Cardiovascular Disease Prevention by Diet Modification

JACC Health Promotion Series

Edward Yu, ScD,[a,b] Vasanti S. Malik, ScD,[a] Frank B. Hu, MD, PhD[a,b,c]

ABSTRACT

Reduction in excess calories and improvement in dietary composition may prevent many primary and secondary cardiovascular events. Current guidelines recommend diets high in fruits, vegetables, whole grains, nuts, and legumes; moderate in low-fat dairy and seafood; and low in processed meats, sugar-sweetened beverages, refined grains, and sodium. Supplementation can be useful for some people but cannot replace a good diet. Factors that influence individuals to consume a low-quality diet are myriad and include lack of knowledge, lack of availability, high cost, time scarcity, social and cultural norms, marketing of poor-quality foods, and palatability. Governments should focus on cardiovascular disease as a global threat and enact policies that will reach all levels of society and create a food environment wherein healthy foods are accessible, affordable, and desirable. Health professionals should be proficient in basic nutritional knowledge to promote a sustainable pattern of healthful eating for cardiovascular disease prevention for both healthy individuals and those at higher risk. (J Am Coll Cardiol 2018;72:914–26) © 2018 by the American College of Cardiology Foundation.

Behavior modification is a key strategy that may prevent a large number of primary and secondary cardiovascular events (1). Suboptimal diet was responsible for an estimated 1 in 5 premature deaths globally from 1990 to 2016 (2).

Observational studies of human diet and health outcomes are challenging due to difficulties in measuring dietary intakes (3) and potential problems with generalizability and confounding (4). Although randomized trials provide stronger potential for causal inference, they typically have small sample sizes, short durations of follow-up, noncompliance, high attrition rates, and ethical constraints (5). Thus, current dietary recommendations are based on a combination of human observational and intervention trial evidence supplemented by findings from mechanistic studies (6).

In the present review, we first summarize the current state of knowledge regarding various food groups and nutrients. Subsequent sections explore factors driving individual food choice, where preventive action can be implemented, and what potential roadblocks may hinder progress.

PATHOPHYSIOLOGICAL EFFECTS OF DIETARY COMPONENTS

The **Central Illustration** demonstrates the prevention of cardiovascular disease (CVD) and disease risk factors through a healthy eating pattern. Current evidence suggests that the impact of dietary composition is relatively consistent for primordial, primary, and secondary prevention of CVD with certain dietary factors that reduce CVD incidence also being important for secondary prevention among myocardial infarction (MI) survivors.

EXCESS CALORIC INTAKE. Healthy eating is based on maintaining caloric balance. A large body of published reports supports calorie restriction for cardiometabolic benefit, specifically for improvements in insulin sensitivity, blood glucose, and inflammation (7). Chronic positive energy balance leads to overweight and obesity, the details of which are discussed in a separate paper in this series. For most people, significant and sustained weight loss through dieting is extremely difficult, and the majority of weight loss trials feature high degrees of dropout and noncompliance due to the difficulty of long-term caloric restriction (8). Emerging evidence suggests that dietary composition and overall diet quality are important for minimizing overconsumption, and that low-carbohydrate and Mediterranean diets are superior to low-fat diets in maintaining weight loss (9). Some, but not all, trials that examine macronutrient composition for weight loss reported greater long-term benefit for individuals consuming higher amounts of protein and fat compared with those who consumed higher amounts of carbohydrates (10–12).

ABBREVIATIONS AND ACRONYMS

AHEI = Alternative Healthy Eating Index

CHD = coronary heart disease

CI = confidence interval

CVD = cardiovascular disease

HR = hazard ratio

MI = myocardial infarction

SSB = sugar-sweetened beverage

FOODS AND FOOD GROUPS. Figure 1 shows the summary estimates from various meta-analyses of key individual foods and food groups, and dietary patterns with CVD. The failure of most supplementation trials to detect significant reductions in risk among healthy populations (13) has led to dietary recommendations primarily based on eating whole food items and maintaining high-quality diets.

Intake of total fruits and vegetables has been inversely associated with CVD risk (14). However, benefits for subgroups have been less studied, and may vary considerably. Various phytochemicals and micronutrients such as folate, potassium, fiber, and flavonoids found in fruits and vegetables are hypothesized to be responsible for the observed benefits (15). Potatoes have been viewed with skepticism due to their high starch content; higher potato intake, especially from French fries, has been associated with greater risk of hypertension, type 2 diabetes, and coronary heart disease (CHD) risk (16–18).

Whole grain intake is associated with a substantially lower risk of CVD, whereas refined grain intake is suggestive of an increased, but nonsignificant, association (19). The bran and germ layers, present in whole grains, but removed from refined grains, are rich in fiber, lignans, micronutrients, fatty acids, and other phytonutrients (20). Depletion of these nutrients during the milling process partially explains why whole grain consumption is generally related to higher satiety and a lower glycemic response compared with refined grains (21).

Marine fish is rich in long-chain omega-3 fatty acids, which are thought to reduce arrhythmias, thrombosis, inflammation, blood pressure, as well as favorably modify the lipid profile (22). A meta-analysis suggests that a 15-g/day increment in fish intake is associated with a hazard ratio (HR) of 0.96 (95% confidence interval [CI]: 0.90 to 0.98) for CHD mortality (23).

Nuts and legumes are beneficial through their high unsaturated fat, fiber, micronutrient, and phytochemical content (24). A meta-analysis of 25 observational studies found that a 4 serving/week increase in nut intake was associated with a HR of 0.76 (95% CI: 0.69 to 0.84) for fatal CHD and a HR of 0.78 (95% CI: 0.67 to 0.92) for nonfatal CHD (24). Small intervention studies have reported lower total cholesterol, low-density lipoprotein cholesterol, apolipoprotein B, and triglycerides among those randomized to consuming tree nuts compared with the control arms (25).

Dairy products have shown null or weakly inverse associations with CVD. For example, fermented dairy (i.e., sour milk products, cheese, yogurt) showed a HR per 20 g/day of 0.98 (95% CI: 0.97 to 0.99) (26) with similar associations observed across different dairy products. Similar associations have also been observed for total dairy and type 2 diabetes (HR per 200 g/day: 0.97; 95% CI: 0.95 to 1.00) and yogurt (HR per 80 g/day: 0.86; 95% CI: 0.83 to 0.90) (27). Potential benefits of fermented dairy may be due to its probiotic content (28).

