% of the “daily value” of nutrients, and the ACS does not recommend the use of dietary supplements for cancer prevention, consistent with WCRF/AICR guidelines.⁴

Alcohol Consumption

Recommendation: It is best not to drink alcohol

- People who do choose to drink alcohol should limit their consumption to no more than 1 drink per day for women and 2 drinks per day for men.

Alcohol consumption is the third major modifiable cancer risk factor after tobacco use and excess body weight.² A standard drink of alcohol is defined as 12 ounces of beer, 5 ounces of wine, or 1.5 ounces of 80-proof distilled spirits, which contain approximately 14 grams of ethanol, the primary form of alcohol found in alcoholic beverages.

Alcohol consumption is an established cause of at least 7 types of cancer. In 1987, an expert working group convened by the IARC first classified the consumption of alcoholic beverages as carcinogenic to humans.¹¹¹ The evidence for causality was found to be sufficient for cancers of the upper aerodigestive tract (UADT) (ie, oral cavity, pharynx, larynx, squamous cell carcinoma of the esophagus) and liver. A second IARC expert working group convened in 2007 confirmed that alcohol consumption causes UADT and liver cancer, and they also found that there was sufficient evidence of causality for colorectal and female breast cancers. This second working group also found for the first time that “ethanol in alcoholic beverages” is carcinogenic to humans¹¹²; thus alcoholic beverages of all types increase risk. A 2009 IARC working group reaffirmed the previous conclusions, and added that both ethanol and acetaldehyde—the primary metabolite of ethanol ingestion associated with the consumption of alcoholic beverages—are a cause of cancers of the UADT.¹¹³ More recently, a 2018 WCRF/AICR Continuous Update Project report reaffirmed the strong evidence for those cancers (reported previously by other agencies) and also found that alcohol consumption probably increases the risk of stomach cancer.⁴ Importantly, alcohol consumption also interacts synergistically with tobacco use to increase the risk of cancers of the UADT considerably more than the risk associated with either drinking alcohol or tobacco use alone.¹¹² Of particular relevance for cancer prevention guidelines is evidence showing that consumption of any amount of alcohol increases risk of some types of cancer, most notably breast cancer.⁴

Broadly, the carcinogenic effects of ethanol found in alcoholic beverages and acetaldehyde involve DNA and protein damage and alterations, oxidative stress, inhibition of DNA repair and cell death, increased cell proliferation, nutritional malabsorption, changes in DNA methylation, and, for breast cancer, increased estrogen levels.^{114,115} In addition, carcinogenic contaminants can be introduced during alcoholic beverage production.

In 2016, approximately 50.7% of the US population aged ≥ 12 years reported current (ie, in the past 30 days) alcohol consumption, approximately 6% were heavy alcohol drinkers (ie, drank ≥ 5 alcoholic beverages on the same occasion on ≥ 5 days in the past 30 days), and approximately 24.2% of the population were binge drinkers (ie, drank at least ≥ 5 alcoholic beverages on the same occasion on at least 1 day

in the past 30 days).¹¹⁶ There are complex disparities in the prevalence of alcohol consumption. For both men and women, the prevalence of alcohol abstinence is higher among Hispanics, African Americans, Asians, and Native Americans than among non-Hispanic whites; however, among current drinkers, the prevalence of heavy weekly drinking is highest among Native Americans, and the prevalence of heavy daily drinking is highest among Hispanic men.¹¹⁷

It was recently estimated that in 2014, alcoholic beverage consumption caused 5.6% of all incident cancer cases and 4% of all cancer deaths among males and females in the United States.² An estimated 40.9% of oral cavity/pharynx cancers, 23.2% of larynx cancers, 21.6% of liver cancers, 21% of esophageal cancers, and 12.8% of colorectal cancers in 2014 were attributed to alcohol consumption and, among women, 16.4% (ie, 39,060) of all breast cancers were attributable to alcohol consumption.²

Despite the fact that a substantial number of cancer cases are attributed to alcohol consumption in the United States, and that reducing alcoholic beverage consumption is one of the WHO Best Buys for reducing noncommunicable diseases,¹¹⁸ public awareness about the carcinogenicity of alcohol, and its primary metabolite acetaldehyde, is low. Furthermore, fewer than one-half of the CDC-funded comprehensive cancer control plans specify goals, objectives, or strategies for alcohol control.¹¹⁹ Finally, alcohol control has benefits beyond those for cancer, and recently a report from the Global Burden of Disease Study found that “consuming zero standard drinks daily minimizes the overall risk to health.”¹²⁰

Recommendation for Community Action

- Public, private, and community organizations should work collaboratively at national, state, and local levels to develop, advocate for, and implement policy and environmental changes that increase access to affordable, nutritious foods; provide safe, enjoyable, and accessible opportunities for physical activity; and limit access to alcoholic beverages for all individuals.

Social, economic, and cultural factors strongly influence an individual's body weight, physical activity, dietary patterns, and alcohol intake. Limited access to and affordability of healthy foods and the widespread availability and extensive marketing of high-calorie foods and beverages of low nutritional value, as well as barriers to the ability of individuals to be physically active for recreation and transportation in communities have all been implicated as contributors to the obesity trend in the United States.¹²¹ Therefore, the ability of an individual to avoid many unhealthy lifestyle factors, including those related to food and beverage intake and physical inactivity, is often influenced by factors outside of his or her direct control.

The factors contributing to trends in excess body weight specifically are complex and multifaceted, and reversing these trends will require a broad range of innovative, coordinated, and multilevel strategies that engage a variety of stakeholders; involve multiple systems and sectors (food and agriculture, transportation, urban planning, childcare centers, schools, employers, health care, and more); and emphasize policy, system, and environmental changes.¹¹³ Therefore this guideline addresses the importance of public, private, and community organizations working collaboratively at national, state, and local levels to develop, advocate for, and implement policy, system, and environmental changes to reduce obesogenic environments and promote access to affordable, nutritious foods and provide safe, enjoyable, and accessible opportunities for physical activity for all individuals.

Although most Americans face obstacles to engaging in health-promoting behaviors, these challenges are often compounded for lower income individuals, racial and ethnic minority groups, persons with disabilities, and those residing in rural communities, who frequently face additional barriers to the adoption of cancer-preventive behaviors.¹²² Importantly, these barriers contribute in part to the greater health disparities documented among certain populations.¹²³ For instance, access to supermarkets has been associated with improved diet quality, increased consumption of fruits and vegetables, and a lower prevalence of obesity.¹²⁴ Communities with a greater proportion of ethnic minorities and residents with low socioeconomic status are often also identified as low-income and low-access areas, characterized by fewer supermarkets with healthy, affordable, high-quality foods. In these areas, residents may not have the economic resources to purchase adequate and nutritious food to feed themselves and their families. Frequently, a plethora of “fast-food” restaurants and convenience stores also is readily available in these communities.^{125,126} Thus residents with limited access to neighborhood supermarkets often purchase foods at local convenience stores, where fewer perishables, such as fresh produce, and more highly processed convenience items are readily available. Even in neighborhoods where supermarkets are readily available, low-income residents may continue to purchase less expensive, energy-dense foods; studies have suggested that foods of lower diet quality make up a greater proportion of the dietary patterns of lower income individuals compared with individuals who have higher incomes.^{127–129} The neighborhoods where people live can also affect their alcohol consumption. This is especially true in neighborhoods in which convenience and liquor stores are overconcentrated^{128,129} and where alcohol is heavily promoted by commercial interests^{130,131}

Disparities in the built environment also affect physical activity patterns. Safe and inviting access to parks, playgrounds, schools, sidewalks and trails, bicycle paths, and

workout facilities and gyms as well as the availability of public transit all provide additional opportunities for daily physical activity. Sidewalks and bicycle paths within close proximity and connected to residential areas with common or everyday destinations, retail stores, jobs, schools, child-care, and recreation centers also promote more physically active lifestyles.¹³²⁻¹³⁴ Yet significantly fewer sports areas, parks, greenways, well-maintained sidewalks, and bike paths are available in areas of poverty compared with more affluent areas. Even when these facilities are available, transportation and financial barriers often provide challenges to use for lower-income populations. Connecting public transportation systems to everyday destinations can promote active travel,¹³⁴ and connecting public transportation to jobs may help address, in part, economic disparities and poverty. Efforts to be inclusive of persons with disabilities will also require that built environment and programmatic opportunities to be physically active are both available and accessible to all people regardless of age and abilities. Campaigns and initiatives to promote walking and walkable communities should be inclusive of those who walk and those who rely on assistive equipment or wheelchairs for mobility.¹³⁵

In general, fewer opportunities exist for engaging in health-promoting dietary and physical activity patterns among marginalized populations (e.g., people living in poverty, people of color, LGBTQ, people who have a disability or who live in a rural community, and others who have historically been excluded), thus further increasing health inequities. Strategies aimed at the general population are often less effective among racial/ethnic minority groups and those of low socioeconomic status. Initiatives must address the unique challenges and barriers that certain groups often face when attempting to modify lifestyle behaviors, with culturally appropriate tailoring and equitable support to promote healthy behaviors.

Trends in excess body weight among youth are also a significant public health concern; children with obesity are more likely than normal-weight children to become adults with obesity, and their obesity in adulthood is likely to be more severe.¹³⁶ Promoting obesity prevention and positive lifestyle behaviors during youth is more effective, and often more successful, than efforts to change unhealthy patterns of behavior in adult populations.¹³⁷ Therefore, creating health-promoting and antiobesogenic environments that make it easier for children to establish positive eating and physical activity behaviors early in life are critical.

Improving Healthy Eating and Active Living-Related Environments

Effective strategies are being tested to address healthy eating and active living by numerous organizations that have created evidence-based recommendations, including the

WHO,¹³⁸ the National Academy of Medicine,¹³⁹ the CDC, the WCRF/AICR,⁴ and the American Heart Association.¹⁴⁰ A consensus among these recommendations is a call for policy and systems change, the identification of key environments to promote change, and the need for multiple sectors to work collaboratively to reduce barriers to healthy eating and active living, particularly among lower income and racial/ethnic minority and rural communities. Thus, health promotion considerations should be incorporated into urban, rural, and regional planning and development. To reduce health-related inequities among specific population groups, community-based approaches should be adapted to meet the needs of the target community.¹³⁹

More evidence is needed to develop, implement, and evaluate which strategies, or combination of strategies, are most effective in facilitating sustained healthy eating and active living among all individuals. Although not exhaustive, the following recommendations have been identified by reputable organizations as potential strategies that multiple sectors should consider to promote improved dietary and physical activity patterns among all individuals.

