



HHS Public Access

Author manuscript

J Am Coll Cardiol. Author manuscript; available in PMC 2019 August 21.

Published in final edited form as:

J Am Coll Cardiol. 2018 August 21; 72(8): 914–926. doi:10.1016/j.jacc.2018.02.085.

Cardiovascular Disease Prevention by Diet Modification: JACC Health Promotion Series

Edward Yu, MSc^{1,2}, Vasanti S. Malik, ScD¹, and Frank B. Hu, MD, PhD^{1,2,3}

¹Department of Nutrition, Harvard T.H. Chan School of Public Health, Boston Massachusetts

²Department of Epidemiology, Harvard T. H. Chan School of Public Health, Boston Massachusetts

³Channing Division of Network Medicine, Brigham and Women's Hospital, Harvard Medical School, Boston Massachusetts

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

CONDENSED ABSTRACT:

Health professionals should emphasize the importance of reducing excess calories and eating 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. Knowledge of healthfulness of food and beverage groups is instrumental to promote healthy eating. Behavioral change begins with small steps, such as substituting whole grains for refined grains or water for sugar-sweetened beverages. Policies are needed to create a food environment wherein healthy foods are accessible, affordable, and desirable.

Corresponding author: Frank B. Hu, MD, PhD, 655 Huntington Ave, Building II 3rd Floor, Boston Massachusetts 02115, Telephone: 617-432-1333, Fax: 617-432-2435, fhu@hsph.harvard.edu, Twitter: @HSPHnutrition | @HarvardChanSPH.

Disclosures: None to report.

Publisher's Disclaimer: This is a PDF file of an unedited manuscript that has been accepted for publication. As a service to our customers we are providing this early version of the manuscript. The manuscript will undergo copyediting, typesetting, and review of the resulting proof before it is published in its final citable form. Please note that during the production process errors may be discovered which could affect the content, and all legal disclaimers that apply to the journal pertain.

Keywords

Diet; Risk factors; Prevention; Epidemiology; Health Promotion

Introduction

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

Observational study of human diet and health outcomes are challenging due to difficulties in measuring dietary intakes (3) and potential problems with generalizability and confounding (4). While 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 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 cardiovascular disease (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 literature 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 article 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 to those who consumed higher amounts of carbohydrates (10–12).

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 (T2D), and 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 15g/d increment in fish intake is associated with a HR = 0.96 (95% CI = 0.90, 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 an HR = 0.76 (95% CI = 0.69, 0.84) for fatal CHD and HR = 0.78 (95% CI = 0.67, 0.92) for nonfatal CHD (24). Small intervention studies have reported lower total cholesterol, LDL-c, ApoB, and triglycerides among those randomized to consuming tree nuts compared to 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 20g/d = 0.98 (95% CI = 0.97–0.99) (26) with similar associations observed across different dairy products. Similar associations have also been observed for total dairy and T2D (HR per 200g/d = 0.97, 95% CI = 0.95, 1.00), and yogurt (HR per 80g/d = 0.86, 95% CI = 0.83, 0.90) (27). Potential benefits of fermented dairy may be due to its probiotics contents (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 coronary heart disease (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 to 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 1–2 drinks a day have the lowest risk (37). Moderate alcohol intake has been shown to increase HDL-c, apolipoprotein A1, adiponectin, and decrease fibrinogen levels (38,39).

Higher consumption of sugar-sweetened beverages 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 increase postprandial blood glucose and insulin concentrations through a high glycemic load, as well as conferring 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 3–5 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, lignans, and phytochemicals. Short term trials of coffee report higher insulin sensitivity and a favorable inflammatory marker profile, but excess intake (>8 cups/d) 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 (aMED), Alternative Healthy Food Index (AHEI), and Dietary Approaches to Stop Hypertension (DASH) 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 Women's Health Initiative, high HEI, AHEI, aMED, and DASH scores were consistently associated with around a 20% reduction in CVD mortality (50). Sotos-Prieto et al. reported that improvement in these scores was also associated with lower risk of total and CVD mortality in two large cohorts (51,52). Similar findings have been observed among MI survivors. Li et al, found that a greater increase in the AHEI score from pre- to post-MI was significantly associated with lower all-cause and cardiovascular mortality (53) and Lopez-Garcia et al. found that adherence to a Mediterranean-style dietary pattern was associated with lower all-cause mortality among individuals with CVD (54).