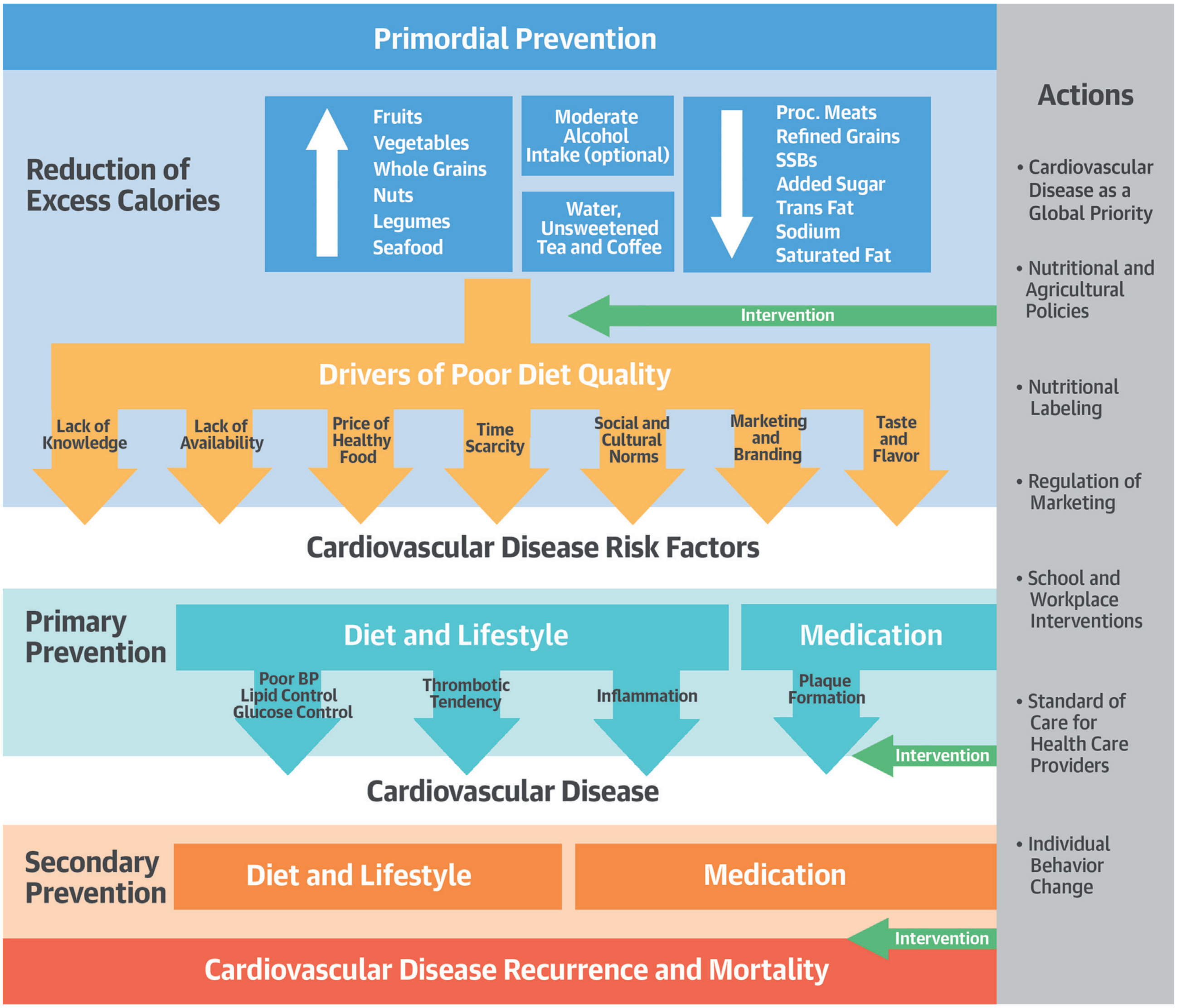
Intake of processed meat (i.e., hamburgers, hot dogs, deli meats) has been shown to increase the risk of CVD in a robust linear fashion (29,30). Higher consumption of unprocessed red meat has also been associated with increased risk of CVD mortality (29). Replacing processed and unprocessed red meat with other sources of protein such as fish, poultry, and nuts was associated with lower incidence of CHD (31). Low-carbohydrate diets high in animal protein and fat were associated with higher risk of total and cardiovascular death among MI survivors (32). Important bioactive molecules in red meat include heme iron, sodium, nitrates, and L-carnitine that may lead to significant elevations in blood pressure, worsening oxidative stress, greater lipid peroxidation, and unfavorable alterations of the gut microbiome (33,34).

BEVERAGES. Alcohol is related to CVD risk in a U-shaped relationship, with both abstainers and heavy drinkers having an increased risk compared with moderate drinkers (35). The exact nadir of risk differs according to age, sex, ethnicity, and baseline disease (36), but the consistent observation is that individuals who consume 1 to 2 drinks a day have the lowest risk (37). Moderate alcohol intake has been shown to increase high-density lipoprotein cholesterol, apolipoprotein A1, and adiponectin, and decrease fibrinogen levels (38,39).

Higher consumption of sugar-sweetened beverages (SSBs) has been associated with risk of CVD in a dose-dependent relationship (40). This association is partially mediated by an increase in body weight; high intake of liquid calories does not seem to reduce later intake of solid foods (41). Independent of weight change, intake of SSBs increases postprandial blood glucose and insulin concentrations through a high glycemic load, as well as confer adverse effects on fat deposition, lipid metabolism, blood pressure, insulin sensitivity, and lipogenesis (42).

Regular consumption of coffee has been consistently associated with lower risk of CVD, with the greatest risk reduction occurring at around 3 to 5 cups per day conferring an 11% lower risk (43). Biological mechanisms for the cardioprotective effects of moderate coffee consumption include a high concentration of chlorogenic acid, micronutrients,

CENTRAL ILLUSTRATION **Flow Diagram of the Development of CVD and Possible Prevention by a Healthy Diet**



Yu, E. et al. J Am Coll Cardiol. 2018;72(8):914–26.

Avoiding excess calories is an integral part of halting the development of cardiovascular disease risk factors (i.e., primordial prevention). Unfavorable eating patterns are driven by a variety of biological, social, economic, and psychological factors, and a robust intervention from all levels of society may steer populations toward a healthier diet and prevent disease progression. Diet and other lifestyle changes remain crucial steps in primary and secondary prevention of cardiovascular disease, although the relative importance of medication and clinical procedures increases over time with disease progression. CVD = cardiovascular disease; SSB = sugar-sweetened beverage.

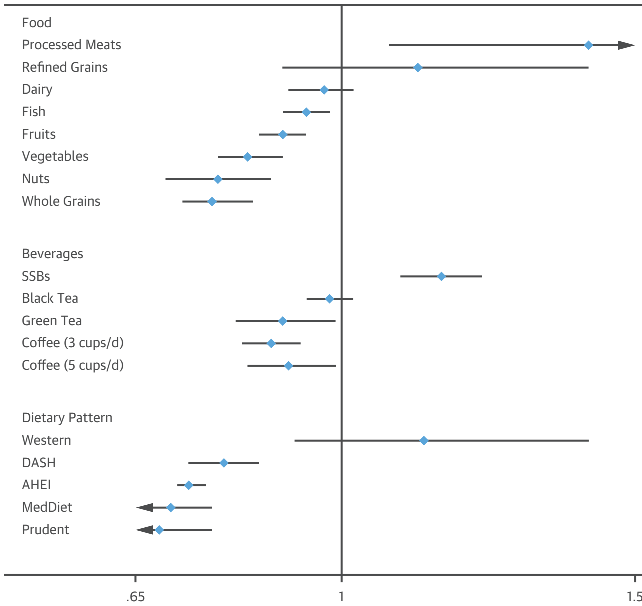
lignans, and phytochemicals. Short-term trials of coffee report higher insulin sensitivity and a favorable inflammatory marker profile, but excess intake (>8 cups/day) may lead to acute elevations in blood pressure (44).

Tea has likewise been reported to be inversely associated with CVD incidence (45). Tea flavonoids, specifically flavonols, have received considerable attention and are themselves independently associated with reduced CVD risk (46).

DIETARY PATTERNS AND DIET QUALITY. Dietary patterns and quality are the most comprehensive metrics of assessing eating habits and include indices based on a priori scoring, such as the Alternative Mediterranean diet score, Alternative Healthy Eating

JACC VOL. 72, NO. 8, 2018
AUGUST 21, 2018:914–26

FIGURE 1 Summary of Various Meta-Analyses for the Associations of Key Foods and Food Groups, and Dietary Patterns With Incident CVD



Dietary Component	RR (95% CI)	Reference	No. of Studies	Analysis Unit	Outcome
Food					
Processed Meats	1.42 (1.08, 1.89)	Micha 2012	7	Per 50 g/d	CHD Incidence
Refined Grains	1.13 (0.90, 1.42)	Aune 2016	4	Per 90 g/d	CHD Incidence
Dairy	0.97 (0.91, 1.02)	Guo 2017	8	Per 200 g/d	CVD Incidence
Fish	0.94 (0.90, 0.98)	Zheng 2012	17	Per 15 g/d	CHD Mortality
Fruits	0.90 (0.86, 0.94)	Aune 2017	24	Per 200 g/d	CVD Incidence
Vegetables	0.84 (0.79, 0.90)	Aune 2017	20	Per 200 g/d	CVD Incidence
Nuts	0.79 (0.70, 0.88)	Aune 2016	12	Per 28 g/d	CVD Incidence
Whole Grains	0.78 (0.73, 0.85)	Aune 2016	10	Per 90 g/d	CVD Incidence
Beverages					
SSBs	1.17 (1.10, 1.24)	Xi 2015	4	Per serv/d	CHD Incidence
Black Tea	0.98 (0.94, 1.02)	Wang 2011	13	Per 1 cup/d	CHD Incidence
Green Tea	0.90 (0.82, 0.99)	Wang 2011	5	Per 1 cup/d	CHD Incidence
Coffee (3 cups/d)	0.88 (0.83, 0.93)	Ding 2014	36	vs. nondrinkers	CVD Incidence
Coffee (5 cups/d)	0.91 (0.84, 0.99)	Ding 2014	36	vs. nondrinkers	CVD Incidence
Dietary Pattern					
Western	1.14 (0.92, 1.42)	Rodriguez-Monforte 2015	5	Extreme categories	CVD Incidence
DASH	0.80 (0.74, 0.86)	Salehi-Abargouei 2013	6	Extreme categories	CVD Incidence
AHEI	0.74 (0.72, 0.77)	Schwingshackl 2015	6	Extreme categories	CVD Incidence
MedDiet	0.71 (0.65, 0.78)	Grosso 2015	13	Extreme categories	CVD Incidence
Prudent	0.69 (0.60, 0.78)	Rodriquez-Monforte 2015	5	Extreme categories	CVD Incidence

High amounts of processed meat, SSBs, and refined grain consumption are associated with greater CVD incidence; moderate coffee and alcohol intake, and high fruit/vegetable, dairy (low-fat), whole grain, fish, and nut intake are associated with lower incidence. High adherence to Mediterranean, DASH, AHEI, and Prudent dietary patterns are significantly predictive of lower CVD incidence. AHEI = Alternative Healthy Eating Index; CHD = coronary heart disease; CI = confidence interval; CVD = cardiovascular disease; DASH = Dietary Approaches to Stop Hypertension; MedDiet = Mediterranean diet; RR = risk ratio; SSB = sugar-sweetened beverage.

