Recent Advances in Preventive Cardiology and Lifestyle Medicine

Components of a Cardioprotective Diet
New Insights

Dariush Mozaffarian, MD, DrPH; Lawrence J. Appel, MD, MPH; Linda Van Horn, PhD, RD

The global burdens of cardiovascular diseases (CVD), type 2 diabetes mellitus (DM), and obesity are rising, producing enormous losses of life and disability-adjusted life-years in both developed and developing nations.1 Most of these burdens are preventable and are occurring at unnecessarily younger ages, largely owing to suboptimal lifestyle, which includes poor diet quality, excess caloric intake, physical inactivity, and smoking.2–5 Worldwide, striking differences in dietary habits and rates of chronic diseases exist. The identification and targeting of dietary factors with the greatest potential for reducing CVD, DM, and obesity are of major scientific and public health importance.

The science of diet and chronic disease is relatively young, spanning perhaps only half a century.6 New advances offer substantial evidence from complementary research paradigms on cardiometabolic effects of specific dietary factors. Several recent evidence-based reviews conducted in conjunction with national and international policy-making efforts provide the context for the present report. The need to prioritize selected foods and overall dietary patterns rather than only individual nutrients, the relevance of carbohydrate and fat quality as well as quantity, the effects and policy implications of sodium consumption, the importance of energy balance, and the role of dietary supplements represent several key findings of recent evidence-based reviews conducted in conjunction with national and international policy-making efforts providing the context for the present report. The need to prioritize selected foods and overall dietary patterns rather than only individual nutrients, the relevance of carbohydrate and fat quality as well as quantity, the effects and policy implications of sodium consumption, the importance of energy balance, and the role of dietary supplements represent several key findings of evidence-based insights into successful individual and public health strategies for behavior change are also addressed. Overall, the present report is intended to provide a useful framework for health practitioners and policy makers to understand contemporary issues related to the effects of diet on CVD.

Methods

Evidence for effects of dietary factors on cardiometabolic health was reviewed, often derived from recent comprehensive reviews performed for policy-making or similar activities, including those of the American Heart Association (AHA) 2020 Impact Goals Committee (D.M., L.J.A.),7 the AHA Nutrition Committee (D.M., L.J.A., L.V.H.),8,9 the US Dietary Guidelines Advisory Committee (L.J.A., L.V.H.),10 the World Health Organization Expert Consultation on Fats and Fatty Acids in Human Nutrition (D.M.),11 the World Health Organization Global Burden of Diseases, Risk Factors, and Injuries Nutrition and Chronic Diseases Expert Group (D.M.),12 and the Institute of Medicine Report on Strategies to Reduce Sodium Intake (L.J.A.).13 The views expressed in the present report are those of the authors and do not necessarily represent the views or conclusions of the committees on which they served.

The present report was not intended to cover all possible nutritional topics related to CVD. Rather, it reviews selected topics with reasonably robust evidence yielding important new insights. Several relevant criteria currently used to evaluate evidence for effects of dietary habits on chronic diseases were adopted, including those of Bradford Hill,14 the World Health Organization,15 and similar criteria. Evidence from human studies evaluating established cardiometabolic risk factors or clinical end points was prioritized, including systematic reviews that provided quantitative pooled effect estimates from multiple studies.

Foods

Randomized, controlled trials (RCTs) of cardiometabolic risk factors and prospective cohort studies of disease end points provide strong concordant evidence for cardiovascular effects of several specific foods. In contrast with individual nutrients in isolation, health effects of foods likely represent the synergy of composite effects and interactions of multiple factors, including carbohydrate quality, fiber content, specific fatty acids and proteins, preparation methods, food structure, and bioavailability of inherent micronutrients and phytochemicals.6,16,17 Dietary patterns based on particular foods/components (Table 1) have established cardiometabolic benefits and are higher in dietary fiber, healthy fatty acids, vitamins, antioxidants, potassium, other minerals, and phytochemicals and lower in refined carbohydrates, sugars, salt, saturated fatty acid (SFA), dietary cholesterol, and trans fat.