Increasing Access to Healthy, Affordable Foods

Community food retail strategies

The food retail environment has a significant impact on the health of communities, and a healthy food retail environment is one in which it is easier to make healthy choices by encouraging the purchase of vegetables, fruits, whole grains, and other nutritious items, rather than energy-dense foods and beverages of low nutritional value. Community partners, including public health agencies, retailers and vendors, business leaders, health care systems and providers, local farmers, food pantries and banks, community development organizations, community members, and other stakeholders, can collaborate in a variety of ways to develop and maintain a healthy retail environment. Both full-service and smaller grocery stores can market and promote healthier choices through leveraging shelf labeling systems to help consumers identify healthier choices and place these items at eye level, in-store promotions of healthy options and recipes, and healthy checkout aisles that limit foods and beverages of low nutritional value. A store can also apply to become an approved vendor for federal nutrition assistance programs funded and administered through the USDA, which provides financial incentives to participants in federal nutrition assistance programs for the purchase of healthier options. Smaller stores face unique challenges in providing healthy options for a variety of reasons, including, among others, difficulty in meeting distributors' minimum order requirements to receive reasonable prices on healthy options. Working collaboratively, smaller stores can establish group purchasing collectives to increase purchasing power to facilitate the provision of healthier food and beverage options.^{124,141}

Food away from home

Over the past several decades, Americans have grown to rely on the convenience of foods prepared outside the home. Unfortunately, consuming food away from home, from restaurants, cafeterias, food trucks, and vending machines, typically results in the consumption of fewer fruits and vegetables and more calories, saturated fat, added sugars, and sodium than eating foods prepared at home. Hence, food consumption away from home has been associated with obesity.²³ Restaurants should ensure that their menus include a variety of nutrient-dense, lower energy dining options, including those for children. Employers, including hospitals, health care systems, schools, parks and recreation centers, government facilities, and businesses, can adopt voluntary guidelines to ensure that competitively priced, healthy food and beverage offerings are standard practice and policy in cafeterias, vending machines, and other areas where foods and beverages are available. Such employer policies and initiatives can also promote the adoption of “healthy” and active meetings and other workplace-supported events. In addition, opportunities to partner with local farmers to establish on-site farmers markets should be explored. Faith-based communities, childcare facilities, schools, and housing developments can explore the implementation of gardening initiatives and link into land-grant university extension personnel and master gardeners for free nutrition education and training. Communities can also convert vacant spaces to community gardens and can work collaboratively to bring health-focused food trucks or mobile markets into areas considered to be low-income and low-access.^{56,126,139,140}

Increasing Access to Opportunities for Physical Activity, Play, Leisure Time Activity, and Transportation

The built environment

The built environment can support efforts especially designed to increase weekly amounts of physical activity through active travel. There is extensive evidence documenting that community architecture and design affect physical activity levels among community members.¹⁴² Approaches that increase walking or bicycle transportation in a community are effective in increasing both transportation-related and recreational physical activity as well as total walking time.^{23,124,134} Considering active transportation systems (pedestrian and bicycle routes) and land use and environmental design with community health and wellness in mind can foster greater uptake of healthy lifestyle behaviors across communities. Communities should include policies or initiatives designed to create or enhance mixed land use environments that increase the diversity and proximity of local destinations where people live, work, and spend their recreation and leisure time. Such initiatives require shared values and synergistic efforts from communities, including

community members, community planners, health professionals, transportation officials, and governments, and can be effective means to make it easier for community members to be more physically active.¹²⁹ Transportation system interventions that are designed to increase (or improve) street connectivity; sidewalk, bicycle, and trail infrastructure; and public transit structure and access have proven effective in this regard.^{134,143}

Shared use agreements

Shared use occurs when government entities or other organizations agree to open their facilities for use by the broader community. Community-based school facilities can be an excellent resource for recreation, physical activity, and play in locations where there is limited space or private options are too expensive. Shared use arrangements can provide for many other types of physical activity promotion spaces, such as gyms, walking/running tracks, pools, playing fields, parks, and walking trails. Although these are not substitutes for adequate public infrastructure investments, they can be an important component of larger initiatives to promote healthy living, safe places to be physically active, and engaged neighborhoods and to advance health equity.¹⁴⁴

Physical education and physical activity in schools

To reach the ≥ 60 minutes per day and types of physical activity for youth recommended by the PAGAC,²³ quality school physical education programs should be a regular component of a comprehensive, well-rounded education for students across the country in kindergarten to 12th grade (K-12). Strategies that schools can implement to increase physical activity include executing a well-designed physical education curriculum, changing instructional practices to better incorporate more time for MVPA and play, hiring trained physical education teachers, and providing educators with professional development and training in evidence-based strategies.^{12,23} Other activities to supplement physical education can occur before, during, or after the school day. These include frequent classroom physical activity breaks, daily recess, intramural programs and activity clubs, walk-to-school or bike-to-school programs, and afterschool programs incorporating physical activity. Promoting MVPA throughout the day can help fill the gap between the amount of physical activity students receive through quality physical education and the recommended ≥ 60 minutes per day.

Decreasing Access to Alcoholic Beverages

Numerous community-level strategies to reduce harmful alcohol use outlined by the US Community Preventive Services Task Force’s Guide to Community Preventive Services¹⁴⁵ and the WHO’s Best Buys¹³⁸ include regulating the density of alcohol retail outlets through licensing or zoning processes; maintaining limits on the days that alcohol

can legally be sold in retail outlets and on the hours that alcohol can legally be sold where it is consumed on premise; enhancing the enforcement of laws prohibiting sales to minors, including increasing compliance checks at alcohol retailers (such as bars, restaurants, and liquor stores); and restricting or prohibiting promotions of alcoholic beverages in connection with sponsorships and activities that target youth.

Clinical Strategies to Promote Healthy Eating and Active Living and Limiting Alcohol

Among the key partners to promote cancer preventive behaviors are the health care providers and the health care systems in which clinical services are provided. Although few programs have comprehensive uptake across systems, the Exercise Is Medicine (exerciseismedicine.org/) and Park Prescriptions America (parkrxamerica.org) initiatives provide a prototype of how routine screening of physical activity behaviors and exercise prescriptions may support individual improvements in physical activity behaviors. Incorporation of questions about exercise as a vital sign—asked during routine visits just as blood pressure and weight are measured—has been associated with modest weight loss and lower hemoglobin A1c levels,¹⁴⁶ with growing evidence of these effects.¹⁴⁷ The Walk with a Doc program also promotes community-clinical linkages by encouraging providers to start doctor-led walking groups to promote walking as a health-enhancing behavior among their patients (walkwithadoc.org). Screening for food insecurity is also an important factor to identify those individuals at risk of dietary patterns inconsistent with cancer prevention. Health care providers can be a valuable referral source to community efforts designed to support food insecure community members, such as food assistance programs, food banks, voucher programs to retail stores and farmers markets, and more.¹⁴⁸ Inclusion of questions related to food choices as a vital sign during routine clinical encounters may also promote healthful eating behaviors. Furthermore, health care insurance communities provide a resource to support new program initiatives with a focus not only on changes in diet and activity behaviors but also on cost incentives and savings that are critical to the sustainability of health promotion programs. These partnerships warrant further attention and evaluation to attain optimal health for all individuals.

The US Preventive Services Task Force recommendations include clinical approaches to support healthy weight and for reducing alcohol consumption. It is recommended that clinicians offer or refer adults with a BMI ≥ 30 kg/m² to intensive, multicomponent behavioral interventions, as evidence suggests that such interventions can lead to clinically significant improvements in weight status as well as reduce the incidence of type 2 diabetes

among adults with obesity and elevated plasma glucose levels.¹⁴⁹ Recommendations regarding reducing alcohol consumption include alcohol screening and behavioral counseling interventions in primary care settings to identify those individuals, including pregnant women, whose alcohol consumption does not meet the criteria for alcohol dependence but places them at higher risk of alcohol-related harms.¹⁵⁰

Public Policy Approaches to Promote Healthy Eating and Active Living

Implementation of public policy initiatives is a critically important component of a comprehensive approach to supporting all individuals in limiting alcohol consumption, eating healthy diets, and living a physically active lifestyle. Policies that improve access to healthy foods and beverages; provide information to consumers to support and facilitate healthier choices; limit marketing, advertising, and accessibility to foods and beverages of low nutritional value (including alcoholic and sugar-sweetened beverages); and establish standards for and increase funding for physical activity-related infrastructure in communities all may be effective in improving healthy lifestyles and ultimately improving the primary prevention of cancer, other chronic diseases and disabilities, and other related health outcomes.^{139,151,152}

Various public policies designed to improve dietary patterns have demonstrated positive impacts. Initiatives in nutrition assistance programs, including the Special Supplemental Nutrition Program for Women, Infants, and Children Program (WIC) and the Supplemental Nutrition Assistance Program (SNAP), which allow for and provide incentives for healthy food purchases such as vegetables and fruit, have resulted in healthier food purchases and dietary patterns¹⁵² and could result in substantial health benefits.¹⁵³ Standards for foods and beverages served in schools and in early childcare and education settings help to ensure youth are exposed to nutrient-dense options and that energy-dense options are limited.¹⁵⁴ And, although research is mixed regarding the results of the overall impact of menu labeling legislation, it is possible that labeling is more effective in specific types of restaurants and that restaurants are reformulating menu items in part because of this legislation.¹⁵⁵

Numerous recommended policy approaches to promote more lifelong physical activity among Americans include a variety of policies designed to impact youth: comprehensive school physical activity programming, including high-quality physical education and daily recess; requirements for physical activity in afterschool and in early childcare programs; and active transport opportunities to school, among others, all have the potential to increase physical activity levels among youth.¹⁵²

Funding that continues to expand and support community design that facilitates active transport (eg, complete streets policies), that expands and improves zoning policies that encourage mixed-use development, and that invests in more public transportation options will also be important to facilitate more physically active lifestyles.¹¹¹

Finally, it is well established that raising excise taxes on tobacco products leads to higher prices, which, in turn, cause declines in consumption, and recent research suggests that raising excise taxes on sugar-sweetened beverages and alcohol also can reduce consumption of these products.^{156,157} Tax revenues, in turn, can be reallocated back to promote societal well-being.¹⁵⁸

Ensuring that all individuals have access to affordable, healthy food choices and opportunities for safe physical activity will require multiple strategies and bold action, ranging from the implementation of community, worksite, school, childcare, and other health promotion programs to policies that affect community planning, architecture, transportation, school-based physical education, food advertising and marketing, and food services. Special attention must also be paid to recognizing that individuals and populations with the greatest needs, the least resources, and/or those facing increased and unique burdens and challenges require different, not equal, effort and resources to improve their health. Policies, programs, and services should be explicit about prioritizing these populations to achieve health equity. Public, private, and community organizations at local, state, and national levels should consider the implementation and testing of new policies and the reallocation or expansion of resources to weight management, greater physical activity, and healthful diet choices (including avoidance of alcohol) that will improve health. Health care professionals; school, business, faith group, and other community leaders; and elected officials and policy makers are in unique and critical positions to provide leadership and advocate for purposeful changes in public policy and in community environments that are necessary to help all individuals maintain a healthy body weight and remain physically active throughout life, and to engage community members in the design, implementation, and evaluation of these strategies within the aforementioned sectors.¹¹¹

Common Questions and Answers

This section is intended to assist clinicians, public health professionals, and policymakers in addressing questions that commonly arise within the general public.