Index (AHEI), and DASH (Dietary Approaches to Stop Hypertension) diet score, as well as exploratory methods including principal component analysis and cluster analysis (47). Holistic evaluation of the diet is useful because it captures potential food and nutrient interactions that studies of single nutritional items cannot (48).

Individual diet indices differ in their components and weighting, but most emphasize high intake of fruits and vegetables, whole grains, nuts; moderate intake of low/nonfat dairy and alcohol; and low intake of sodium, processed meats, added sugar, and saturated fat (49). In the WHI (Women's Health Initiative) study, high Healthy Eating Index, AHEI, Alternative Mediterranean diet, and DASH scores were consistently associated with around a 20% reduction in CVD mortality (50). Sotos-Prieto et al. (51,52) reported that improvement in these scores was also associated with lower risk of total and CVD mortality in 2 large cohorts. Similar findings have been observed among MI survivors. Li et al. (53) found that a greater increase in the AHEI score from pre- to post-MI was significantly associated with lower all-cause and cardiovascular mortality, and Lopez-Garcia et al. (54) found that adherence to a Mediterranean-style dietary pattern was associated with lower all-cause mortality among individuals with CVD.

Principal component and factor analyses have generally identified 2 dietary patterns that explain most of the variation in population-level eating habits: prudent and Western. Prudent diets are rich in fruits, vegetables, legumes, whole grains, fish, and poultry, whereas Western diets include high amounts of processed meat, French fries, desserts, SSBs, red meat, and high-fat dairy (55). A meta-analysis of 22 cohort studies found that those in the highest category of adherence to a prudent diet had a 31% lower risk of CVD compared with those with the lowest adherence (56), whereas a Western dietary pattern was associated with a 14% increase in risk (56). The consistency of findings from cohort studies across many countries for various dietary factors and indices, and similar findings from intervention trials support the causal role of a high-quality diet in CVD prevention (57,58).

CARBOHYDRATES. Both quality and quantity of carbohydrates are important in a healthy eating pattern. Diets high in glycemic index and glycemic load (metrics that rate foods based on the magnitude of postprandial glucose level) have been associated with a higher risk of CHD, whereas diets low in glycemic index or load have been inversely associated with CHD (59). In the Nurses' Health Study and Health Professionals Follow-Up Study, a greater adherence to a low-carbohydrate diet with higher amounts of plant-based fats and protein was associated with lower all-cause and cardiovascular mortality among generally healthy individuals (60) and among MI survivors (32). However, greater adherence to a low-carbohydrate diet high in animal sources of fat and protein was associated with higher all-cause and cardiovascular mortality among healthy individuals (60).

Added sugars such as sucrose and high fructose corn syrup derived from industrial processes have been associated with a significant increase in CVD risk (61), with the greatest source and majority of evidence originating from SSBs (includes soda, flavored fruit juices, sports drinks, and energy drinks), which account for 6.9% of daily calories in the United States (62). Higher consumption of added sugars appears to increase risk of CVD independent of body weight or other dietary components (63), likely through lowered high-density lipoprotein cholesterol (64), increased plasma triglyceride concentration (65), and higher blood pressure (66).

Dietary fiber has been consistently demonstrated to lower risk of CVD and improve cardiovascular risk factors in both observational (67) and dietary intervention studies (68,69). In a meta-analysis of 22 cohorts, a 7-g/day increase in fiber intake was associated with a 9% decrease in CHD incidence (67). Intake of fiber, particularly cereal fiber, has also been shown to reduce all-cause mortality among MI survivors, with a 27% (HR: 0.73; 95% CI: 0.58 to 0.91) reduction in risk of death in the highest compared with the lowest quintile of cereal fiber intake (70). It is thought that the cardioprotective action of fiber operates through decreased low-density lipoprotein cholesterol, decreased serum triglycerides, blunted postprandial glucose response (71), and changes in bile acid metabolism (72).

DIETARY FAT. Of the 3 primary types of dietary fat—trans fatty acids, saturated fatty acids, and unsaturated (includes mono- and polyunsaturated fats) fatty acids—trans fatty acids have been most strongly associated with adverse cardiovascular outcomes (73), and its ban in the United States is one of the greatest success stories in public health, the details of which are discussed later in the review. Among the other types of fat, saturated fatty acids have received the most controversy. Higher intake of saturated fat has been found to be either harmful or neutral for CVD risk in most meta-analyses (74). One explanation for the inconsistent findings is that most observational studies did not specify a comparison or replacement macronutrient for saturated fat, leaving carbohydrates (primarily from refined grains and added sugar) as the default comparison macronutrient.

Analyses that employed substitution models reported that substituting either carbohydrates or saturated fats with unsaturated fats was associated with a lower risk of CVD, with polyunsaturated fat showing a consistently larger magnitude of benefit (75). Supplementation with long-chain omega-3 fatty acids, a type of polyunsaturated fat derived primarily from fish oil, has shown mixed results (76,77), and the potential benefits of omega-3 fatty acids on reductions in sudden cardiac death remain to be confirmed. Long-chain omega-3 fatty acids have been shown to maintain cell membrane fluidity, reduce blood viscosity and clotting tendency, and promote the formation of anti-inflammatory mediators (78,79).

FACTORS INFLUENCING FOOD CHOICE

Despite the immeasurable gains that researchers have made in understanding *what* constitutes a healthy diet, less attention has been given to understanding *why* people eat (or do not eat) a healthy diet. Eating habits are forged over a lifetime and are influenced by a multitude of factors from all levels of society including biological, economic, physical, social, and psychological determinants (80). The assumption that most people would replace unhealthy dietary components in light of new scientific evidence is overly optimistic (80,81). Well-known randomized trials of diet, such as the WHI study, have not been successful in achieving target macronutrient compositions or sustaining them after 6 months despite targeted behavioral intervention and in the case of the WHI study, unrealistic goals for low fat intake (82,83). By contrast, the PREDIMED (PREvención con DIeta MEDiterránea) trial, which evaluated the effects of a Mediterranean diet versus a low-fat control diet, achieved and sustained intervention goals over 4 years of follow-up (84,85), largely because olive oil and nuts were provided to participants.

Lack of nutrition knowledge has been suggested as a contributor to poor diet (86), particularly among low-income or minority populations (87,88), and in low-income countries (89,90), where access to education is limited. However, most individuals in high-income countries appear to possess a reasonable level of nutritional knowledge, with elements such as fruits and vegetables being widely recognized as healthy and highly processed grain products, added sugar, and salt as unhealthy (91). In a large European study of 14,331 participants, lack of knowledge was not cited as a common barrier to healthy eating (92).

On the other hand, lack of availability of healthful foods has been identified as a potential driver of unhealthy eating. "Food deserts" refers to areas with long distances to supermarkets and low access to fresh foods, whereas "food swamps" refers to areas with an abundance of unhealthy processed and fast foods (93,94). This simultaneous availability of cheap, low-quality food and expensive or lack of availability of high-quality food can drive individuals to choose unhealthy eating options (95,96). These elements together create an obesogenic environment that can lead to excess adiposity and subsequent cardiometabolic disease (97).