Principal component and factor analyses have generally identified two 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, sugar sweetened beverages, 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 to 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 magnitude of postprandial glucose level) have been associated with 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 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 accounts for 6.9% of daily calories in the US (62). Higher consumption of added sugars appears to increase risk of CVD independent of body weight or other dietary components (63), likely through lowered HDL-c (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 7g/d 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, 0.91) reduction in risk of death in the highest compared to lowest quintile of cereal fiber intake (70). It is thought that the cardioprotective action of fiber operates through decreased LDL-c, decreased serum triglycerides, blunted postprandial glucose response (71), and changes in bile acid metabolism (72).

Dietary Fat

Of the three 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 U.S. 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 studies that most observational studies did not specify 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 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 remains 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 don't 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 Women's Health Initiative (WHI), have not been successful in achieving target macronutrient compositions or sustaining them after 6 months despite targeted behavioral intervention and in the case of WHI, unrealistic goals for low fat intake (82,83). In contrast, the PREDIMED trial, which evaluated the effects of a Mediterranean diet vs. a low-fat control diet, achieved and sustained intervention goals over 4 years of follow-up (84), largely because olive oil and nuts were provided to participants.

Lack of nutrition knowledge has been suggested as a contributor to poor diet (85), particularly among low income or minority populations (86,87), and in low income countries (88,89), 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 (90). In a large European study of 14,331 participants, lack of knowledge was not cited as a common barrier to healthy eating (91).

On the other hand, lack of availability of healthful foods has been identified as a potential driver of unhealthy eating. ‘Food deserts’ refer to areas with long distances to supermarkets and low access to fresh foods, while food swamps refer to areas with an abundance of unhealthy processed and fast foods (92,93). 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 (94,95). These elements together create an obesogenic environment that can lead to excess adiposity and subsequent cardiometabolic disease (96).

Price is an important roadblock to better eating. Rao et al. reported in a recent metaanalysis 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 (97). Time scarcity has also been shown to promote poor food choice (98) and is a major factor in the decline of home cooking in recent decades (99). Eating out is a significant predictor of overconsumption, and lower micronutrient intake (100). Bernstein et al. suggest that affordable and healthy plant-based diets are achievable with proper knowledge and preparation time (101).

Palatability is an obvious but underappreciated determinant of diet. Human attraction to sweet and savory foods is rooted in evolutionary and anthropologic processes (102), a fact that food companies have exploited by adding large amounts of sugar and sodium to most processed products (103,104). 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 (105–107).

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 (108). Regulations on food branding and restriction of advertising to children have also been proposed as ways to improve diet quality and reduce obesity (109,110).

Social determinants of food choice include influences of culture, friends/family, and community. Social norms have powerful influences on eating patterns, and that healthy food norms can result in healthier food choices (111). Sacks et al. reported that the number of support sessions attended was the strongest predictor of weight loss at 2 years (0.2 kg for every session attended) regardless of macronutrient composition (82). Recent analyses indicate that food choices tend to be shared among family members (112), and that alcohol drinking and snacking were the most “transmissible” eating patterns (113).

ROLE OF PREVENTIVE ACTION

Given the magnitude of the CVD burden in the US and globally and 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 US with the removal of *trans* fats from the FDA's generally regarded as safe category. This action which will be implemented in 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 (114).

Some governments are considering taxing select foods and beverages, particularly SSB's, 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 (115). In Mexico, a peso-per-liter (roughly \$0.80 per liter) tax on sugar-sweetened beverages enacted in 2014 has resulted in an average reduction in sales of 7.6 % of taxed beverages two 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 beverage increased 2.1% (116). To date, at least eight cities in the US 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 US, access to fruit and vegetables has been shown to differ by race and socioeconomic status (117). Amending the US farm bill, the primary agricultural policy tool in the US, could be an effective way to improve diet quality at the population level (118). In particular, this includes amending the Supplemental Nutrition Assistance Program, which provides US\$75 billion per year in subsidies to 47 million US citizens that can be used for the purchase sugar-sweetened beverages and other foods and beverages that adversely affect health (119).

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 efforts to curb childhood obesity (120). Some of these nutrition standards including the sodium and whole grain requirements

have been recently relaxed by the USDA due to concerns of perceived palatability and food wastage (121).