Acrylamide

What is acrylamide, and is it associated with an increased risk of cancer?

Acrylamide is a chemical used in industrial processing and is also found in food and tobacco smoke. Acrylamide in food is formed as a by-product of the Maillard reaction, in which

the amino acid asparagine reacts with certain sugars when heated to high temperatures. The major sources of acrylamide in our diets are French fries and potato chips; crackers, bread, and cookies; breakfast cereals; canned black olives; prune juice; and coffee.

Acrylamide is classified by the IARC as a “probable carcinogen,” based primarily on experiments in animals. However, large numbers of epidemiologic studies (both case-control and cohort studies) in humans have found no strong evidence that dietary acrylamide exposure is associated with the risk of any type of cancer.^{159–161}

Alcoholic beverages

Is there a safe level of consumption? Do some types of alcohol present less risk?

There is a scientific evidence that alcohol consumption causes several types of cancer and that to reduce the risk of developing several types of cancer, there is no safe level of consumption. The evidence indicates that the more alcohol a person drinks, the higher his or her risk of developing an alcohol-associated cancer. The risk of some cancers increases at even less than one drink a day. The recommendation for those who do choose to drink alcohol – no more than 2 drinks per day for men and no more than one drink per day for women – is not intended as advice for an average over several days, but rather the amount consumed on any single day.⁴

All alcohol, regardless of the type – beer, wine, liquor – contains ethanol, which is the cancer-causing compound in alcoholic beverages.¹¹² No type of alcohol beverages is less risky in terms of its impact on cancer risk.

Antioxidants

What are antioxidants, and what do they have to do with cancer?

The body uses certain nutrients, bioactive food components, and endogenously produced compounds for protection against damage to tissues that is constantly occurring as a result of normal oxidative metabolism. Because such damage is associated with increased cancer risk, some antioxidants are thought to protect against cancer. Antioxidants obtained from the diet include vitamin C, vitamin E, carotenoids, and many other bioactive food components. Studies suggest that people who eat more vegetables and fruits, which are rich sources of antioxidants, may have a lower risk for some types of cancer.⁴ However, this does not mean that the benefits of vegetables and fruits result primarily from their antioxidant content rather than from other bioactive food components. Several clinical trials of antioxidant supplements have not demonstrated a reduction in cancer risk from these supplements; indeed, some demonstrated an increased risk of cancer among those taking supplements.⁴ To reduce cancer risk, the best advice is to consume antioxidants through whole food sources rather than supplements.

Arsenic

What is arsenic? Does it cause cancer?

The WHO,¹⁶² the US National Toxicology Program,¹⁶³ and others have classified arsenic as carcinogenic to humans. Arsenic is a naturally occurring element that can be found in rocks and soil, water, air, plants, and animals as well as in industrial and agricultural compounds. It is through these sources that arsenic can enter our water and food supply and increase human exposure. Arsenic is found in 2 forms: inorganic and organic compounds. Inorganic arsenic compounds are used in industry and in building products (such as some “pressure-treated” woods) and are found in arsenic-contaminated water. This tends to be the more toxic form of arsenic and has been linked to cancer. The organic arsenic compounds are thought to be much less toxic than the inorganic arsenic compounds and are not thought to be linked to cancer.

The main sources of human exposure to arsenic are water and food. Water in some areas of the United States, especially in the Southwest, New England, upper Midwest, and West, may be higher in arsenic.¹⁶⁴ Natural arsenic levels tend to be higher in drinking water that comes from ground sources, such as wells. For most people, food is the largest source of arsenic, although much of this is likely to be in the less dangerous, organic form. The highest levels of arsenic in foods are found in seafood, rice and other rice products, mushrooms, and poultry, although many other foods, including some fruit juices, can contain arsenic.

Studies have identified that exposure to arsenic in drinking water may cause lung, bladder, and skin cancers.^{4,165} Because arsenic has been linked to cancer and other negative health effects, several US government agencies regulate arsenic levels and exposures. Although arsenic is a naturally occurring element and thus cannot be avoided completely, there are things individuals can do that may lower their exposure. Those whose drinking water comes from a public source can obtain publicly available information about the levels of certain substances in drinking water, including arsenic. If water is secured from a private source such as a well, individuals can have arsenic levels tested by a reputable laboratory. Those who live in areas with high levels of arsenic in the water may consider using alternative sources of drinking water, such as bottled water. Common household water filters do not effectively remove arsenic. Avoiding excessive consumption of foods known to contain high levels of arsenic, including seafood, rice and rice products, and fruit juice, would also help lower exposure,^{162,163} and maintaining good folate status is important for the elimination of arsenic in the body.¹⁶⁵

Coffee

Does drinking coffee impact cancer risk?

Whether coffee consumption reduces or increases the risk of different types of cancers has been an active area of research. Studies have suggested that coffee consumption likely

reduces the risk of liver and endometrial cancers, although confounding by smoking may explain this latter association.⁴ There is some evidence that coffee reduces the risk of cancers of the mouth, pharynx, and larynx as well as basal cell skin cancer in both men and women, and possibly malignant melanoma in women.^{4,166}

On a related topic, previous studies have suggested that consuming very hot beverages, above 149 degrees Fahrenheit, such as coffee and/or tea, may increase the risk of esophageal cancer, and a recent meta-analysis supported this conclusion.¹⁶⁷ There may be an advantage to consuming coffee and other beverages at a modest (rather than very hot) temperature.

The potential mechanisms by which coffee may exert beneficial effects on the risk of some cancers are not completely understood. Hundreds of biologically active compounds, including caffeine, flavonoids, lignans, and other polyphenols, are found in roasted coffee. These and other coffee compounds have been shown to increase energy expenditure, inhibit cellular damage, regulate genes involved in DNA repair, have anti-inflammatory properties, and/or inhibit metastasis.^{4,166} Coffee also influences intestinal transit time and liver metabolism of carcinogens, and therefore these factors may also contribute to a lower risk for some digestive cancers.

Genetically Modified Crops

What are genetically modified crops, and are they safe?

Genetically modified or bioengineered crops are made by adding genes from other plants or organisms to increase a plant's resistance to insect pests; retard spoilage; or improve transportability, flavor, nutrient composition, or other desired qualities. Certain foods produced from genetically modified crops have been approved for sale in the United States since the mid-1990s, and >70% of all highly processed foods on US supermarket shelves—including pizza, potato chips, cookies, ice cream, salad dressing, corn syrup, and baking powder—contain ingredients from bioengineered soybeans, corn, or canola plants. Growing public concern about the potential harmful effects of genetically modified foods, in part, led to federal legislation in 2016 requiring uniform labeling of foods containing genetically engineered ingredients.^{168,169}

In theory, these added genes might create substances that could cause adverse reactions among sensitized or allergic individuals or may result in the presence of elevated levels of compounds with adverse health effects. However, there is currently no evidence that foods containing genetically engineered ingredients or the substances found in them that are now on the market are harmful to human health or that they would either increase or decrease cancer risk.⁴ The WHO, the American Medical Association, the National Academy of Sciences, and the American Association for the Advancement of Science have all taken the stance that

current evidence suggests that foods containing genetically engineered ingredients are safe.

Gluten-Free Diet

Does eating a gluten-free diet help to reduce cancer risk?

Gluten is a protein in wheat, rye, and barley that, for most people, causes no ill effects. For those who have celiac disease, gluten triggers an immune response that damages the lining of the small intestine and could increase the risk of cancer. Some individuals experience gluten sensitivity without overt celiac disease, and, in these individuals, gluten may contribute to inflammation within the gut, one mechanism that may drive gastrointestinal cancers. However, these associations have not been well characterized, and there is scant evidence relating gluten intake to the risk of gastrointestinal cancers in the general population. For those individuals without celiac disease, there is no evidence that consuming a gluten-free diet is associated with a lower cancer risk, and numerous studies suggest that consuming whole grains, including those containing gluten, probably reduces the risk of colon cancer.⁴

Glycemic Index and Glycemic Load

What are these, and do they impact cancer risk?

The glycemic index is a measure of the increase in the level of blood glucose after eating a specific carbohydrate-rich food compared with eating a standard amount of glucose. Foods with a high glycemic index release glucose quickly and show a rapid rise in blood glucose. Foods with a low glycemic index release glucose into the blood more slowly, with a lower overall peak in blood glucose over time. In general, foods with a high glycemic index are highly refined, processed grain products with added sugars and low fiber content, as well as some starchy vegetables. The glycemic index can be considered a measure of carbohydrate-rich food quality. Also important in addition to quality is quantity. Beyond the glycemic index, glycemic load captures both the quality and quantity of carbohydrates consumed. The glycemic load gives a truer picture of how blood glucose is elevated in relation to the intake of a specific food item.

Much research has been conducted examining the potential impact of the glycemic load of a diet on cancer risk.¹⁷⁰ Most recent comprehensive reports indicate that eating a dietary pattern high in glycemic load is associated with a higher risk of endometrial cancer. More research is needed to determine the impact on additional cancer sites.

Inflammation and Anti-Inflammatory Strategies

Do anti-inflammatory diets reduce cancer risk?

Inflammation has long been recognized as a physiological response to tissue injury, and its relationship to microbial infection was recognized hundreds of years ago. However, the role of inflammation in carcinogenesis was recognized

more recently, and the relationships of diet, inflammation, and risk of cancer (as well as cardiovascular disease and overall mortality) are still an evolving area of research.

A combination of laboratory experimentation and epidemiological research has identified certain foods and their chemical components that promote systemic inflammation as well as chronic inflammation of certain tissues.^{171,172} This information is the basis of anti-inflammatory dietary patterns, which share some characteristics with the recommendations in this guideline, such as high intake of vegetables and fruit and low intake of red and processed meat.

Irradiated Foods

Why are foods irradiated, and can these foods increase cancer risk?

Food irradiation (the application of ionizing radiation to food) is a technology that improves the safety and extends the shelf life of foods by reducing or eliminating microorganisms and insects. Similar to pasteurizing milk and canning fruits and vegetables, irradiation can make food safer for the consumer. Irradiation does not make foods radioactive; compromise nutritional quality; or noticeably change the taste, texture, or appearance of food. In fact, changes made by irradiation are so minimal that it is not easy to tell if a food has been irradiated.