Price is an important roadblock to better eating. Rao et al. (98) reported in a recent meta-analysis that the healthiest diets cost approximately $1.50 a day, or about $550 more a year, than the unhealthiest diets defined by various dietary indices. Time scarcity has also been shown to promote poor food choice (99) and is a major factor in the decline of home cooking in recent decades (100). Eating out is a significant predictor of overconsumption and lower micronutrient intake (101). Bernstein et al. (102) suggest that affordable and healthy plant-based diets are achievable with proper knowledge and preparation time.

Palatability is an obvious, but underappreciated, determinant of diet. Human attraction to sweet and savory foods is rooted in evolutionary and anthropologic processes (103), a fact that food companies have exploited by adding large amounts of sugar and sodium to most processed products (104,105). A prominent example of this practice is with SSBs, where high amounts of added sugar coupled with the inability of liquid calories to trigger satiety may have contributed to the obesity epidemic and cardiometabolic risk in the United States (106–108).

Branding and marketing are also major factors that influence both taste and choice. Advertising has long been known to affect taste, possibly by linking positive sensory thoughts with the target product (109). Regulations on food branding and restriction of advertising to children have also been proposed as ways to improve diet quality and reduce obesity (110,111).

Social determinants of food choice include influences of culture, friends/family, and community. Social norms have powerful influences on eating patterns, and those healthy food norms can result in healthier food choices (112). Sacks et al. (82) reported that the number of support sessions attended during a trial was the strongest predictor of weight loss at 2 years (0.2 kg for every session attended) regardless of macronutrient composition. Recent analyses indicate that food choices tend to be shared among family members (113), and that alcohol drinking and snacking were the most "transmissible" eating patterns (114).

ROLE OF PREVENTIVE ACTION

Given the magnitude of the CVD burden in the United States and globally, and the complexity of dietary risk factor modification, simultaneous prevention strategies and policies across multiple societal levels are needed to make a measurable impact on reducing prevalence rates. In contrast to clinical decision making where the evidence base is dominated by randomized clinical trials and large cohort studies, there is a paucity of data evaluating preventive actions to improve diet. Thus, to gauge the effectiveness of prevention strategies, we also consider different types of evidence such as natural experiments and simulation models, and discuss actions that have great potential for benefit and scalability that represent important knowledge gaps.

SOCIETAL/AUTHORITATIVE. Nutrition and agricultural policies are powerful instruments for reducing CVD risk if they align with evidence-based dietary goals to improve diet quality. One example is nutrition labeling of industrially produced trans fats and legislation for removal of trans fats from the food supply, which was recently enacted in the United States with the removal of trans fats from the Food and Drug Administration's generally regarded as safe category. This action, which was implemented in June 2018, is expected to reduce as many as 20,000 coronary events and 7,000 deaths from coronary causes each year in the United States (115).

Some governments are considering taxing select foods and beverages, particularly SSBs, as a means to improve consumer choice and generate revenue. Whether these programs will have the desired effect is yet to be determined. Some studies have suggested that for such interventions to have an appreciable impact, tax increases of at least 10% are needed (116). In Mexico, a peso-per-liter (roughly $0.80 per liter) tax on SSBs enacted in 2014 has resulted in an average reduction in sales of 7.6% of taxed beverages 2 years after implementation. Households at the lowest socioeconomic level had the largest decreases in purchases of taxed beverages over this time, and purchases of untaxed beverages increased 2.1% (117). To date, at least 8 cities in the United States have enacted an SSB tax along with a number of countries including Mexico, Chile, France, Norway, Finland, the United Kingdom, and Hungary. Careful evaluation will be key in determining the effectiveness of these strategies on reducing intake of these beverages and subsequently on reducing prevalence of obesity and cardiometabolic disease.

Other pricing policies such as agricultural subsidies to increase accessibility and affordability of fruits, vegetables, legumes, nuts, and whole grains should also be emphasized. In parts of the United States, access to fruit and vegetables has been shown to differ by race and socioeconomic status (118). Amending the U.S. farm bill, the primary agricultural policy tool in the United States, could be an effective way to improve diet quality at the population level (119). In particular, this includes amending the Supplemental Nutrition Assistance Program, which provides US$75 billion per year in subsidies to 47 million U.S. citizens that can be used for the purchase of SSBs and other foods and beverages that adversely affect health (120).

Government regulation of school lunch programs has the potential to improve diet quality of children on a large scale. In 2012, the nutrition standards of federally assisted meal programs were updated for the first time in 15 years to reduce sodium, saturated fat, and trans fats, and increase fruits, vegetables, and whole grains, largely based on recommendations by the Institute of Medicine of the National Academies, as part of their efforts to curb childhood obesity (121). Some of these nutrition standards, including the sodium and whole grain requirements, have been recently relaxed by the U.S. Department of Agriculture due to concerns of perceived palatability and food wastage (122).

Regulations for labeling of calorie and nutrient content of foods—particularly saturated fat, trans fat, added sugar, and sodium levels—can guide consumers to make healthy and informed dietary choices. As part of the proposed revisions to the U.S. nutrition facts label, a line and the percent daily value for added sugar will be included. Some countries have considered other strategies such as front-of-package labeling, which usually places a simple, clear label or symbol conveying essential nutrition information in a more prominent manner. For example, in the United Kingdom, a traffic light system on food packaging has been employed where high, medium, and low levels of fat, saturated fat, sugar, and salt are indicated by traffic light colors red, amber, and green. Compared with nutrition facts panels, which consumers use to draw their own conclusions about how healthful a product is based on the nutrient content of foods, these systems would identify foods that benefit health to help consumers make healthy choices.

Displaying calorie information in menus at chain restaurants is another strategy the U.S. Department of Agriculture implemented in May 2018. A systematic review and meta-analysis suggests that this strategy can be effective in reducing caloric intake (123). However, for greatest benefit, educational campaigns should precede or accompany food package and point-of-purchase nutrition labeling to raise awareness and help with interpretation among consumers.

Food marketing and advertising are able to create major shifts in food demand because marketing leads people to increase their consumption of advertised products (124). A growing body of evidence indicates that food marketing can influence the food preferences and consumption habits of children (125). However, evidence from systematic reviews is lacking, and few studies have evaluated the impact of advertising on energy intake or body weight. A systematic review of 7 randomized trials aiming to assess the effect of television advertising on food intake of children from 4 to 12 years of age concluded that there is a positive association between television and energy intake, but this association is based on a limited number of trials lacking a solid ground of first-level evidence (126). In 2010, the World Health Organization released a set of recommendations on the marketing of foods and nonalcoholic beverages high in fat, sugar, and salt to children in an effort to encourage healthy dietary choices and promote the maintenance of healthy weight (127). In France, marketing of foods high in fat, sugar, and salt is banned unless they are taxed and labeled with a health warning. At the same time, governments can institute zoning laws, if available, that limit the number of fast food restaurants in a given area.

EDUCATION/COMMUNITY. School-based programs and initiatives to improve diet by providing healthy school meals and healthier snack options in vending machines and cafeterias are effective strategies to improve diet quality in children. These strategies are likely to be more effective if reinforced through curriculum-based education about healthy diets and active lifestyles, and efforts to engage parents and families. A recent systematic review including 115 school-based interventions concluded that moderately strong evidence supports the effectiveness of school-based interventions for preventing childhood obesity (128). Similar to the school setting, worksite-based interventions can overcome barriers to choosing a healthy lifestyle by providing resources and a socially supportive environment for change at a place where individuals spend much of their week and by offering programs at low or no cost. A meta-analysis of worksite-based physical activity programs in high-income countries showed significant positive improvements in body weight, cardiometabolic risk factors, physical activity and fitness, and diet quality as well as lower absenteeism and job stress (129). A systematic review of 17 studies in Europe focusing on promoting a healthy diet in the workplace found limited to moderate evidence of effectiveness for prevention of obesity and obesity-related conditions (130). Another systematic review of 16 studies mostly in Europe and North America found that diet-based worksite interventions of moderate methodological quality led to positive changes in fruits, vegetables, and total fat intake (131).

Physicians and other health care providers should monitor the body weight of patients and be trained on how to measure waist circumference, which may be more informative than weight as a marker for cardiometabolic risk. Clinicians should provide suitable evidence-based advice about weight management (116) and refer individuals identified as high-risk for screening of metabolic risk factors. Evaluation of such actions is needed to address this evidence gap. Medical associations and nongovernmental organizations also have central roles in advocacy and can influence policy on issues related to health and the environment. For example, the American Heart Association released a scientific statement calling for a reduction in intake of added sugar to improve health (61), which has become an integral part of the dialogue regarding regulation of SSBs. Nutrition education in medical schools and continuing medical education programs can improve nutrition literacy and nutrition communication skills among health care providers.