Regulations for labeling of calorie and nutrient content of foods—particularly saturated fat, *trans* fats, added sugar, and sodium levels—can guide consumers to make healthy and informed dietary choices. As part of the proposed revisions to the US Nutrition facts label, a line and % DV 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 UK, 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 to 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 USDA is expected to implement in 2018. A systematic review and meta-analysis suggests that this strategy can be effective in reducing caloric intake (122). 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 (123). A growing body of evidence indicates that food marketing can influence the food preferences and consumption habits of children (124). 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 seven 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 (125). In 2010, the WHO 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 (126). 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 (127). 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 (128). 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 (129). 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 (130).

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 (115) 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 (131), which has become an integral part of the dialogue regarding regulation of sugar-sweetened beverages. 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 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 (133). 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; 3) where to access information on nutrition research. Additional nutritional skills may include: 4) how to read, interpret, and understand peer-reviewed nutrition literature; 5) pros and cons of supplementation; 6) information regarding fad diets and foods. Since nutritional research is dynamic and complex, it is unlikely for most healthcare professionals to keep abreast of the latest findings at all times. Thus, familiarity of well-established knowledge regarding which common food 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 sugar-sweetened beverages 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 longterm 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 (142).

SUMMARY AND FUTURE DIRECTIONS

Cardiovascular disease 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 literature 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 sugar sweetened beverages. 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 US 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.

Acknowledgement

This research is supported by NIH grants HL 60712 and DK46200. Edward Yu is supported by F31 DK114938.

ABBREVIATIONS

CHD	coronary heart disease
CI	confidence interval
CVD	cardiovascular disease
g/d	grams per day
HDL-c	high density lipoprotein cholesterol
HR	hazard ratio
LDL-c	low density lipoprotein cholesterol
MI	myocardial infarction
SSB	sugar sweetened beverage

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;106:388–391. [PubMed: 12119259]
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;390:1345–1422. [PubMed: 28919119]
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;106:1567–1572. [PubMed: 27459459]
4. Maki KC, Slavin JL, Rains TM, Kris-Etherton PM. Limitations of Observational Evidence: Implications for Evidence-Based Dietary Recommendations. *Advances in Nutrition: An International Review Journal* 2014;5:7–15.
5. Satija A, Yu E, Willett WC, Hu FB. Understanding Nutritional Epidemiology and Its Role in Policy. *Advances in Nutrition: An International Review Journal* 2015;6:5–18.
6. Health UDo, Services H. 2015–2020 dietary guidelines for Americans Washington (DC): USDA 2015.
7. Soare A, Weiss EP, Pozzilli P. Benefits of caloric restriction for cardiometabolic health, including type 2 diabetes mellitus risk. *Diabetes/metabolism research and reviews* 2014;30 Suppl 1:41–7. [PubMed: 24532291]
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. *Journal of the American Dietetic Association* 2007;107:1755–1767. [PubMed: 17904936]
9. Shai I, Schwarzfuchs D, Henkin Y et al. Weight Loss with a Low-Carbohydrate, Mediterranean, or Low-Fat Diet. *New England Journal of Medicine* 2008;359:229–241. [PubMed: 18635428]
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. *International Journal Of Obesity* 1999;23:528. [PubMed: 10375057]
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;139:514–521. [PubMed: 19158228]
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. [PubMed: 22735432]
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;159:824–834. [PubMed: 24217421]
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. *International J epidemiology* 2017.
15. Bazzano LA, Serdula MK, Liu S. Dietary intake of fruits and vegetables and risk of cardiovascular disease. *Current Atherosclerosis Reports* 2003;5:492–499. [PubMed: 14525683]
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. [PubMed: 26681722]
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.
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. [PubMed: 27413134]

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.
20. Okarter N, Liu RH. Health benefits of whole grain phytochemicals. *Crit Rev Food Sci Nutr* 2010;50:193–208. [PubMed: 20301011]
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. [PubMed: 11478473]
22. Galli C, Rise P. Fish consumption, omega 3 fatty acids and cardiovascular disease. The science and the clinical trials. *Nutrition health* 2009;20:11–20. [PubMed: 19326716]
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. [PubMed: 21914258]
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;100:278–288. [PubMed: 24898241]
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;102:1347–1356. [PubMed: 26561616]
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;32:269–287. [PubMed: 28374228]
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. [PubMed: 26912494]
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. *Advances in Nutrition: An International Review Journal* 2016;7:1026–1040.
29. Pan A, Sun Q, Bernstein AM et al. Red Meat Consumption and Mortality: Results from Two Prospective Cohort Studies. *Archives of internal medicine* 2012;172:555–563. [PubMed: 22412075]
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. *Current atherosclerosis reports* 2012;14:515–524. [PubMed: 23001745]
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. [PubMed: 20713902]
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. [PubMed: 25246449]
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.
34. Micha R, Michas G, Lajous M, Mozaffarian D. Processing of meats and cardiovascular risk: time to focus on preservatives. *BMC Medicine* 2013; 11:136. [PubMed: 23701737]
35. Ronsley 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.
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. *Journal of studies on alcohol and drugs* 2017;78:375–386. [PubMed: 28499102]
37. Mukamal K, Lazo M. Alcohol and cardiovascular disease. *BMJ* 2017;356.
38. Brien SE, Ronsley 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.