The US Food and Drug Administration has evaluated the safety of irradiated food for >30 years and has found the process to be safe. The WHO, the CDC, and the USDA have also endorsed the safety of irradiated food. There is currently no evidence that irradiation of foods causes cancer or has harmful human health effects.^{168,173}

Juicing/Cleanses/Detox

Can periods of limiting food intake to juices remove toxins and provide protection against cancer?

Fruit and vegetable juices can be a convenient way to consume beneficial, bioactive food components in vegetables and fruit and, in moderation, can be a worthwhile component of healthful dietary patterns. However, juices contain less fiber, lower levels of some other beneficial nutrients, and more naturally occurring sugar than the whole fruits and vegetables they are made from and thus are not the best way to obtain nutrients from plant-based foods.

There is no scientific evidence to support claims that exclusively consuming juices for ≥ 1 days reduces cancer risk or provides other health benefits. Known as juice cleanses or juice detoxification, this kind of diet is promoted as a way to remove “toxins,” but that claim is not supported by scientific evidence. Toxins that enter our body through the foods and beverages we consume are continually eliminated by the kidneys and liver, regardless of whether a person is consuming liquid or solid foods. Although vegetable juicing may be one way to increase micronutrient intake,¹⁷⁴ a diet limited to juice may

also be inadequate in some important nutrients and, in select cases, may contain dangerous levels of some substances that can cause kidney damage and other health problems.¹⁷⁵⁻¹⁷⁷

Microwaving Food and General Food Preservation, Preparation, and Storage

Can using microwave ovens or other cooking methods increase cancer risk?

Microwaves are a form of nonionizing electromagnetic radiation and their use in cooking does not increase cancer risk.¹⁷⁸ Conversely, grilling, smoking, or pan-frying meats (including red meats as well as poultry and fish) at high temperatures can cause chemical reactions of amino acids, sugars, and creatine or creatinine to form carcinogenic heterocyclic amines.^{95,96}

Goals of food preservation, processing, and preparation that are relevant to individual and public health include: removing or inactivating any harmful chemical or microbiological contaminants, avoiding the addition or production of harmful substances, and maintaining the amount and bioavailability of nutrients. For example, proper canning or freezing methods can maintain the nutrient content of vegetables and fruits to expand consumers' access to these products. Conversely, certain methods of preserving red meats introduce nitrates that are metabolized by certain bacteria in the stomach to form carcinogenic N-nitroso compounds.

Contamination of foods by substances from storage containers or cookware is another concern of some consumers. Plastic containers can release substances such as phthalates (some of which are classified as possible carcinogens) or phenolic compounds such as bisphenol A (a probable carcinogen) during storage of food or during cooking in a microwave oven. Use of Teflon-coated cookware may release perfluorooctanoic acid (a possible carcinogen) into foods. These substances have adverse biological effects in some in vitro or animal models and may influence the onset of puberty,¹⁷⁹⁻¹⁸¹ a possible factor in the long-term risk of cancers such as breast cancer. However, long-term impacts of exposure to these chemicals on cancer risk in epidemiologic studies are lacking. Nonetheless, consumers who are concerned about possible harm from these exposures can choose glass or metal storage containers and cookware.

Non-Nutritive Sweeteners/Sugar Substitutes

Do non-nutritive sweeteners/sugar substitutes cause cancer?

Non-nutritive sweeteners are substances used instead of sugars (ie, sucrose, corn syrup, honey, agave nectar) to sweeten foods, beverages, and other products such as oral care products and certain medications. There are currently numerous non-nutritive sweeteners approved by the US Food and Drug Administration, including aspartame, acesulfame

potassium, saccharin, sucralose, and stevia. These sweeteners contain few or no calories or nutrients. They may be derived from herbs and other plants, or from sugar itself, and typically are many times sweeter than sugar, enabling smaller quantities to be used. Additional sugar substitutes include sugar alcohols, such as sorbitol, xylitol, and mannitol.

There is no clear evidence that these sweeteners, at the levels typically consumed in human diets, cause cancer.¹⁸² Questions about artificial sweeteners and cancer risk arose when early studies showed that saccharin caused bladder cancer in laboratory animals, but studies in humans have demonstrated no increased cancer risk. People with one rare genetic disorder, phenylketonuria, metabolize aspartame abnormally, resulting in nervous system toxicity, and for this reason should avoid aspartame in their diets. With that exception, all of these sweeteners appear to be safe when consumed in moderation, although larger quantities of sugar alcohols may cause bloating and abdominal discomfort in some people.

Organic Foods

Are foods labeled “organic” more effective in lowering cancer risk?

The term “organic” is popularly used to designate foods grown without the addition of artificial chemicals. Under USDA regulations, animal-derived foods that are labeled as organic come from animals raised without the addition of hormones or antibiotics to the feed provided. Plant foods that are organic come from agricultural methods that do not use most conventional insecticides or herbicides, chemical fertilizers, or sewage sludge as fertilizer. Organic foods also exclude the use of industrial solvents or food irradiation in processing, and genetically modified foods are also excluded. A primary benefit of organic food consumption is to support environmentally sustainable agricultural practices. In addition, many consumers believe that the consumption of organic foods may provide health benefits, but there is little evidence that organic produce has higher nutrient levels than conventionally grown produce. Little research has been conducted on the association of organic food consumption and cancer risk, although a recent study found an inverse association of organic produce consumption and the risk of non-Hodgkin lymphoma.¹⁸³ Although these findings should be replicated, they are consistent with the strong and consistent association noted between occupational pesticide exposure and this form of cancer. Washing conventionally grown produce can remove some of the pesticide residue; it is also important to wash all produce to minimize the risk of ill health effects from microbial contamination. Because organic produce is often more expensive than similar, conventionally produced items, it is important for individuals with limited resources to recognize that meeting the recommendation for vegetable and

fruit intake is a higher priority for cancer prevention and overall health than choosing organic produce.^{183–185}

Pesticides

Do pesticides in foods cause cancer?

Insecticides and herbicides, 2 types of pesticides, can be toxic when used improperly in industrial, agricultural, or other occupational settings. The IARC classifies 3 common agricultural herbicides (glyphosate, malathion, and diazinon) as probable human carcinogens. All 3 are associated with a higher risk of non-Hodgkin lymphoma. In addition, malathion and diazinon are associated with excess risk of prostate and lung cancers, respectively.

Currently, scientific evidence supports the overall health benefits and cancer-protective effects of eating vegetables and fruits, regardless of whether they are grown using organic or conventional practices. Washing conventionally grown produce can remove some of the pesticide residues, and is also important to minimize the risk of microbial contamination.

Sleep

How does sleep impact diet, physical activity, and cancer risk?

Increasing evidence suggests there is an important interplay among sleep, diet, physical inactivity, and cancer risk. Disordered sleep has been associated with a higher cancer risk,¹⁸⁶ and sleep deprivation (usually defined as <7 hours per night) has been associated with a higher risk for obesity, overeating, and related metabolic syndrome,¹⁸⁷ a known risk factor for several cancers. Alternately, a healthy sleep pattern has been associated with better weight maintenance after weight loss.¹⁸⁸ Studies also found that high levels of sedentary time were associated with poor sleep quality and shorter sleep duration.¹⁸⁹ Inadequate sleep has been associated with elevated stress hormones and inflammation, known mechanisms driving cancer risk.

Soy and Soy Products

Can soy-based foods reduce cancer risk?

As with other beans or legumes, soy and foods derived from soy are an excellent source of protein and thus provide a healthier alternative to meat. Soy contains several bioactive food components, including isoflavones, which have a structure similar to that of estrogens and are capable of binding to estrogen receptors, leading to weak estrogenic effect, antiestrogenic effects, or no effects, depending on conditions, specific tissue, and dose.¹⁹⁰

There is some evidence from epidemiologic and laboratory studies that the consumption of traditional soy foods such as tofu may decrease the risk of cancers of the breast¹⁹¹ and prostate,¹⁹² but overall, the evidence remains too limited for a firm conclusion.⁴ Many of the supportive studies are

based on Asian populations with a high lifelong consumption of soy foods, and their relevance to soy consumption by Western populations at low levels and for a short-to-medium duration remains uncertain. There are no data to support the use of supplements containing isolated soy phytochemicals or soy protein powders used in some food products for reducing cancer risk. In fact, a recent study found increased risk among users of soy supplements for estrogen receptor-negative breast cancer (an aggressive type) and for women with a family history of breast cancer.¹⁹³ Therefore, although soy from food sources appears to be safe and may even have multiple beneficial health effects,¹⁹⁴ soy supplements should be used with caution, if at all.

Sugar

Does sugar increase cancer risk?

Several types of sugars are found in foods and beverages. These sugars vary in their chemical structures but, once they are consumed, they have similar metabolic effects. All sugars in foods and beverages contribute to caloric intake, so, by promoting obesity, a high sugar intake can indirectly increase cancer risk. There is also evidence that a dietary pattern high in added sugars influences levels of insulin and related hormones in ways that may increase the risk of certain cancers.³⁹ Brown (unrefined) sugar contains the same chemical form of sugar (sucrose) as white (refined) sugar and also contains extremely small amounts of other substances that affect its color and flavor but do not influence the unfavorable effects of sucrose on body weight or insulin. Fructose, the natural sugar in fruit and in many sugar-sweetened beverages in the form of high fructose corn syrup, is similar to sucrose with regard to its effects on weight and insulin, as is honey, which contains a mixture of fructose and glucose (another form of sugar).

Laboratory studies have shown that metabolism of glucose occurs more rapidly in cancer cells than in normal cells. This fact is often misinterpreted by people who are unfamiliar with the relevant metabolic pathways, who assume (incorrectly) that sugars in foods and beverages directly “feed” cancer cells.

Nonetheless, limiting highly processed foods containing high levels of added sugars, such as cakes, candy, cookies, and sweetened cereals, as well as sugar-sweetened beverages, such as soda, sports drinks, and energy drinks, can help to reduce caloric intake, minimize weight gain, and promote a healthier body weight as well as lower insulin secretion in individuals with metabolic abnormalities, such as those with prediabetes or type 2 diabetes.

Vegetarian/Vegan Diets

Do vegetarian diets reduce cancer risk?