INDIVIDUALS. Improvements in diet ultimately rest on individual behavioral change. Behavioral economics, the study of psychological influences on economical decision making, has clear applications in eating habits via implementation of subconscious nudges that may enhance the effectiveness of nutritional policies (132). For example, displaying healthful foods more prominently in school cafeterias may draw more attention to them and thus may increase the purchase of these foods (132). Roberto and Kawachi (133) suggested that the design of dietary interventions could be improved by altering default options, providing simple and meaningful nutrition information, carefully constructing and framing of public health messages, and designing policies to minimize unintended consequences, such as compensation and substitution for unhealthy foods that were reduced with other equally unhealthy options. Combined with financial incentives to produce and purchase healthy foods, and disincentives to produce and purchase unhealthy foods, regulation of food marketing and greater access to healthy foods help individuals create healthy environments in their homes and communities, and make better food choices (134).

RECOMMENDATIONS AND CHALLENGES TO ACHIEVING HEALTHIER DIETS

Physicians, nurses, nutritionists, and community leaders are instrumental in improving diet quality. Basic nutritional skills include knowing: 1) what foods or beverages can be included in a healthy diet; 2) what it means for a food to be healthy; and 3) where to access information on nutrition research. Additional nutritional skills may include: 4) how to read, interpret, and understand peer-reviewed nutrition published reports; 5) pros and cons of supplementation; and 6) information regarding fad diets and foods. Because nutritional research is dynamic and complex, it is unlikely for most health care professionals to keep abreast of the latest findings at all times. Thus, familiarity of well-established knowledge regarding which common foods or beverages are healthy should be the starting point. Small changes are also meaningful. For example, consuming brown rice instead of white rice or choosing fruits or nuts instead of candy bars or potato chips as snacks are excellent first steps. Lastly, improved diet contributes to all stages of prevention, and health professionals should promote better eating, especially among healthy or young patients where no cardiovascular risk factors are apparent.

Achieving healthful eating is a major challenge with many anticipated roadblocks. Socioeconomic disparities has led to a widening gap in diet quality between rich and poor communities from 1999 to 2010 in the United States (135). Food insecurity is an important issue that low-income families often face where food choice is a luxury instead of a reality (136). Continued promotion of nutrition assistance programs and targeted policies for low-income women and children to improve diet quality will be important steps to help close the socioeconomic gap. Better meal offerings in schools are necessary to improve nutrition outcomes in low-income families (137).

Pushback against regulation from the food and beverage industries continues to be an important issue. Consumers consistently cite "healthiness" as a priority when buying food, often leading to the alignment of public health goals and the self-regulation of the food industry, as in the case of the elimination of trans fats from most foods before its government ban (138). However, as SSBs move into the federal crosshairs, the beverage industry has begun to combat regulation with fierce lobbying and public relations campaigns despite clear support for both health and cost savings with the implementation of a penny-per-ounce tax (139). Nutrition labeling is another contentious issue where corporate resistance to highlighting calories, servings, and added sugar exemplifies the conflict between short-term profit and long-term health (140).

Advances in technology may facilitate dietary behavioral changes. Mobile phones are currently capable of scanning barcodes to produce nutritional information in an instant. Text messages encouraging healthier food choices have also been shown to be effective in intervention studies (141). Finally, camera and web-based methods to assess diet may be more cost-effective, easier to use, and less laborious than conventional methods of dietary assessment (142).

SUMMARY AND FUTURE DIRECTIONS

CVD is a global health concern amenable to behavior modifications. Diet is a vital lifestyle component that affects cardiovascular risk through body weight and many other pathways. The large volume of nutritional published reports produced in the last few decades emphasizes avoidance of excess caloric intake; greater consumption of fruits, vegetables, whole grains, fish, nuts, and legumes; moderate consumption of alcohol, coffee, and low-fat/fat-free dairy; and lower consumption of processed meats, refined grains, sodium, and SSBs. Most supplementation trials of individual vitamins or other nutrients among healthy persons do not produce the same magnitudes of risk reduction observed with consuming a high-quality diet. Thus, current preventive efforts should be focused on promotion of better overall eating habits with supplementation as a strategy for subgroups of individuals.

Various biological, economical, physical, social, and psychological factors influence food choices. Interventions targeting these factors can lead to meaningful improvements in long-term eating habits. Much of the improvement in diet quality observed in the United States in recent years has been due to the phasing out of trans fats from the food supply, signifying that public policies arising from evidence-based approaches are instrumental in reducing CVD risk (135). Additional improvements in diet quality can be achieved from a combination of policy strategies across multiple levels including excise taxes on SSBs, economic incentives for the production of healthy foods, regulation of food marketing, healthy school and work environments, and education campaigns. Health professionals and community leaders have a great responsibility to promote cardiovascular health and disease prevention but require a basic nutrition knowledge base. A concerted effort from all levels of society will be needed to fundamentally change the current food environment and the global food system.

ADDRESS FOR CORRESPONDENCE: Dr. Frank B. Hu, Harvard School of Public Health, 655 Huntington Avenue, Building II 3rd Floor, Boston, Massachusetts 02115. E-mail: fhu@hsph.harvard.edu. Twitter: @HSPHnutrition, @HarvardChanSPH.

REFERENCES

1. Pearson TA, Blair SN, Daniels SR, et al. AHA guidelines for primary prevention of cardiovascular disease and stroke: 2002 update. Consensus Panel Guide to Comprehensive Risk Reduction for Adult Patients Without Coronary or Other Atherosclerotic Vascular Diseases. Circulation 2002;106:388–91.

2. Abajobir AA, Abate KH, Abbafati C, et al. Global, regional, and national comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks, 1990–2016: a systematic analysis for the Global Burden of Disease Study 2016. Lancet 2017;390:1345–422.

3. Hu FB, Satija A, Rimm EB, et al. Diet assessment methods in the Nurses' Health Studies and contribution to evidence-based nutritional policies and guidelines. Am J Public Health 2016;106:1567–72.

4. Maki KC, Slavin JL, Rains TM, Kris-Etherton PM. limitations of observational evidence: implications for evidence-based dietary recommendations. Adv Nutr 2014;5:7–15.

5. Satija A, Yu E, Willett WC, Hu FB. Understanding nutritional epidemiology and its role in policy. Adv Nutr 2015;6:5–18.

6. U.S, Department of Health and Human Services, U.S. Department of Agriculture. 2015–2020 Dietary Guidelines for Americans. Washington, DC: U.S. Department of Agriculture, 2015.

7. Soare A, Weiss EP, Pozzilli P. Benefits of caloric restriction for cardiometabolic health, including type 2 diabetes mellitus risk. Diabetes Metab Res Rev 2014;30 Suppl 1:41–7.

8. Franz MJ, VanWormer JJ, Crain AL, et al. Weight-loss outcomes: a systematic review and meta-analysis of weight-loss clinical trials with a minimum 1-year follow-up. J Am Diet Assoc 2007;107:1755–67.

9. Shai I, Schwarzfuchs D, Henkin Y, et al. Weight loss with a low-carbohydrate, Mediterranean, or low-fat diet. N Engl J Med 2008;359:229–41.

10. Skov AR, Toubro S, Rønn B, Holm L, Astrup A. Randomized trial on protein vs carbohydrate in ad libitum fat reduced diet for the treatment of obesity. Int J Obes (Lond) 1999;23:528–36.

11. Layman DK, Evans EM, Erickson D, et al. A moderate-protein diet produces sustained

weight loss and long-term changes in body composition and blood lipids in obese adults. J Nutrition 2009;139:514–21.

12. Ebbeling CB, Swain JF, Feldman HA, et al. Effects of dietary composition on energy expenditure during weight-loss maintenance. JAMA 2012; 307:2627–34.

13. Fortmann SP, Burda BU, Senger CA, Lin JS, Whitlock EP. Vitamin and mineral supplements in the primary prevention of cardiovascular disease and cancer: an updated systematic evidence review for the U.S. Preventive Services Task Force. Ann Internal Med 2013;159:824–34.