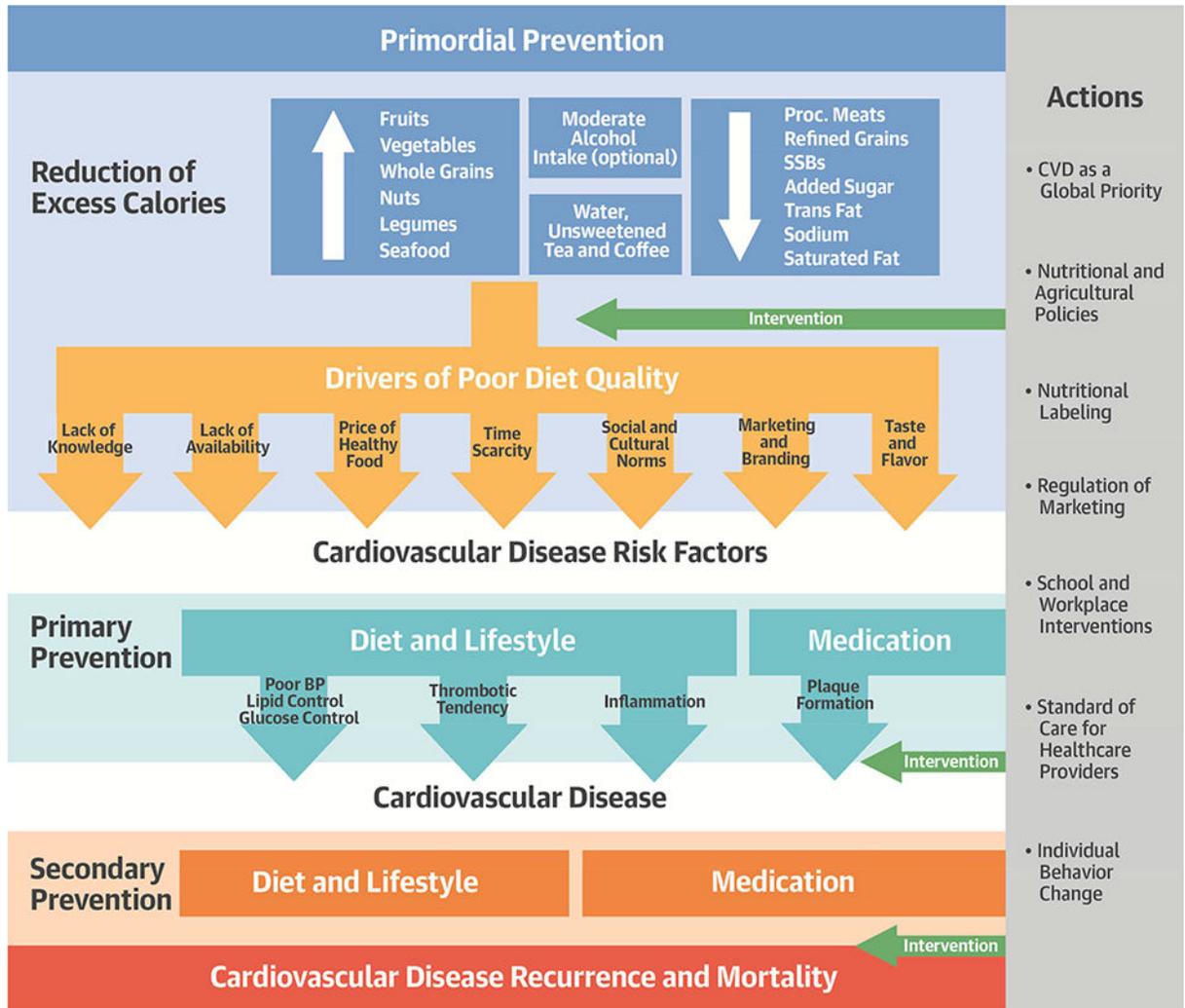
39. Rimm EB, Williams P, Fosher K, Criqui M, Stampfer MJ. Moderate alcohol intake and lower risk of coronary heart disease: meta-analysis of effects on lipids and haemostatic factors. *BMJ* : *BMJ* 1999;319:1523–1528. [PubMed: 10591709]
40. Xi B, Huang Y, Reilly KH et al. Sugar-sweetened beverages and risk of hypertension and CVD: a dose-response meta-analysis. *British 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 clinical nutrition* 2006;84:274–288. [PubMed: 16895873]
42. Malik VS, Popkin BM, Bray GA, Despres J-P, Hu FB. Sugar Sweetened Beverages, Obesity, Type 2 Diabetes and Cardiovascular Disease risk. *Circulation* 2010;121: 1356–1364. [PubMed: 20308626]
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* 2013.
44. Butt MS, Sultan MT. Coffee and its Consumption: Benefits and Risks. *Critical Reviews in Food Science and Nutrition* 2011;51:363–373. [PubMed: 21432699]
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 Clinical Nutrition* 2011;93:506–515. [PubMed: 21248184]
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. *British J nutrition* 2014;111:1–11.
47. Kant AK. Indexes of Overall Diet Quality: A Review. *J Am Dietetic Assoc.* 1996;96:785–791.
48. Hu FB. Dietary pattern analysis: a new direction in nutritional epidemiology. *Current opinion in lipidology* 2002;13:3–9. [PubMed: 11790957]
49. Kourlaba G, Panagiotakos DB. Dietary quality indices and human health: a review. *Maturitas* 2009;62:1–8. [PubMed: 19128905]
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 Epidemiology* 2014;180:616–625.
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;132:2212–2219. [PubMed: 26644246]
52. Sotos-Prieto M, Bhupathiraju SN, Mattei J et al. Association of Changes in Diet Quality with Total and Cause-Specific Mortality. *New Engl J Med.* 2017;377:143–153. [PubMed: 28700845]
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. [PubMed: 23999993]
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. [PubMed: 24172306]
55. Newby PK, Tucker KL. Empirically Derived Eating Patterns Using Factor or Cluster Analysis: A Review. *Nutrition Reviews* 2004;62:177–203. [PubMed: 15212319]
56. Rodriguez-Monforte M, Flores-Mateo G, Sanchez E. Dietary patterns and CVD: a systematic review and meta-analysis of observational studies. *British J nutrition* 2015;114:1341–59.
57. Lassale C, Gunter MJ, Romaguera D et al. Diet Quality Scores and Prediction of AllCause, Cardiovascular and Cancer Mortality in a Pan-European Cohort Study. *PLoS ONE* 2016;11:e0159025. [PubMed: 27409582]
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. [PubMed: 25680825]
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. [PubMed: 23284926]
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;153:289–298. [PubMed: 20820038]