Vegetarian diets can include many health-promoting features: they tend to be low in saturated fat and high in fiber,

vitamins, and other bioactive food components¹⁹⁵ and do not include red and processed meats. Thus, it is reasonable to suggest that vegetarian diets may be beneficial for cancer risk reduction. Many studies of vegetarians indicate a lower risk of cancer overall relative to people who also eat meat. Whether vegetarian diets confer any special benefits over diets that include smaller amounts of animal products than are typically consumed in Western diets is less clear; indeed, in a large British study, people who ate fish, but not other meats, appeared to have the same overall cancer risk as vegetarians.¹⁹⁶

The available evidence supports the recommendation of a dietary pattern that consists predominantly of foods from plant sources, with limited if any intake of red and processed

meats.⁴ In addition to a modest level of risk reduction for some forms of cancer relative to a more typical Western dietary pattern with higher levels of meat consumption, vegetarian dietary patterns are associated with a lower risk of cardiovascular disease and type 2 diabetes and are generally more affordable. Individuals consuming strict vegetarian diets that omit all animal products, including milk and eggs, referred to as “vegan” diets, need supplementation with vitamin B12, zinc, and iron (or foods fortified with these nutrients), especially for children and premenopausal women. They should also aim to achieve adequate calcium intake, as people consuming vegan diets with relatively low calcium content have been shown to carry a higher risk of fractures compared with people consuming vegetarian or meat-containing diets.¹⁹⁵ ■

References

1. Siegel RL, Miller KD, Jemal A. Cancer statistics, 2018. *CA Cancer J Clin*. 2018; 68:7-30.
2. Islami F, Goding Sauer A, Miller KD, et al. Proportion and number of cancer cases and deaths attributable to potentially modifiable risk factors in the United States. *CA Cancer J Clin*. 2018;68: 31-54.
3. World Cancer Research Fund/American Institute for Cancer Research. Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective. American Institute for Cancer Research; 2007. Accessed July 21, 2019. discovery.ucl.ac.uk/id/eprint/4841/1/4841.pdf
4. World Cancer Research Fund/American Institute for Cancer Research. Diet, Nutrition, Physical Activity and Cancer: A Global Perspective. Continuous Update Project. The Third Expert Report. American Institute for Cancer Research; 2018. Accessed July 21, 2019. wcrf.org/dietandcancer
5. Kushi LH, Doyle C, McCullough M, et al. American Cancer Society Guidelines on nutrition and physical activity for cancer prevention: reducing the risk of cancer with healthy food choices and physical activity. *CA Cancer J Clin*. 2012;62: 30-67.
6. US Department of Health and Human Services. 2018 Physical Activity Guidelines Advisory Committee. 2018 Physical Activity Guidelines Advisory Committee Scientific Report. Office of Disease Prevention and Health Promotion, US Department of Health and Human Services; 2018.
7. US Department of Agriculture, Dietary Guidelines Advisory Committee. Scientific Report of the 2015 Dietary Guidelines Advisory Committee. 2015. Accessed July 21, 2019. health.gov/dietaryguidelines/2015-scientific-report/
8. Toledo E, Salas-Salvado J, Donat-Vargas C, et al. Mediterranean diet and invasive breast cancer risk among women at high cardiovascular risk in the PREDIMED trial: a randomized clinical trial. *JAMA Intern Med*. 2015;175:1752-1760.
9. Chlebowski RT, Luo J, Anderson GL, et al. Weight loss and breast cancer incidence in postmenopausal women. *Cancer*. 2019;125:205-212.
10. McCullough ML, Zoltick ES, Weinstein SJ, et al. Circulating vitamin D and colorectal cancer risk: an international pooling project of 17 cohorts. *J Natl Cancer Inst*. 2019;111:158-169.
11. Luo J, Chlebowski RT, Hendryx M, et al. Intentional weight loss and endometrial cancer risk. *J Clin Oncol*. 2017;35: 1189-1193.
12. US Department of Health and Human Services. 2018 Physical Activity Guidelines Advisory Committee Scientific Report. Accessed September 12, 2019. health.gov/paguidelines/second-edition/report/
13. Campbell PT, Newton CC, Kitahara CM, et al. Body size indicators and risk of gallbladder cancer: a pooled analysis of individual-level data from 19 prospective cohort studies. *Cancer Epidemiol Biomarkers Prev*. 2017;26:597-606.
14. Carreras-Torres R, Johansson M, Haycock PC, et al. Obesity, metabolic factors and risk of different histological types of lung cancer: a Mendelian randomization study. *PLoS One*. 2017;12:e0177875.
15. Keum N, Greenwood DC, Lee DH, et al. Adult weight gain and adiposity-related cancers: a dose-response meta-analysis of prospective observational studies. *J Natl Cancer Inst*. 2015;107:djv088.
16. Genkinger JM, Kitahara CM, Bernstein L, et al. Central adiposity, obesity during early adulthood, and pancreatic cancer mortality in a pooled analysis of cohort studies. *Ann Oncol*. 2015;26: 2257-2266.
17. Wilson KM, Shui IM, Mucci LA, Giovannucci E. Calcium and phosphorus intake and prostate cancer risk: a 24-y follow-up study. *Am J Clin Nutr*. 2015;101:173-183.
18. Steele CB, Thomas CC, Henley SJ, et al. Vital signs: trends in incidence of cancers associated with overweight and obesity—United States, 2005-2014. *MMWR Morb Mortal Wkly Rep*. 2017; 66:1052-1058.
19. Kitahara CM, McCullough ML, Franceschi S, et al. Anthropometric factors and thyroid cancer risk by histological subtype: pooled analysis of 22 prospective studies. *Thyroid*. 2016;26:306-318.
20. Mahabir S, Willett WC, Friedenreich CM, et al. Research strategies for nutritional and physical activity epidemiology and cancer prevention. *Cancer Epidemiol Biomarkers Prev*. 2018;27:233-244.
21. Mayne ST, Playdon MC, Rock CL. Diet, nutrition, and cancer: past, present and future. *Nat Rev Clin Oncol*. 2016;13: 504-515.
22. Grosso G, Bella F, Godos J, et al. Possible role of diet in cancer: systematic review and multiple meta-analyses of dietary patterns, lifestyle factors, and cancer risk. *Nutr Rev*. 2017;75:405-419.

23. US Department of Health and Human Services. Physical Activity Guidelines for Americans 2nd edition. 2018. Accessed July 21, 2019. health.gov/paguidelines/second-edition/pdf/Physical_Activity_Guidelines_2nd_edition.pdf
24. Bowen KJ, Sullivan VK, Kris-Etherton PM, Petersen KS. Nutrition and cardiovascular disease—an update. *Curr Atheroscler Rep*. 2018;20:8.
25. Eckel RH, Jakicic JM, Ard JD, et al. 2013 AHA/ACC guideline on lifestyle management to reduce cardiovascular risk: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol*. 2014;63:2960-2984.
26. National Academies of Sciences, Engineering, and Medicine. Sustainable Diets, Food, and Nutrition: Proceedings of a Workshop—in Brief. The National Academies Press; 2018.
27. Williams CM, Lovegrove JA, Griffin BA. Dietary patterns and cardiovascular disease. *Proc Nutr Soc*. 2013;72:407-411.
28. National Institute of Diabetes and Digestive and Kidney Diseases. National Institute of Diabetes and Digestive and Kidney Diseases. Accessed June 24, 2019. niddk.nih.gov/
29. Flegal KM, Shepherd JA, Looker AC, et al. Comparisons of percentage body fat, body mass index, waist circumference, and waist-stature ratio in adults. *Am J Clin Nutr*. 2009;89:500-508.
30. World Health Organization. Physical Status: The Use and Interpretation of Anthropometry. Report of a WHO Expert Committee (Technical Report Series No. 854). World Health Organization; 1995.
31. Lew EA, Garfinkel L. Variations in mortality by weight among 750,000 men and women. *J Chronic Dis*. 1979;32:563-576.
32. International Agency for Research on Cancer. IARC Handbooks of Cancer Prevention: Weight Control and Physical Activity. Vol 6. World Health Organization/IARC; 2002.
33. Lauby-Secretan B, Scoccianti C, Loomis D, et al. Body fatness and cancer—viewpoint of the IARC Working Group. *N Engl J Med*. 2016;375:794-798.
34. Teras LR, Kitahara CM, Birmann BM, et al. Body size and multiple myeloma mortality: a pooled analysis of 20 prospective studies. *Br J Haematol*. 2014;166:667-676.
35. Sung H, Siegel RL, Rosenberg PS, Jemal A. Emerging cancer trends among young adults in the USA: analysis of a population-based cancer registry. *Lancet Public Health*. 2019;4:e137-e147.
36. Look AHEAD Research Group, Pi-Sunyer X, Blackburn G, et al. Reduction in weight and cardiovascular disease risk factors in individuals with type 2 diabetes: one-year results of the Look AHEAD trial. *Diabetes Care*. 2007;30:1374-1383.
37. Knowler WC, Barrett-Connor E, Fowler SE, et al. Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med*. 2002;346:393-403.
38. Giovannucci E, Harlan DM, Archer MC, et al. Diabetes and cancer: a consensus report. *CA Cancer J Clin*. 2010;60:207-221.
39. Perez-Hernandez AI, Catalan V, Gomez-Ambrosi J, Rodriguez A, Fruhbeck G. Mechanisms linking excess adiposity and carcinogenesis promotion. *Front Endocrinol (Lausanne)*. 2014;5:65.
40. Djuric Z. Obesity-associated cancer risk: the role of intestinal microbiota in the etiology of the host proinflammatory state. *Transl Res*. 2017;179:155-167.
41. van Gemert WA, Schuit AJ, van der Palen J, et al. Effect of weight loss, with or without exercise, on body composition and sex hormones in postmenopausal women: the SHAPE-2 trial. *Breast Cancer Res*. 2015;17:120.
42. Hales CM, Carroll MD, Fryar CD, Ogden CL. Prevalence of obesity among adults and youth: United States, 2015-2016. *NCHS Data Brief*. 2017;288:1-8.
43. National Center for Health Statistics, National Health Interview Survey, Sample Adult Core Component. Figure 7.1. Percentage of adults aged 18 and over who met 2008 federal physical activity guidelines for aerobic activity through leisure-time aerobic activity: United States, 2006-2018. Accessed July 21, 2019. public.tableau.com/profile/tina.norris#!/vizhome/FIGURE7_1/Dashboard7_1
44. Centers for Disease Control and Prevention. Trends in the Prevalence of Physical Activity and Sedentary Behaviors National Youth Risk Behavior Surveillance System (YRBS): 1991-2017. Centers for Disease Control and Prevention; 2019.
45. Chau JY, Merom D, Grunseit A, Rissel C, Bauman AE, van der Ploeg HP. Temporal trends in non-occupational sedentary behaviours from Australian Time Use Surveys 1992, 1997 and 2006. *Int J Behav Nutr Phys Act*. 2012;9:76.
46. Yang L, Cao C, Kantor ED, et al. Trends in sedentary behavior among the US population, 2001-2016. *JAMA*. 2019;321:1587-1597.
47. Wilmot EG, Edwardson CL, Achana FA, et al. Sedentary time in adults and the association with diabetes, cardiovascular disease and death: systematic review and meta-analysis. *Diabetologia*. 2012;55:2895-2905.
48. Ford ES, Caspersen CJ. Sedentary behaviour and cardiovascular disease: a review of prospective studies. *Int J Epidemiol*. 2012;41:1338-1353.
49. Hojman P, Gehl J, Christensen JF, Pedersen BK. Molecular mechanisms linking exercise to cancer prevention and treatment. *Cell Metab*. 2018;27:10-21.
50. Koelwyn GJ, Quail DF, Zhang X, White RM, Jones LW. Exercise-dependent regulation of the tumour microenvironment. *Nat Rev Cancer*. 2017;17:620-632.
51. Neilson HK, Conroy SM, Friedenreich CM. The influence of energetic factors on biomarkers of postmenopausal breast cancer risk. *Curr Nutr Rep*. 2014;3:22-34.
52. Lynch B, Mahmood S, Boyle T. Sedentary behaviour and cancer. In: Leitzmann M, ed. *Sedentary Behaviour Epidemiology*. Springer International Publishing; 2018:245-298.
53. Kerr J, Anderson C, Lippman SM. Physical activity, sedentary behaviour, diet, and cancer: an update and emerging new evidence. *Lancet Oncol*. 2017;18:e457-e471.
54. Zhang FF, Cudhea F, Shan Z, et al. Preventable cancer burden associated with poor diet in the United States. *JNCI Can Spec*. 2019;3:pkz034.
55. Willett WC. *Nutritional Epidemiology*. 3rd ed. Oxford University Press; 2013.
56. US Department of Health and Human Services, US Department of Agriculture. 2015-2020 Dietary Guidelines for Americans. US Department of Health and Human Services; 2015.
57. Liese AD, Krebs-Smith SM, Subar AF, et al. The Dietary Patterns Methods Project: synthesis of findings across cohorts and relevance to dietary guidance. *J Nutr*. 2015;145:393-402.
58. Fung TT, McCullough ML, Newby PK, et al. Diet-quality scores and plasma concentrations of markers of inflammation and endothelial dysfunction. *Am J Clin Nutr*. 2005;82:163-173.
59. Trichopoulou A, Costacou T, Bamia C, Trichopoulos D. Adherence to a Mediterranean diet and survival in a Greek population. *N Engl J Med*. 2003;348:2599-2608.