14. Aune D, Giovannucci E, Boffetta P, et al. Fruit and vegetable intake and the risk of cardiovascular disease, total cancer and all-cause mortality-a systematic review and dose-response meta-analysis of prospective studies. Int J Epidemiol 2017; 46:1029–56.

15. Bazzano LA, Serdula MK, Liu S. Dietary intake of fruits and vegetables and risk of cardiovascular disease. Curr Atheroscler Rep 2003;5:492–9.

16. Muraki I, Rimm EB, Willett WC, Manson JE, Hu FB, Sun Q. Potato consumption and risk of type 2 diabetes: results from three prospective cohort studies. Diabetes Care 2016;39:376–84.

17. Borgi L, Rimm EB, Willett WC, Forman JP. Potato intake and incidence of hypertension: results from three prospective US cohort studies. BMJ 2016;353:i2351.

18. Borch D, Juul-Hindsgaul N, Veller M, Astrup A. Potatoes and risk of obesity, type 2 diabetes, and cardiovascular disease in apparently healthy adults: a systematic review of clinical intervention and observational studies. Am J Clin Nutr 2016; 104:489–98.

19. Aune D, Keum N, Giovannucci E, et al. Whole grain consumption and risk of cardiovascular disease, cancer, and all cause and cause specific mortality: systematic review and dose-response meta-analysis of prospective studies. BMJ 2016; 353:i2716.

20. Okarter N, Liu RH. Health benefits of whole grain phytochemicals. Crit Rev Food Sci Nutr 2010;50:193–208.

21. Holt SH, Brand-Miller JC, Stitt PA. The effects of equal-energy portions of different breads on blood glucose levels, feelings of fullness and subsequent food intake. J Am Diet Assoc 2001; 101:767–73.

22. Galli C, Rise P. Fish consumption, omega 3 fatty acids and cardiovascular disease. The science and the clinical trials. Nutr Health 2009;20:11–20.

23. Zheng J, Huang T, Yu Y, Hu X, Yang B, Li D. Fish consumption and CHD mortality: an updated meta-analysis of seventeen cohort studies. Public Health Nutr 2012;15:725–37.

24. Afshin A, Micha R, Khatibzadeh S, Mozaffarian D. Consumption of nuts and legumes and risk of incident ischemic heart disease, stroke, and diabetes: a systematic review and meta-analysis. Am J Clinical Nutr 2014;100:278–88.

25. Del Gobbo LC, Falk MC, Feldman R, Lewis K, Mozaffarian D. Effects of tree nuts on blood lipids, apolipoproteins, and blood pressure: systematic review, meta-analysis, and dose-response of 61 controlled intervention trials. Am J Clinical Nutr 2015;102:1347–56.

26. Guo J, Astrup A, Lovegrove JA, Gijsbers L, Givens DI, Soedamah-Muthu SS. Milk and dairy consumption and risk of cardiovascular diseases and all-cause mortality: dose-response meta-analysis of prospective cohort studies. Eur J Epidemiol 2017;32:269–87.

27. Gijsbers L, Ding EL, Malik VS, de Goede J, Geleijnse JM, Soedamah-Muthu SS. Consumption of dairy foods and diabetes incidence: a dose-response meta-analysis of observational studies. Am J Clin Nutr 2016;103:1111–24.

28. Drouin-Chartier J-P, Brassard D, Tessier-Grenier M, et al. Systematic review of the association between dairy product consumption and risk of cardiovascular-related clinical outcomes. Adv Nutr 2016;7:1026–40.

29. Pan A, Sun Q, Bernstein AM, et al. Red meat consumption and mortality: results from two prospective cohort studies. Arch Intern Med 2012; 172:555–63.

30. Micha R, Michas G, Mozaffarian D. Unprocessed red and processed meats and risk of coronary artery disease and type 2 diabetes – an updated review of the evidence. Curr Atheroscler Rep 2012;14:515–24.

31. Bernstein AM, Sun Q, Hu FB, Stampfer MJ, Manson JE, Willett WC. Major dietary protein sources and risk of coronary heart disease in women. Circulation 2010;122:876–83.

32. Li S, Flint A, Pai JK, et al. Low carbohydrate diet from plant or animal sources and mortality among myocardial infarction survivors. J Am Heart Assoc 2014;3:e001169.

33. Etemadi A, Sinha R, Ward MH, et al. Mortality from different causes associated with meat, heme iron, nitrates, and nitrites in the NIH-AARP Diet and Health Study: population based cohort study. BMJ 2017;357:j1957.

34. Micha R, Michas G, Lajous M, Mozaffarian D. Processing of meats and cardiovascular risk: time to focus on preservatives. BMC Med 2013;11:136.

35. Ronksley PE, Brien SE, Turner BJ, Mukamal KJ, Ghali WA. Association of alcohol consumption with selected cardiovascular disease outcomes: a systematic review and meta-analysis. BMJ 2011;342: d671.

36. Zhao J, Stockwell T, Roemer A, Naimi T, Chikritzhs T. Alcohol consumption and mortality from coronary heart disease: an updated meta-analysis of cohort studies. J Stud Alcohol Drugs 2017;78:375–86.

37. Mukamal K, Lazo M. Alcohol and cardiovascular disease. BMJ 2017;356:j1340.

38. Brien SE, Ronksley PE, Turner BJ, Mukamal KJ, Ghali WA. Effect of alcohol consumption on biological markers associated with risk of coronary heart disease: systematic review and meta-analysis of interventional studies. BMJ 2011;342: d636.

39. Rimm EB, Williams P, Fosher K, Criqui M, Stampfer MJ. Moderate alcohol intake and lower risk of coronary heart disease: meta-analysis of effects on lipids and haemostatic factors. BMJ 1999;319:1523–8.

40. Xi B, Huang Y, Reilly KH, et al. Sugar-sweetened beverages and risk of hypertension and CVD: a dose-response meta-analysis. Br J Nutr 2015;113: 709–17.

41. Malik VS, Schulze MB, Hu FB. Intake of sugar-sweetened beverages and weight gain: a systematic review. Am J Clin Nutr 2006;84:274–88.

42. Malik VS, Popkin BM, Bray GA, Després J-P, Hu FB. Sugar sweetened beverages, obesity, type 2 diabetes and cardiovascular disease risk. Circulation 2010;121:1356–64.

43. Ding M, Bhupathiraju SN, Satija A, van Dam RM, Hu FB. Long-term coffee consumption and risk of cardiovascular disease: a systematic review and a dose-response meta-analysis of prospective cohort studies. Circulation 2014;129:643–59.

44. Butt MS, Sultan MT. Coffee and its consumption: benefits and risks. Crit Rev Food Sci Nutr 2011;51:363–73.

45. Wang Z-M, Zhou B, Wang Y-S, et al. Black and green tea consumption and the risk of coronary artery disease: a meta-analysis. Am J Clin Nutr 2011;93:506–15.

46. Wang X, Ouyang YY, Liu J, Zhao G. Flavonoid intake and risk of CVD: a systematic review and meta-analysis of prospective cohort studies. Br J Nutr 2014;111:1–11.

47. Kant AK. Indexes of overall diet quality: a review. J Am Dietetic Assoc 1996;96:785–91.

48. Hu FB. Dietary pattern analysis: a new direction in nutritional epidemiology. Curr Opin Lipidol 2002;13:3–9.

49. Kourlaba G, Panagiotakos DB. Dietary quality indices and human health: a review. Maturitas 2009;62:1–8.

50. George SM, Ballard-Barbash R, Manson JE, et al. Comparing indices of diet quality with chronic disease mortality risk in postmenopausal women in the Women's Health Initiative Observational Study: evidence to inform national dietary guidance. Am J Epidemiol 2014;180:616–25.

51. Sotos-Prieto M, Bhupathiraju SN, Mattei J, et al. Changes in diet quality scores and risk of cardiovascular disease among US men and women. Circulation 2015;132:2212–9.

52. Sotos-Prieto M, Bhupathiraju SN, Mattei J, et al. Association of changes in diet quality with total and cause-specific mortality. N Engl J Med 2017;377:143–53.

53. Li S, Chiuve SE, Flint A, et al. Better diet quality and decreased mortality among myocardial infarction survivors. JAMA Intern Med 2013;173:1808–18.

54. Lopez-Garcia E, Rodriguez-Artalejo F, Li TY, et al. The Mediterranean-style dietary pattern and mortality among men and women with cardiovascular disease. Am J Clin Nutr 2014;99:172–80.