61. Johnson RK, Appel LJ, Brands M et al. Dietary Sugars Intake and Cardiovascular Health. A Scientific Statement From the American Heart Association 2009;120:1011–1020.
62. Rosinger A, Herrick K, Gahche J, Park S. Sugar-sweetened Beverage Consumption Among U.S. Adults, 2011–2014. NCHS data brief 2017:1–8.
63. Siri-Tarino PW, Sun Q, Hu FB, Krauss RM. Saturated fat, carbohydrate, and cardiovascular disease. The American Journal of Clinical Nutrition 2010;91:502–509. [PubMed: 20089734]
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. [PubMed: 6609630]
65. Parks EJ, Hellerstein MK. Carbohydrate-induced hypertriglycerolemia: historical perspective and review of biological mechanisms. Am J Clinical Nutr. 2000;71:412–433. [PubMed: 10648253]
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 (Dallas, Tex: 1979) 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.
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. Archives of internal medicine 2005;165:150–6. [PubMed: 15668359]
69. Brown L, Rosner B, Willett WW, Sacks FM. Cholesterol-lowering effects of dietary fiber: a meta-analysis. The American Journal of Clinical Nutrition 1999;69:30–42. [PubMed: 9925120]
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. [PubMed: 24782515]
71. Viuda-Martos M, Lopez-Marcos MC, Fernandez-Lopez J, Sendra E, Lopez-Vargas JH, Perez-Alvarez JA. Role of Fiber in Cardiovascular Diseases: A Review. Comprehensive Reviews in Food Science and Food Safety 2010;9:240–258.
72. Story JA, Kritchevsky D. Bile acid metabolism and fiber. The American Journal of Clinical Nutrition 1978;31:S199–S202. [PubMed: 707373]
73. Kris-Etherton PM. Trans-Fats and Coronary Heart Disease. Critical Reviews in Food Science and Nutrition 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? The American Journal of Clinical Nutrition 2011;93:684–688. [PubMed: 21270379]
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. Journal of the American College of Cardiology 2015;66:1538–1548. [PubMed: 26429077]
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 Cardiology 2018.
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 2017;135:e867–e884.
78. Endo J, Arita M. Cardioprotective mechanism of omega-3 polyunsaturated fatty acids. Journal of Cardiology 2016;67:22–27. [PubMed: 26359712]
79. Kinsella JE, Lokesh B, Stone RA. Dietary n-3 polyunsaturated fatty acids and amelioration of cardiovascular disease: possible mechanisms. The American Journal of Clinical Nutrition 1990;52:1–28. [PubMed: 2193500]
80. Nestle M, Wing R, Birch L et al. Behavioral and Social Influences on Food Choice. Nutrition Reviews 1998;56:50–64.
81. Popkin BM, Duffey K, Gordon-Larsen P. Environmental influences on food choice, physical activity and energy balance. Physiology & Behavior 2005;86:603–613. [PubMed: 16246381]