60. Appel L, Moore T, Obarzanek E, et al. A clinical trial of the effects of dietary patterns on blood pressure. *N Engl J Med*. 1997;336:1117-1124.
61. Fung TT, Chiuve SE, McCullough ML, Rexrode KM, Logroscino G, Hu FB. Adherence to a DASH-style diet and risk of coronary heart disease and stroke in women. *Arch Intern Med*. 2008;168:713-720.
62. Guenther PM, Casavale KO, Reedy J, et al. Update of the Healthy Eating Index: HEI-2010. *J Acad Nutr Diet*. 2013;113:569-580.
63. Chiuve SE, Fung TT, Rimm EB, et al. Alternative dietary indices both strongly predict risk of chronic disease. *J Nutr*. 2012;142:1009-1018.
64. Steck SE, Guinter M, Zheng J, Thomson CA. Index-based dietary patterns and colorectal cancer risk: a systematic review. *Adv Nutr*. 2015;6:763-773.
65. Schwingshackl L, Bogensberger B, Hoffmann G. Diet quality as assessed by the Healthy Eating Index, Alternate Healthy Eating Index, Dietary Approaches to Stop Hypertension Score, and health outcomes: an updated systematic review and meta-analysis of cohort studies. *J Acad Nutr Diet*. 2018;118:74-100.e111.
66. Schwingshackl L, Hoffmann G. Adherence to Mediterranean diet and risk of cancer: an updated systematic review and meta-analysis of observational studies. *Cancer Med*. 2015;4:1933-1947.
67. Nelson ME, Hamm MW, Hu FB, Abrams SA, Griffin TS. Alignment of healthy dietary patterns and environmental sustainability: a systematic review. *Adv Nutr*. 2016;7:1005-1025.
68. Willett W, Rockstrom J, Loken B, et al. Food in the Anthropocene: the EAT-Lancet Commission on healthy diets from sustainable food systems. *Lancet*. 2019;393:447-492.
69. Tabung FK, Brown LS, Fung TT. Dietary patterns and colorectal cancer risk: a review of 17 years of evidence (2000-2016). *Curr Colorectal Cancer Rep*. 2017;13:440-454.
70. Singh RK, Chang HW, Yan D, et al. Influence of diet on the gut microbiome and implications for human health. *J Transl Med*. 2017;15:73.
71. O'Keefe SJD, Li JV, Lahti L, et al. Fat, fiber and cancer risk in African Americans and rural Africans. *Nat Commun*. 2015;6:6342.
72. Bakker MF, Peeters PH, Klaasen VM, et al. Plasma carotenoids, vitamin C, tocopherols, and retinol and the risk of breast cancer in the European Prospective Investigation into Cancer and Nutrition cohort. *Am J Clin Nutr*. 2016;103:454-464.
73. Eliassen AH, Liao X, Rosner B, Tamimi RM, Tworoger SS, Hankinson SE. Plasma carotenoids and risk of breast cancer over 20 y of follow-up. *Am J Clin Nutr*. 2015;101:1197-1205.
74. Rolls BJ, Ello-Martin JA, Tohill BC. What can intervention studies tell us about the relationship between fruit and vegetable consumption and weight management? *Nutr Rev*. 2004;62:1-17.
75. He FJ, Nowson CA, MacGregor GA. Fruit and vegetable consumption and stroke: meta-analysis of cohort studies. *Lancet*. 2006;367:320-326.
76. Lichtenstein AH, Appel LJ, Brands M, et al. Summary of American Heart Association Diet and Lifestyle Recommendations revision 2006. *Arterioscler Thromb Vasc Biol*. 2006;26:2186-2191.
77. Roger VL, Go AS, Lloyd-Jones DM, et al. Heart disease and stroke statistics—2011 update: a report from the American Heart Association. *Circulation*. 2011;123:e18-e209.
78. US Department of Agriculture. Choose-MyPlate. Accessed July 21, 2019. [choosemyplate.gov/ten-tips-choose-myplate](https://www.choosemyplate.gov/ten-tips-choose-myplate)
79. He FJ, Nowson CA, Lucas M, MacGregor GA. Increased consumption of fruit and vegetables is related to a reduced risk of coronary heart disease: meta-analysis of cohort studies. *J Hum Hypertens*. 2007;21:717-728.
80. Schwingshackl L, Schwedhelm C, Hoffmann G, et al. Food groups and risk of colorectal cancer. *Int J Cancer*. 2018;142:1748-1758.
81. Benisi-Kohansal S, Saneei P, Salehi-Marzijarani M, Larijani B, Esmailzadeh A. Whole-grain intake and mortality from all causes, cardiovascular disease, and cancer: a systematic review and dose-response meta-analysis of prospective cohort studies. *Adv Nutr*. 2016;7:1052-1065.
82. Sawicki CM, Livingston KA, Obin M, Roberts SB, Chung M, McKeown NM. Dietary fiber and the human gut microbiota: application of evidence mapping methodology. *Nutrients*. 2017;9:E125.
83. Cuevas-Sierra A, Ramos-Lopez O, Riezu-Boj JI, Milagro FI, Martinez JA. Diet, gut microbiota, and obesity: links with host genetics and epigenetics and potential applications. *Adv Nutr*. 2019;10(suppl 1):S17-S30.
84. Bonithon-Kopp C, Kronborg O, Giacosa A, Rath U, Faivre J. Calcium and fibre supplementation in prevention of colorectal adenoma recurrence: a randomised intervention trial. European Cancer Prevention Organisation Study Group. *Lancet*. 2000;356:1300-1306.
85. Alberts DS, Martinez ME, Roe DJ, et al. Lack of effect of a high-fiber cereal supplement on the recurrence of colorectal adenomas. Phoenix Colon Cancer Prevention Physicians' Network. *N Engl J Med*. 2000;342:1156-1162.
86. Bouvard V, Loomis D, Guyton KZ, et al. Carcinogenicity of consumption of red and processed meat. *Lancet*. 2015;16:1599-1600.
87. International Agency for Research on Cancer (IARC) Monographs Working Group. Consumption of Red Meat and Processed Meat. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Vol 114. World Health Organization/IARC; 2015.
88. Diallo A, Deschasaux M, Latino-Martel P, et al. Red and processed meat intake and cancer risk: results from the prospective NutriNet-Sante cohort study. *Int J Cancer*. 2018;142:230-237.
89. Inoue-Choi M, Sinha R, Gierach GL, Ward MH. Red and processed meat, nitrite, and heme iron intakes and postmenopausal breast cancer risk in the NIH-AARP Diet and Health Study. *Int J Cancer*. 2016;138:1609-1618.
90. Wu K, Spiegelman D, Hou T, et al. Associations between unprocessed red and processed meat, poultry, seafood and egg intake and the risk of prostate cancer: a pooled analysis of 15 prospective cohort studies. *Int J Cancer*. 2016;138:2368-2382.
91. Johnston BC, Zeraatkar D, Han MA, et al. Unprocessed red meat and processed meat consumption: dietary guideline recommendations from the Nutritional Recommendations (NutriRECS) Consortium. *Ann Intern Med*. Published online October 1, 2019. doi:10.7326/M19-1621
92. Han MA, Zeraatkar D, Guyatt GH, et al. Reduction of red and processed meat intake and cancer mortality and incidence: a systematic review and meta-analysis of cohort studies. *Ann Intern Med*. Published online October 1, 2019. doi:10.7326/M19-0699
93. Prentice RL, Aragaki AK, Howard BV, et al. Low-fat dietary pattern among postmenopausal women influences long-term cancer, cardiovascular disease, and diabetes outcomes. *J Nutr*. 2019;149:1565-1574.
94. Joosen AM, Kuhnle GG, Aspinall SM, et al. Effect of processed and red meat on