55. Newby PK, Tucker KL. Empirically derived eating patterns using factor or cluster analysis: a review. Nutr Rev 2004;62:177–203.

56. Rodriguez-Monforte M, Flores-Mateo G, Sanchez E. Dietary patterns and CVD: a systematic review and meta-analysis of observational studies. Br J Nutr 2015;114:1341–59.

57. Lassale C, Gunter MJ, Romaguera D, et al. Diet quality scores and prediction of all-cause, cardiovascular and cancer mortality in a pan-European cohort study. PLoS One 2016;11:e0159025.

58. Schwingshackl L, Hoffmann G. Diet quality as assessed by the Healthy Eating Index, the Alternate Healthy Eating Index, the Dietary Approaches to Stop Hypertension score, and health outcomes: a systematic review and meta-analysis of cohort studies. J Acad Nutr Diet 2015;115:780–800.e5.

59. Fan J, Song Y, Wang Y, Hui R, Zhang W. Dietary glycemic index, glycemic load, and risk of coronary heart disease, stroke, and stroke mortality: a systematic review with meta-analysis. PLoS One 2012;7:e52182.

60. Fung TT, van Dam RM, Hankinson SE, Stampfer M, Willett WC, Hu FB. Low-carbohydrate diets and all-cause and cause-specific mortality: two cohort studies. Ann Intern Med 2010;153:289–98.

61. Johnson RK, Appel LJ, Brands M, et al. Dietary sugars intake and cardiovascular health: a scientific statement from the American Heart Association. Circulation 2009;120:1011–20.

62. Rosinger A, Herrick K, Gahche J, Park S. Sugar-sweetened beverage consumption among U.S. adults, 2011-2014. NCHS Data Brief 2017;(270):1–8.

63. Siri-Tarino PW, Sun Q, Hu FB, Krauss RM. Saturated fat, carbohydrate, and cardiovascular disease. Am J Clin Nutr 2010;91:502–9.

64. Nutrient intake and its association with high-density lipoprotein and low-density lipoprotein cholesterol in selected US and USSR subpopulations. The US-USSR Steering Committee for Problem Area I: the pathogenesis of atherosclerosis. Am J Clin Nutr 1984;39:942–52.

65. Parks EJ, Hellerstein MK. Carbohydrate-induced hypertriacylglycerolemia: historical perspective and review of biological mechanisms. Am J Clinical Nutr 2000;71:412–33.

66. Brown IJ, Stamler J, Van Horn L, et al. Sugar-sweetened beverage, sugar intake of individuals, and their blood pressure: international study of macro/micronutrients and blood pressure. Hypertension 2011;57:695–701.

67. Threapleton DE, Greenwood DC, Evans CEL, et al. Dietary fibre intake and risk of cardiovascular disease: systematic review and meta-analysis. BMJ 2013;347:f6879.

68. Streppel MT, Arends LR, van 't Veer P, Grobbee DE, Geleijnse JM. Dietary fiber and blood pressure: a meta-analysis of randomized placebo-controlled trials. Arch Intern Med 2005;165:150–6.

69. Brown L, Rosner B, Willett WW, Sacks FM. Cholesterol-lowering effects of dietary fiber: a meta-analysis. Am J Clin Nutr 1999;69:30–42.

70. Li S, Flint A, Pai JK, et al. Dietary fiber intake and mortality among survivors of myocardial infarction: prospective cohort study. BMJ 2014;348:g2659.

71. Viuda-Martos M, López-Marcos MC, Fernández-López J, Sendra E, López-Vargas JH, Pérez-Álvarez JA. Role of fiber in cardiovascular diseases: a review. Compr Rev Food Sci Food Saf 2010;9:240–58.

72. Story JA, Kritchevsky D. Bile acid metabolism and fiber. Am J Clin Nutr 1978;31 Suppl:S199–202.

73. Kris-Etherton PM. Trans-fats and coronary heart disease. Crit Rev Food Sci Nutr 2010;50:29–30.

74. Astrup A, Dyerberg J, Elwood P, et al. The role of reducing intakes of saturated fat in the prevention of cardiovascular disease: where does the evidence stand in 2010? Am J Clin Nutr 2011;93:684–8.

75. Li Y, Hruby A, Bernstein AM, et al. Saturated fats compared with unsaturated fats and sources of carbohydrates in relation to risk of coronary heart disease. J Am Coll Cardiol 2015;66:1538–48.

76. Aung T, Halsey J, Kromhout D, et al. Associations of omega-3 fatty acid supplement use with cardiovascular disease risks: meta-analysis of 10 trials involving 77 917 individuals. JAMA Cardiol 2018;3:225–34.

77. Siscovick DS, Barringer TA, Fretts AM, et al. Omega-3 polyunsaturated fatty acid (fish oil) supplementation and the prevention of clinical cardiovascular disease. a science advisory from the American Heart Association. Circulation 2017;135:e867–84.

78. Endo J, Arita M. Cardioprotective mechanism of omega-3 polyunsaturated fatty acids. J Cardiol 2016;67:22–7.

79. Kinsella JE, Lokesh B, Stone RA. Dietary n-3 polyunsaturated fatty acids and amelioration of cardiovascular disease: possible mechanisms. Am J Clin Nutr 1990;52:1–28.

80. Nestle M, Wing R, Birch L, et al. Behavioral and social influences on food choice. Nutr Rev 1998;56:50–64.

81. Popkin BM, Duffey K, Gordon-Larsen P. Environmental influences on food choice, physical activity and energy balance. Physiol Behav 2005;86:603–13.

82. Sacks FM, Bray GA, Carey VJ, et al. Comparison of weight-loss diets with different compositions of fat, protein, and carbohydrates. N Engl J Med 2009;360:859–73.

83. Howard BV, Van Horn L, Hsia J, et al. Low-fat dietary pattern and risk of cardiovascular disease: the Women's Health Initiative Randomized Controlled Dietary Modification Trial. JAMA 2006;295:655–66.

84. Downer MK, Gea A, Stampfer M, et al. Predictors of short- and long-term adherence with a Mediterranean-type diet intervention: the PREDIMED randomized trial. Int J Behav Nutr Phys Act 2016;13:67.

85. Estruch R, Salas-Salvadó J, Covas MI, et al. Primary prevention of cardiovascular disease with a mediterranean diet supplemented with extra-virgin olive oil or nuts. N Eng J Med 2018;378:e34.

86. Spronk I, Kullen C, Burdon C, O'Connor H. Relationship between nutrition knowledge and dietary intake. Br J Nutr 2014;111:1713–26.

87. Cluss PA, Ewing L, King WC, Reis EC, Dodd JL, Penner B. Nutrition knowledge of low income parents of obese children. Transl Behav Med 2013;3:218–25.

88. Variyam JN, Blaylock J, Smallwood DM. Modelling nutrition knowledge, attitudes, and diet-disease awareness: the case of dietary fibre. Stat Med 1996;15:23–35.

89. Townsend N, Williams J, Wickramasinghe K, et al. Barriers to healthy dietary choice amongst students in Sri Lanka as perceived by school principals and staff. Health Promot Int 2017;32:91–101.

90. Musaiger AO, Al-Mannai M, Tayyem R, et al. Perceived barriers to healthy eating and physical activity among adolescents in seven Arab Countries: a cross-cultural study. ScientificWorldJournal 2013;2013:232164.

91. Paquette M-C. Perceptions of healthy eating: state of knowledge and research gaps. Can J Public Health 2005;96 Suppl:S15–9.

92. Lappalainen R, Kearney J, Gibney M. A pan EU survey of consumer attitudes to food, nutrition and health: an overview. Food Qual Prefer 1998;9:467–78.

93. Whelan A, Wrigley N, Warm D, Cannings E. Life in a 'food desert'. Urban Stud 2002;39:2083–100.

94. Larsen K, Gilliland J. A farmers' market in a food desert: evaluating impacts on the price and availability of healthy food. Health Place 2009;15:1158–62.

95. Cummins S, Macintyre S. Food environments and obesity—neighbourhood or nation? Int J Epidemiol 2006;35:100–4.

96. Walker RE, Keane CR, Burke JG. Disparities and access to healthy food in the United States: a review of food deserts literature. Health Place 2010;16:876–84.