82. Sacks FM, Bray GA, Carey VJ et al. Comparison of Weight-Loss Diets with Different Compositions of Fat, Protein, and Carbohydrates. *New England Journal of Medicine* 2009;360:859–873. [PubMed: 19246357]
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. [PubMed: 16467234]
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. *The international journal of behavioral nutrition and physical activity* 2016;13:67. [PubMed: 27297426]
85. Spronk I, Kullen C, Burdon C, O'Connor H. Relationship between nutrition knowledge and dietary intake. *British Journal of Nutrition* 2014;111:1713–1726. [PubMed: 24621991]
86. Cluss PA, Ewing L, King WC, Reis EC, Dodd JL, Penner B. Nutrition Knowledge of Low Income Parents of Obese Children. *Translational behavioral medicine* 2013;3:218–225. [PubMed: 24039639]
87. Variyam JN, Blaylock J, Smallwood DM. Modelling nutrition knowledge, attitudes, and diet-disease awareness: the case of dietary fibre. *Statistics in medicine* 1996;15:23–35. [PubMed: 8614743]
88. 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 Promotion International* 2017;32:91–101. [PubMed: 28180258]
89. 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. *The Scientific World Journal* 2013;2013:232164. [PubMed: 24348144]
90. Paquette M-C. Perceptions of Healthy Eating: State of Knowledge and Research Gaps. *Canadian Journal of Public Health / Revue Canadienne de Sante'e Publique* 2005;96:S15–S19.
91. Lappalainen R, Kearney J, Gibney M. A pan EU survey of consumer attitudes to food, nutrition and health: an overview. *Food Quality and Preference* 1998;9:467–478.
92. Whelan A, Wrigley N, Warm D, Cannings E. Life in a 'Food Desert'. *Urban Studies* 2002;39:2083–2100.
93. Larsen K, Gilliland J. A farmers' market in a food desert: Evaluating impacts on the price and availability of healthy food. *Health & Place* 2009;15:1158–1162. [PubMed: 19631571]
94. Cummins S, Macintyre S. Food environments and obesity—neighbourhood or nation? *International journal of epidemiology* 2006;35:100–104. [PubMed: 16338945]
95. 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;16:876–884. [PubMed: 20462784]
96. Lake A, Townshend T. Obesogenic environments: exploring the built and food environments. *The journal of the Royal Society for the Promotion of Health* 2006;126:262–267. [PubMed: 17152319]
97. 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.
98. Jabs J, Devine CM. Time scarcity and food choices: An overview. *Appetite* 2006;47:196–204. [PubMed: 16698116]
99. 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. *Nutrition Journal* 2013;12:45. [PubMed: 23577692]
100. 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 Reviews* 2012;13:329–346. [PubMed: 22106948]
101. 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. [PubMed: 20810972]
102. Drewnowski A Taste preferences and food intake. *Annual review of nutrition* 1997;17:237–253.
103. Lustig RH, Schmidt LA, Brindis CD. Public health: the toxic truth about sugar. *Nature* 2012;482:27–29. [PubMed: 22297952]