- endogenous nitrosation and DNA damage. *Carcinogenesis*. 2009;30:1402-1407.
95. Sinha R, Knize MG, Salmon CP, et al. Heterocyclic amine content of pork products cooked by different methods and to varying degrees of doneness. *Food Chem Toxicol*. 1998;36:289-297.
 96. Sinha R, Rothman N, Salmon CP, et al. Heterocyclic amine content in beef cooked by different methods to varying degrees of doneness and gravy made from meat drippings. *Food Chem Toxicol*. 1998;36:279-287.
 97. International Agency for Research on Cancer (IARC). World Cancer Report. Cancer Research for Cancer Prevention. WHO Press; 2014.
 98. Poti JM, Mendez MA, Ng SW, Popkin BM. Is the degree of food processing and convenience linked with the nutritional quality of foods purchased by US households? *Am J Clin Nutr*. 2015;101:1251-1262.
 99. Fiolet T, Srour B, Sellem L, et al. Consumption of ultra-processed foods and cancer risk: results from NutriNet-Sante prospective cohort. *BMJ*. 2018;360:k322.
 100. Aune D, Navarro Rosenblatt DA, Chan DS, et al. Dairy products, calcium, and prostate cancer risk: a systematic review and meta-analysis of cohort studies. *Am J Clin Nutr*. 2015;101:87-117.
 101. Institute of Medicine (US) Committee to Review Dietary Reference Intakes for Vitamin D and Calcium. Dietary Reference Intakes for Calcium and Vitamin D. National Academies Press; 2011.
 102. Feldman D, Krishnan AV, Swami S, Giovannucci E, Feldman BJ. The role of vitamin D in reducing cancer risk and progression. *Nat Rev Cancer*. 2014;14:342-357.
 103. Baron JA, Barry EL, Mott LA, et al. A trial of calcium and vitamin D for the prevention of colorectal adenomas. *N Engl J Med*. 2015;373:1519-1530.
 104. Wactawski-Wende J, Kotchen JM, Anderson GL, et al. Calcium plus vitamin D supplementation and the risk of colorectal cancer. *N Engl J Med*. 2006;354:684-696.
 105. Manson JE, Cook NR, Lee IM, et al. Vitamin D supplements and prevention of cancer and cardiovascular disease. *N Engl J Med*. 2019;380:33-44.
 106. Schleicher RL, Sternberg MR, Lacher DA, et al. The vitamin D status of the US population from 1988 to 2010 using standardized serum concentrations of 25-hydroxyvitamin D shows recent modest increases. *Am J Clin Nutr*. 2016;104:454-461.
 107. Gaziano JM, Sesso HD, Christen WG, et al. Multivitamins in the prevention of cancer in men: the Physicians' Health Study II randomized controlled trial. *JAMA*. 2012;308:1871-1880.
 108. Hercberg S, Galan P, Preziosi P, et al. The SU.VI.MAX Study: a randomized, placebo-controlled trial of the health effects of antioxidant vitamins and minerals. *Arch Intern Med*. 2004;164:2335-2342.
 109. Chen F, Du M, Blumberg JB, et al. Association among dietary supplement use, nutrient intake, and mortality among U.S. adults: a cohort study. *Ann Intern Med*. 2019;170:604-613.
 110. Kantor ED, Rehm CD, Du M, White E, Giovannucci EL. Trends in dietary supplement use among US adults from 1999-2012. *JAMA*. 2016;316:1464-1474.
 111. International Agency for Research on Cancer (IARC) Monographs Working Group. Alcoholic Beverages. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Vol 44. World Health Organization/IARC; 1988.
 112. International Agency for Research on Cancer (IARC). Monographs on the Evaluation of Carcinogenic Risks to Humans: Alcohol consumption and ethyl carbamate. Vol 96. Lyon, France: IARC Press; 2010.
 113. International Agency for Research on Cancer (IARC) Monographs Working Group. Personal Habits and Indoor Combustions. IARC Monographs on the Evaluation of Carcinogenic Risks to Humans. Vol 100E. World Health Organization/IARC; 2012.
 114. Seitz HK, Stickel F. Molecular mechanisms of alcohol-mediated carcinogenesis. *Nat Rev Cancer*. 2007;7:599-612.
 115. Seitz HK, Stickel F. Acetaldehyde as an underestimated risk factor for cancer development: role of genetics in ethanol metabolism. *Genes Nutr*. 2010;5:121-128.
 116. Substance Abuse and Mental Health Services Administration. Key Substance Use and Mental Health Indicators in the United States: Results from the 2016 National Survey on Drug Use and Health. Center for Behavioral Health Statistics and Quality, Substance Abuse and Mental Health Services Administration; 2017.
 117. Chartier K, Caetano R. Ethnicity and health disparities in alcohol research. *Alcohol Res Health*. 2010;33:152-160.
 118. World Health Organization. Updated Appendix 3 of the WHO Global NCD Action Plan 2013-2020. Accessed May 9, 2018. [who.int/ncds/governance/technical_annex.pdf](http://www.who.int/ncds/governance/technical_annex.pdf)
 119. Henley SJ, Kanny D, Roland KB, et al. Alcohol control efforts in comprehensive cancer control plans and alcohol use among adults in the USA. *Alcohol Alcohol*. 2014;49:661-667.
 120. GBD 2016 Alcohol Collaborators. Alcohol use and burden for 195 countries and territories, 1990-2016: a systematic analysis for the Global Burden of Disease Study 2016. *Lancet*. 2018;392:1015-1035.
 121. Trust for America's Health. The State of Obesity: Better Policies for a Healthier America. Robert Wood Johnson Foundation; 2018. Accessed July 21, 2019. tfah.org/report-details/the-state-of-obesity-better-policies-for-a-healthier-america/
 122. Krueger PM, Reither EN. Mind the gap: race/ethnic and socioeconomic disparities in obesity. *Curr Diab Rep*. 2015;15:95.
 123. Petersen R, Pan L, Blanck HM. Racial and ethnic disparities in adult obesity in the United States: CDC's tracking to inform state and local action. *Prev Chronic Dis*. 2019;16:E46.
 124. Division of Nutrition, Physical Activity, and Obesity; National Center for Chronic Disease Prevention and Health Promotion. Healthier Food Retail: An Action Guide for Public Health Practitioners. Centers for Disease Control and Prevention, Department of Health and Human Services; 2014.
 125. Bower KM, Thorpe RJ Jr, Rohde C, Gaskin DJ. The intersection of neighborhood racial segregation, poverty, and urbanicity and its impact on food store availability in the United States. *Prev Med*. 2014;58:33-39.
 126. Rone A, Ver Ploeg M. ERS's Updated Food Access Research Atlas Shows an Increase in Low-Income and Low-Supermarket Access Areas in 2015. *Amber Waves*. February 6, 2017.
 127. Darmon N, Drewnowski A. Contribution of food prices and diet cost to socioeconomic disparities in diet quality and health: a systematic review and analysis. *Nutr Rev*. 2015;73:643-660.
 128. Bluthenthal RN, Cohen DA, Farley TA, et al. Alcohol availability and neighborhood characteristics in Los Angeles, California and southern Louisiana. *J Urban Health*. 2008;85:191-205.
 129. LaVeist TA, Wallace JM Jr. Health risk and inequitable distribution of liquor stores in African American neighborhood. *Soc Sci Med*. 2000;51:613-617.
 130. Jones-Webb R, McKee P, Hannan P, et al. Alcohol and malt liquor availability and

- promotion and homicide in inner cities. *Subst Use Misuse*. 2008;43:159-177.
131. Moore H, Jones-Webb R, Toomey T, Lenk K. Alcohol advertising on billboards, transit shelters, and bus benches in inner-city neighborhoods. *Contemp Drug Probl*. 2008;35:509-532.
 132. Sallis JF, Floyd MF, Rodriguez DA, Saelens BE. Role of built environments in physical activity, obesity, and cardiovascular disease. *Circulation*. 2012;125:729-737.
 133. Smith M, Ikeda E, Hinckson E, et al. Results from New Zealand's 2018 report card on physical activity for children and youth. *J Phys Act Health*. 2018;15(suppl 2):S390-S392.
 134. The Community Guide. Increasing Physical Activity: Built Environment Approaches. Community Preventive Services Task Force; 2017. Accessed July 21, 2019. thecommunityguide.org/sites/default/files/assets/OnePager-Physical-Activity-built-environment.pdf
 135. Office of the Surgeon General (US). Step It Up! The Surgeon General's Call to Action to Promote Walking and Walkable Communities. Accessed July 21, 2019. ncbi.nlm.nih.gov/books/NBK538433/
 136. Centers for Disease Control and Prevention. Childhood Obesity Causes & Consequences. Accessed July 21, 2019. cdc.gov/obesity/childhood/causes.html
 137. Centers for Disease Control and Prevention. The Power of Prevention: Chronic Disease... The Public Health Challenge of the 21st Century. Centers for Disease Control and Prevention; 2009.
 138. World Health Organization. Tackling NCDs: 'Best Buys' and Other Recommended Interventions for the Prevention and Control of Noncommunicable Diseases. World Health Organization; 2017.
 139. McGuire S; Institute of Medicine. Accelerating Progress in Obesity Prevention: Solving the Weight of the Nation. Washington, DC: the National Academies Press. *Adv Nutr*. 2012;3:708-709.
 140. Mozaffarian D, Afshin A, Benowitz NL, et al. Population approaches to improve diet, physical activity, and smoking habits: a scientific statement from the American Heart Association. *Circulation*. 2012;126:1514-1563.
 141. ChangeLab Solutions. Building Healthier Communities. Accessed July 21, 2019. changelabsolutions.org/
 142. Trust for America's Health. The State of Obesity 2018: Better Policies for a Healthier America. Robert Wood Johnson Foundation; 2018. Accessed July 21, 2019. tfah.org/report-details/the-state-of-obesity-2018/
 143. The Community Guide. Physical Activity: Creating or Improving Places for Physical Activity. Community Preventive Services Task Force; 2001. Accessed July 21, 2019. thecommunityguide.org/content/physical-activity-creation-or-enhanced-access-places-physical-activity-combined
 144. National Physical Activity Plan Alliance. The US National Physical Activity Plan. National Physical Activity Plan Alliance; 2016.
 145. The Community Guide. Excessive Alcohol Consumption. Community Preventive Services Task Force; 2019. Accessed July 21, 2019. thecommunityguide.org/topic/excessive-alcohol-consumption?field_recommendation_tid=All&items_per_page=5
 146. Grant RW, Schmittiel JA, Neugebauer RS, Uratsu CS, Sternfeld B. Exercise as a vital sign: a quasi-experimental analysis of a health system intervention to collect patient-reported exercise levels. *J Gen Intern Med*. 2014;29:341-348.
 147. Golightly YM, Allen KD, Ambrose KR, et al. Physical activity as a vital sign: a systematic review. *Prev Chronic Dis*. 2017;14:E123.
 148. Nutrition & Obesity Network (NOPREN). Clinical Linkages. Accessed July 31, 2019. nopren.org/working_groups/food-security/clinical-linkages/
 149. US Preventive Services Task Force, Curry SJ, Krist AH, et al. Behavioral weight loss interventions to prevent obesity-related morbidity and mortality in adults: US Preventive Services Task Force recommendation statement. *JAMA*. 2018;320:1163-1171.
 150. US Preventive Services Task Force, Curry SJ, Krist AH, et al. Screening and behavioral counseling interventions to reduce unhealthy alcohol use in adolescents and adults: US Preventive Services Task Force recommendation statement. *JAMA*. 2018;320:1899-1909.
 151. Anderson CAM, Thorndike AN, Lichtenstein AH, et al. Innovation to create a healthy and sustainable food system: a science advisory from the American Heart Association. *Circulation*. 2019;139:e1025-e1032.
 152. Pate RR, Flynn JI, Dowda M. Policies for promotion of physical activity and prevention of obesity in adolescence. *J Exerc Sci Fit*. 2016;14:47-53.
 153. Mozaffarian D, Liu J, Sy S, et al. Cost-effectiveness of financial incentives and disincentives for improving food purchases and health through the US Supplemental Nutrition Assistance Program (SNAP): a microsimulation study. *PLoS Med*. 2018;15:e1002661.
 154. Hayes D, Contento IR, Weekly C. Position of the Academy of Nutrition and Dietetics, Society for Nutrition Education and Behavior, and School Nutrition Association: comprehensive nutrition programs and services in schools. *J Acad Nutr Diet*. 2018;118:913-919.
 155. Bleich SN, Economos CD, Spiker ML, et al. A systematic review of calorie labeling and modified calorie labeling interventions: impact on consumer and restaurant behavior. *Obesity (Silver Spring)*. 2017;25:2018-2044.
 156. Chaloupka FJ, Powell LM, Warner KE. The use of excise taxes to reduce tobacco, alcohol, and sugary beverage consumption. *Annu Rev Public Health*. 2019;40:187-201.
 157. Falbe J, Thompson HR, Becker CM, Rojas N, McCulloch CE, Madsen KA. Impact of the Berkeley excise tax on sugar-sweetened beverage consumption. *Am J Public Health*. 2016;106:1865-1871.
 158. Purtle J, Langellier B, Le-Scherban F. A case study of the Philadelphia sugar-sweetened beverage tax policymaking process: implications for policy development and advocacy. *J Public Health Manag Pract*. 2018;24:4-8.
 159. Lipworth L, Sonderman JS, Tarone RE, McLaughlin JK. Review of epidemiologic studies of dietary acrylamide intake and the risk of cancer. *Eur J Cancer Prev*. 2012;21:375-386.
 160. Virk-Baker MK, Nagy TR, Barnes S, Groopman J. Dietary acrylamide and human cancer: a systematic review of literature. *Nutr Cancer*. 2014;66:774-790.
 161. Pelucchi C, Bosetti C, Galeone C, La Vecchia C. Dietary acrylamide and cancer risk: an updated meta-analysis. *Int J Cancer*. 2015;136:2912-2922.
 162. World Health Organization. Arsenic Fact Sheet. World Health Organization; 2018.
 163. National Toxicology Program. Arsenic and Inorganic Arsenic Compounds, Report on Carcinogen. 14th ed. US Department of Health and Human Services, Public Health Service; 2016.
 164. US Geological Survey, Department of the Interior. Arsenic and Drinking Water. Accessed July 21, 2019. usgs.gov/mission-areas/water-resources/science/arsenic-and-drinking-water
 165. Ghose N, Majumdar KK, Ghose AK, Saha CK, Nandy AK, Mazumder DN. Role of folic acid on symptoms of chronic arsenic toxicity. *Int J Prev Med*. 2014;5:89-98.
 166. International Agency for Research on Cancer (IARC) Monographs Working Group. Drinking Coffee, Mate, and Very