97. Lake A, Townshend T. Obesogenic environments: exploring the built and food environments. J R Soc Promot Health 2006;126:262–7.

98. Rao M, Afshin A, Singh G, Mozaffarian D. Do healthier foods and diet patterns cost more than less healthy options? A systematic review and meta-analysis. BMJ Open 2013;3.

99. Jabs J, Devine CM. Time scarcity and food choices: an overview. Appetite 2006;47:196–204.

100. Smith LP, Ng SW, Popkin BM. Trends in US home food preparation and consumption: analysis of national nutrition surveys and time use studies from 1965-1966 to 2007-2008. Nutr J 2013;12:45.

101. Lachat C, Nago E, Verstraeten R, Roberfroid D, Van Camp J, Kolsteren P. Eating out of home and its association with dietary intake: a systematic review of the evidence. Obesity Rev 2012;13:329–46.

102. Bernstein AM, Bloom DE, Rosner BA, Franz M, Willett WC. Relation of food cost to healthfulness of diet among US women. Am J Clin Nutr 2010;92:1197–203.

103. Drewnowski A. Taste preferences and food intake. Ann Rev Nutr 1997;17:237–53.

104. Lustig RH, Schmidt LA, Brindis CD. Public health: the toxic truth about sugar. Nature 2012;482:27–9.

105. Popkin BM, Hawkes C. Sweetening of the global diet, particularly beverages: patterns, trends, and policy responses. Lancet Diabetes Endocrinol 2016;4:174–86.

106. Drewnowski A, Bellisle F. Liquid calories, sugar, and body weight. Am J Clin Nutr 2007;85:651–61.

107. Bray GA, Nielsen SJ, Popkin BM. Consumption of high-fructose corn syrup in beverages may play a role in the epidemic of obesity. Am J Clin Nutr 2004;79:537–43.

108. Hu FB. Resolved: there is sufficient scientific evidence that decreasing sugar-sweetened beverage consumption will reduce the prevalence of obesity and obesity-related diseases. Obes Rev 2013;14:606–19.

109. Elder RS, Krishna A. The effects of advertising copy on sensory thoughts and perceived taste. J Consum Res 2010;36:748–56.

110. Veerman JL, Van Beeck EF, Barendregt JJ, Mackenbach JP. By how much would limiting TV food advertising reduce childhood obesity? Eur J Public Health 2009;19:365–9.

111. Kelly B, Halford JCG, Boyland EJ, et al. Television food advertising to children: a global perspective. Am J Public Health 2010;100:1730–6.

112. Shepherd R. Social determinants of food choice. Proc Nutr Soc 2007;58:807–12.

113. Feunekes GIJ, de Graaf C, Meyboom S, van Staveren WA. Food choice and fat intake of adolescents and adults: associations of intakes within social networks. Prev Med 1998;27:645–56.

114. Pachucki MA, Jacques PF, Christakis NA. Social network concordance in food choice among spouses, friends, and siblings. Am J Public Health 2011;101:2170–7.

115. Dietz WH, Scanlon KS. Eliminating the use of partially hydrogenated oil in food production and preparation. JAMA 2012;308:143–4.

116. Gortmaker SL, Swinburn BA, Levy D, et al. Changing the future of obesity: science, policy, and action. Lancet 2011;378:838–47.

117. Colchero MA, Rivera-Dommarco J, Popkin BM, Ng SW. In Mexico, evidence of sustained consumer response two years after implementing a sugar-sweetened beverage tax. Health Aff (Millwood) 2017;36:564–71.

118. Zenk SN, Schulz AJ, Israel BA, James SA, Bao S, Wilson ML. Fruit and vegetable access differs by community racial composition and socioeconomic position in Detroit, Michigan. Ethn Dis 2006;16:275–80.

119. Weems S, Weber JA. Farm bill offers opportunity to improve nutrition of all Americans. J Am Diet Assoc 2007;107:736–8.

120. Brownell KD, Ludwig DS. The Supplemental Nutrition Assistance Program, soda, and USDA policy: who benefits? JAMA 2011;306:1370–1.

121. Food and Nutrition Service (FNS), USDA. Nutrition standards in the National School Lunch and School Breakfast Programs. Final Rule. Fed Regist 2012;77:4088–167.

122. U.S. Department of Agriculture. USDA Commitment to School Meals, a Proclamation. Washington, DC: U.S. Department of Agriculture, 2017.

123. Long MW, Tobias DK, Cradock AL, Batchelder H, Gortmaker SL. Systematic review and meta-analysis of the impact of restaurant menu calorie labeling. Am J Public Health 2015;105:e11–24.

124. Popkin BM, Adair LS, Ng SW. Global nutrition transition and the pandemic of obesity in developing countries. Nutr Rev 2012;70:3–21.

125. Hawkes C. Regulating and litigating in the public interest: regulating food marketing to young people worldwide: trends and policy drivers. Am J Public Health 2007;97:1962–73.

126. Gregori D, Ballali S, Vecchio MG, Scire AS, Foltran F, Berchialla P. Randomized controlled trials evaluating effect of television advertising on food intake in children: why such a sensitive topic is lacking top-level evidence? Ecol Food Nutr 2014;53:562–77.

127. World Health Organization. Set of Recommendations on the Marketing of Foods and Non-Alcoholic Beverages to Children. Geneva, Switzerland: WHO, 2010. Available at: http://whqlibdoc.who.int/publications/2010/9789241500210_eng.pdf?ua=1. Accessed October 17, 2017.

128. Wang Y, Cai L, Wu Y, et al. What childhood obesity prevention programmes work? A systematic review and meta-analysis. Obes Rev 2015;16:547–65.

129. Conn VS, Hafdahl AR, Moore SM, Nielsen PJ, Brown LM. Meta-analysis of interventions to increase physical activity among cardiac subjects. Int J Cardiol 2009;133:307–20.

130. Maes L, Van Cauwenberghe E, Van Lippevelde W, et al. Effectiveness of workplace interventions in Europe promoting healthy eating: a systematic review. Eur J Public Health 2012;22:677–83.

131. Ni Mhurchu C, Aston LM, Jebb SA. Effects of worksite health promotion interventions on employee diets: a systematic review. BMC Public Health 2010;10:62.

132. Just DR, Mancino L, Wansink B. Could Behavioral Economics Help Improve Diet Quality for Nutrition Assistance Program Participants? Economic Research Report No. (ERR-43). Washington, DC: US Department of Agriculture, Economic Research Service, 2007.

133. Roberto CA, Kawachi I. Use of psychology and behavioral economics to promote healthy eating. Am J Prev Med 2014;47:832–7.

134. Pearson-Stuttard J, Bandosz P, Rehm CD, et al. Reducing US cardiovascular disease burden and disparities through national and targeted dietary policies: a modelling study. PLoS Med 2017;14:e1002311.

135. Wang DD, Leung CW, Li Y, et al. Trends in dietary quality among adults in the united states, 1999 through 2010. JAMA Intern Med 2014;174:1587–95.

136. Leung CW, Epel ES, Ritchie LD, Crawford PB, Laraia BA. Food insecurity is inversely associated with diet quality of lower-income adults. J Acad Nutr Diet 2014;114:1943–53.e2.

137. Finkelstein DM, Hill EL, Whitaker RC. School food environments and policies in US public schools. Pediatrics 2008;122:e251–9.

138. Katan MB. Regulation of trans fats: the gap, the Polder, and McDonald's French fries. Atheroscler Suppl 2006;7:63–6.

139. Novak LN, D. Brownell KD. Taxation as prevention and as a treatment for obesity: the case of sugar-sweetened beverages. Curr Pharm Des 2011;17:1218–22.

140. Nestle M. Food Politics: How the Food Industry Influences Nutrition and Health. Oakland, CA: University of California Press, 2013.

141. Kerr DA, Pollard CM, Howat P, et al. Connecting Health and Technology (CHAT): protocol of a randomized controlled trial to improve nutrition behaviours using mobile devices and tailored text messaging in young adults. BMC Public Health 2012;12:477.

142. Illner AK, Freisling H, Boeing H, Huybrechts I, Crispim SP, Slimani N. Review and evaluation of innovative technologies for measuring diet in nutritional epidemiology. Int J Epidemiol 2012;41:1187–203.

KEY WORDS diet, epidemiology, health promotion, prevention, risk factors

Go to http://www.acc.org/jacc-journals-cme to take the CME/MOC/ECME quiz for this article.