104. Popkin BM, Hawkes C. Sweetening of the global diet, particularly beverages: patterns, trends, and policy responses. *The Lancet Diabetes & Endocrinology* 2016;4:174–186. [PubMed: 26654575]
105. Drewnowski A, Bellisle F. Liquid calories, sugar, and body weight. *The American Journal of Clinical Nutrition* 2007;85:651–661. [PubMed: 17344485]
106. Bray GA, Nielsen SJ, Popkin BM. Consumption of high-fructose corn syrup in beverages may play a role in the epidemic of obesity. *The American Journal of Clinical Nutrition* 2004;79:537–543. [PubMed: 15051594]
107. Hu FB. Resolved: there is sufficient scientific evidence that decreasing sugar-sweetened beverage consumption will reduce the prevalence of obesity and obesity-related diseases. *Obesity reviews : an official journal of the International Association for the Study of Obesity* 2013;14:606–19. [PubMed: 23763695]
108. Elder RS, Krishna A. The Effects of Advertising Copy on Sensory Thoughts and Perceived Taste. *Journal of Consumer Research* 2010;36:748–756.
109. Veerman JL, Van Beeck EF, Barendregt JJ, Mackenbach JP. By how much would limiting TV food advertising reduce childhood obesity? *The European Journal of Public Health* 2009;19:365–369. [PubMed: 19324935]
110. Kelly B, Halford JCG, Boyland EJ et al. Television Food Advertising to Children: A Global Perspective. *American Journal of Public Health* 2010;100:1730–1736. [PubMed: 20634464]
111. Shepherd R Social determinants of food choice. *Proceedings of the Nutrition Society* 2007;58:807–812.
112. 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. *Preventive Medicine* 1998;27:645–656. [PubMed: 9808794]
113. Pachucki MA, Jacques PF, Christakis NA. Social Network Concordance in Food Choice Among Spouses, Friends, and Siblings. *American Journal of Public Health* 2011;101:2170–2177. [PubMed: 21940920]
114. Dietz WH, Scanlon KS. Eliminating the use of partially hydrogenated oil in food production and preparation. *Jama* 2012;308:143–144. [PubMed: 22782414]
115. Gortmaker SL, Swinburn BA, Levy D et al. Changing the future of obesity: science, policy, and action. *Lancet* 2011;378:838–47. [PubMed: 21872752]
116. 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;36:564–571. [PubMed: 28228484]
117. 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. [PubMed: 16599383]
118. Weems S, Weber JA. Farm bill offers opportunity to improve nutrition of all Americans. *J Am Diet Assoc* 2007;107:736–8. [PubMed: 17467364]
119. Brownell KD, Ludwig DS. The Supplemental Nutrition Assistance Program, soda, and USDA policy: who benefits? *Jama* 2011;306:1370–1. [PubMed: 21954481]
120. Food and Nutrition Service (FNS) U. Nutrition Standards in the National School Lunch and School Breakfast Programs. 2012.
121. Agriculture USDo. USDA COMMITMENT TO SCHOOL MEALS, A PROCLAMATION. 2017.
122. 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.
123. Popkin BM, Adair LS, Ng SW. Global nutrition transition and the pandemic of obesity in developing countries. *Nutr Rev* 2012;70:3–21. [PubMed: 22221213]
124. 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. [PubMed: 17901436]
125. Gregori D, Ballali S, Vecchio MG, Scire AS, Foltran F, Berchiolla 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;53:562–577. [PubMed: 25105865]
126. World Health Organization 2010. “Set of Recommendations on the Marketing of Foods and Non-Alcoholic Beverages to Children” WHO, Geneva <http://whqlibdoc.who.int/publications/2010/9789241500210eng.pdf?ua=1>. .
 127. Wang Y, Cai L, Wu Y et al. What childhood obesity prevention programmes work? A systematic review and meta-analysis. *Obesity reviews : an official journal of the International Association for the Study of Obesity* 2015;16:547–65. [PubMed: 25893796]
 128. 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. [PubMed: 18582959]
 129. 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. [PubMed: 21785115]
 130. 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. [PubMed: 20146795]
 131. 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. [PubMed: 19704096]
 132. Just DR, Mancino L, Wansink B. Could behavioral economics help improve diet quality for nutrition assistance program participants?: US Department of Agriculture, Economic Research Service Washington, DC, 2007.
 133. Roberto CA, Kawachi I. Use of psychology and behavioral economics to promote healthy eating. *Am J Prev Med* 2014;47:832–7. [PubMed: 25441239]
 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 Medicine* 2017;14:e1002311. [PubMed: 28586351]
 135. Wang DD, Leung CW, Li Y, et al. Trends in dietary quality among adults in the united states, 1999 through 2010. *JAMA Internal Medicine* 2014;174:1587–1595. [PubMed: 25179639]
 136. Leung CW, Epel ES, Ritchie LD, Crawford PB, Laraia BA. Food insecurity is inversely associated with diet quality of lower-income adults. *Journal of the Academy of Nutrition and Dietetics* 2014;114:1943–1953. e2. [PubMed: 25091796]
 137. Finkelstein DM, Hill EL, Whitaker RC. School Food Environments and Policies in US Public Schools. *Pediatrics* 2008;122:e251–e259. [PubMed: 18595970]
 138. Katan MB. Regulation of trans fats: The gap, the Polder, and McDonald’s French fries. *Atherosclerosis Supplements* 2006;7:63–66. [PubMed: 16713390]
 139. Novak N L, Brownell K D. Taxation as Prevention and as a Treatment for Obesity: The Case of Sugar-Sweetened Beverages. *Current Pharmaceutical Design* 2011;17:1218–1222. [PubMed: 21492083]
 140. Nestle M Food politics: How the food industry influences nutrition and health: Univ 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. [PubMed: 22726532]
 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. *International journal of epidemiology* 2012;41:1187–1203. [PubMed: 22933652]