- Hot Beverages. IARC Monographs on the Evaluation of Carcinogenic Risk to Humans. Vol 116. World Health Organization/IARC; 2016.
167. Okaru AO, Rullmann A, Farah A, Gonzalez de Mejia E, Stern MC, Lachenmeier DW. Comparative oesophageal cancer risk assessment of hot beverage consumption (coffee, mate and tea): the margin of exposure of PAH vs very hot temperatures. *BMC Cancer*. 2018;18:236.
 168. US Food and Drug Administration. Questions & Answers on Food from Genetically Engineered Plants, 2018. July 21, 2019. [fda.gov/food/food-new-plant-varieties/questions-answers-food-genetically-engineered-plants](https://www.fda.gov/food/food-new-plant-varieties/questions-answers-food-genetically-engineered-plants)
 169. US Food and Drug Administration. Labeling of Foods Derived from Genetically Engineered Plants, 2018. July 21, 2019. [fda.gov/food/food-new-plant-varieties/labeling-foods-derived-genetically-engineered-plants](https://www.fda.gov/food/food-new-plant-varieties/labeling-foods-derived-genetically-engineered-plants)
 170. Schlesinger S, Chan DSM, Vingeliene S, et al. Carbohydrates, glycemic index, glycemic load, and breast cancer risk: a systematic review and dose-response meta-analysis of prospective studies. *Nutr Rev*. 2017;75:420-441.
 171. Kaluza J, Hakansson N, Harris HR, Orsini N, Michaelsson K, Wolk A. Influence of anti-inflammatory diet and smoking on mortality and survival in men and women: two prospective cohort studies. *J Intern Med*. 2019;285:75-91.
 172. Giovannucci E. A framework to understand diet, physical activity, body weight, and cancer risk. *Cancer Causes Control*. 2018;29:1-6.
 173. Ravindran R, Jaiswal AK. Wholesomeness and safety aspects of irradiated foods. *Food Chem*. 2019;285:363-368.
 174. Butalla AC, Crane TE, Patil B, Wertheim BC, Thompson P, Thomson CA. Effects of a carrot juice intervention on plasma carotenoids, oxidative stress, and inflammation in overweight breast cancer survivors. *Nutr Cancer*. 2012;64:331-341.
 175. Hu C; UCLA Center for East-West Medicine. Juicing: body cleansing or nutrient depleting? Accessed July 21, 2019. exploreim.ucla.edu/wellness/juicing-body-cleansing-or-nutrient-depleting/#-Cleansing2019
 176. Lien YH. Juicing is not all juicy. *Am J Med*. 2013;126:755-756.
 177. Tavernise S. Misconception: juice cleansing can remove toxins from your system. *New York Times*. April 20, 2016;§D:2. Accessed April 20, 2016. [nytimes.com/2016/04/21/health/juice-cleanse-toxin-misconception.html?searchResultPosition=3](https://www.nytimes.com/2016/04/21/health/juice-cleanse-toxin-misconception.html?searchResultPosition=3)
 178. National Cancer Institute. Electromagnetic Fields and Cancer Fact Sheet. Accessed July 21, 2019. [cancer.gov/about-cancer/causes-prevention/risk/radiation/electromagnetic-fields-fact-sheet](https://www.cancer.gov/about-cancer/causes-prevention/risk/radiation/electromagnetic-fields-fact-sheet)
 179. Harley KG, Berger KP, Kogut K, et al. Association of phthalates, parabens and phenols found in personal care products with pubertal timing in girls and boys. *Hum Reprod*. 2019;34:109-117.
 180. Wolff MS, Pajak A, Pinney SM, et al. Associations of urinary phthalate and phenol biomarkers with menarche in a multiethnic cohort of young girls. *Reprod Toxicol*. 2017;67:56-64.
 181. Wolff MS, Teitelbaum SL, Pinney SM, et al. Investigation of relationships between urinary biomarkers of phytoestrogens, phthalates, and phenols and pubertal stages in girls. *Environ Health Perspect*. 2010;118:1039-1046.
 182. National Cancer Institute. Artificial Sweeteners and Cancer. Accessed November 16, 2016. [cancer.gov/about-cancer/causes-prevention/risk/diet/artificial-sweeteners-fact-sheet](https://www.cancer.gov/about-cancer/causes-prevention/risk/diet/artificial-sweeteners-fact-sheet)
 183. Baudry J, Assmann KE, Touvier M, et al. Association of frequency of organic food consumption with cancer risk: findings from the NutriNet-Sante prospective cohort study. *JAMA Intern Med*. 2018;178:1597-1606.
 184. Bradbury KE, Balkwill A, Spencer EA, et al. Organic food consumption and the incidence of cancer in a large prospective study of women in the United Kingdom. *Br J Cancer*. 2014;110:2321-2326.
 185. Hemler EC, Chavarro JE, Hu FB. Organic foods for cancer prevention—worth the investment? *JAMA Intern Med*. 2018;178:1606-1607.
 186. Palamaner Subash Shantha G, Kumar AA, Cheskin LJ, Pancholy SB. Association between sleep-disordered breathing, obstructive sleep apnea, and cancer incidence: a systematic review and meta-analysis. *Sleep Med*. 2015;16:1289-1294.
 187. McHill AW, Wright KP Jr. Role of sleep and circadian disruption on energy expenditure and in metabolic predisposition to human obesity and metabolic disease. *Obes Rev*. 2017;18(suppl 1):15-24.
 188. Thomson CA, Morrow KL, Flatt SW, et al. Relationship between sleep quality and quantity and weight loss in women participating in a weight-loss intervention trial. *Obesity (Silver Spring)*. 2012;20:1419-1425.
 189. Creasy SA, Crane TE, Garcia DO, et al. Higher amounts of sedentary time are associated with short sleep duration and poor sleep quality in postmenopausal women. *Sleep*. 2019;42:zsz093.
 190. Setchell KDR. The history and basic science development of soy isoflavones. *Menopause*. 2017;24:1338-1350.
 191. Chen M, Rao Y, Zheng Y, et al. Association between soy isoflavone intake and breast cancer risk for pre- and post-menopausal women: a meta-analysis of epidemiological studies. *PLoS One*. 2014;9:e89288.
 192. Applegate CC, Rowles JL, Ranard KM, Jeon S, Erdman JW. Soy consumption and the risk of prostate cancer: an updated systematic review and meta-analysis. *Nutrients*. 2018;10:E40.
 193. Touillaud M, Gelot A, Mesrine S, et al. Use of dietary supplements containing soy isoflavones and breast cancer risk among women aged >50 y: a prospective study. *Am J Clin Nutr*. 2019;109:597-605.
 194. Li N, Wu X, Zhuang W, et al. Soy and isoflavone consumption and multiple health outcomes: umbrella review of systematic reviews and meta-analyses of observational studies and randomized trials in humans. *Mol Nutr Food Res*. Published online October 4, 2019. doi:10.1002/mnfr.201900751
 195. Melina V, Craig W, Levin S. Position of the Academy of Nutrition and Dietetics: Vegetarian Diets. *J Acad Nutr Diet*. 2016;116:1970-1980.
 196. Key TJ, Appleby PN, Crowe FL, Bradbury KE, Schmidt JA, Travis RC. Cancer in British vegetarians: updated analyses of 4998 incident cancers in a cohort of 32,491 meat eaters, 8612 fish eaters, 18,298 vegetarians, and 2246 vegans. *Am J Clin Nutr*. 2014;100(suppl 1):378S-385S.