Central Illustration: Flow diagram of the development of cardiovascular disease and possible prevention by a healthy diet.

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. Abbreviations: AHEI, Alternative Healthy Food 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.

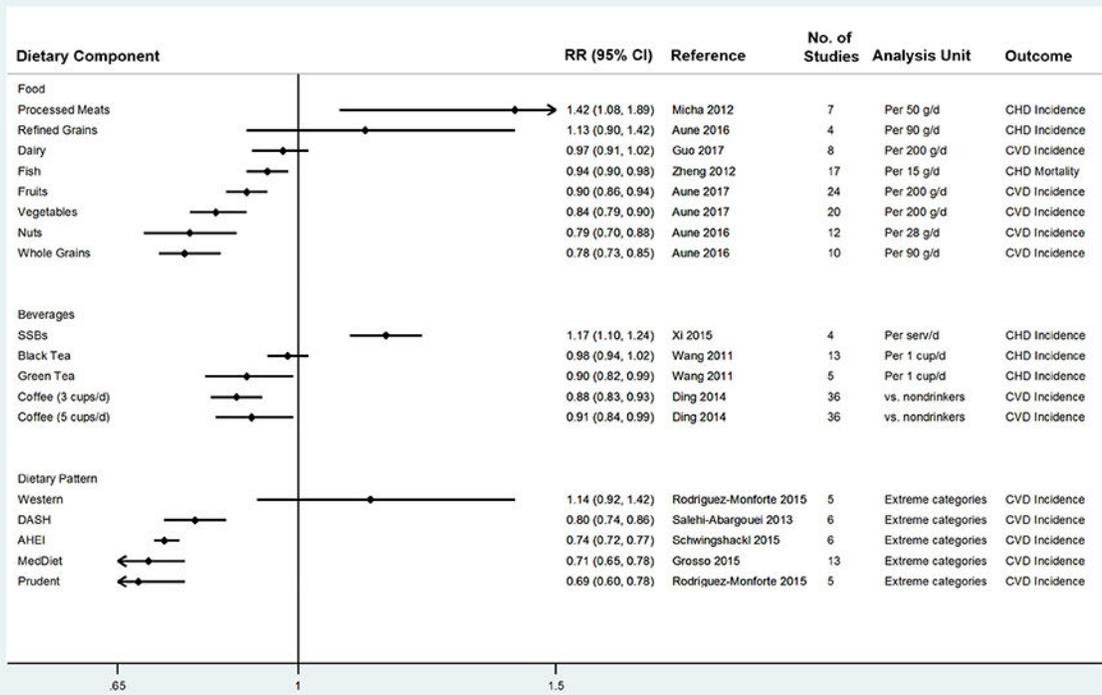


Figure 1: Summary of various meta-analyses for the associations of key foods and food groups, and dietary patterns with incident cardiovascular disease.

High amounts of processed meat, SSB, 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. Abbreviations: AHEI, Alternative Healthy Food 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.