Fruits and Vegetables

In RCTs, diets that emphasize consumption of fruits and vegetables produce substantial improvements in several risk factors, including blood pressure (BP), lipid levels, insulin resistance, inflammatory biomarker levels, endothelial function, and weight control.19–25 Benefits do not appear reproducible with equivalent amounts of representative mineral and fiber supplements,26 nor are they dependent on dietary macronutrient (fat, protein, or carbohydrate) composition.22

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### Table 1. Food-Based Components of Dietary Patterns That Improve Cardiometabolic Health*

<table>
<thead>
<tr>
<th>Goal†</th>
<th>Serving Sizes</th>
<th>Nutrient Benefits</th>
</tr>
</thead>
<tbody>
<tr>
<td>Consume more:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fruits</td>
<td>4 to 5 servings per day</td>
<td>1 medium-sized fruit; ½ cup of fresh, frozen, or unsweetened canned fruit; ¼ cup of dried fruit; ½ cup of 100% juice. Many guidelines recommended no more than 4 oz of juice per day for children.</td>
</tr>
<tr>
<td>Vegetables</td>
<td>4 to 5 servings per day</td>
<td>1 cup of raw leafy vegetable; ½ cup of cut-up raw vegetables, cooked vegetables, or 100% juice. Limit starchy vegetables such as potatoes to ½ cup or less per day.</td>
</tr>
<tr>
<td>Whole grains‡</td>
<td>3 servings per day, in place of refined grains</td>
<td>1 slice of whole grain bread; 1 cup of high-fiber whole grain cereal; ½ cup of cooked whole grain rice, pasta, or cereal.</td>
</tr>
<tr>
<td>Fish and shellfish</td>
<td>2 servings per week, preferably oily (see text)</td>
<td>100 g (3.5 oz). Goals should not be met with commercially prepared deep fried or breaded fish.</td>
</tr>
<tr>
<td>Nuts</td>
<td>4 to 5 servings per week</td>
<td>50 g (1.75 oz). Unsalted nuts are preferable to minimize sodium intake.</td>
</tr>
<tr>
<td>Dairy products</td>
<td>2 to 3 servings per day</td>
<td>1 cup of milk or yogurt; 1 oz of cheese. Most dietary guidelines recommend low-fat or nonfat dairy products.</td>
</tr>
<tr>
<td>Vegetable oils</td>
<td>2 to 6 servings per day</td>
<td>1 teaspoon oil, eg, in cooking or salad dressing; 1 tablespoon vegetable spread. Goals should not be met with coconut, palm kernel, or other tropical oils low in unsaturated fats.</td>
</tr>
<tr>
<td>Consume less:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fats, oils, or foods containing or made with partially hydrogenated vegetable oils</td>
<td>Avoid intake</td>
<td>Reduced consumption of industrially produced trans fatty acids.</td>
</tr>
<tr>
<td>Processed meats (eg, bacon, sausage, hot dogs, processed deli meats)</td>
<td>Modest intake, eg, up to 2 servings per week</td>
<td>50 g (1.75 oz)</td>
</tr>
<tr>
<td>Sugar-sweetened beverages, sweets, and grain-based desserts and bakery foods</td>
<td>Modest intake, eg, up to 5 servings per week</td>
<td>8 oz of soda; 1 small candy, cookie, doughnut, or muffin; 1 slice of cake or pie</td>
</tr>
<tr>
<td>Alcohol§</td>
<td>For those who consume alcohol, up to 2 daily for men, 1 daily for women</td>
<td>5 oz wine; 12 oz beer; 1.5 oz spirits</td>
</tr>
<tr>
<td>Energy balance</td>
<td>Reduce portion sizes, increase physical activity (minimum 150 min/wk moderate or 75 min/wk vigorous activity); limit TV watching, and ensure adequate sleep.</td>
<td>For an average 73-kg adult, 1 hour of brisk walking burns ~277 kcal; hiking, 438 kcal; swimming, 511 kcal; and jogging, tennis, or basketball, 584 kcal.</td>
</tr>
</tbody>
</table>

†Based on a 2000-kcal/d diet. Servings should be adjusted accordingly for higher or lower energy consumption.
‡Or potentially, whole legumes (beans), although evidence for equivalent effects is limited. Characterization of whole grain content in packaged foods is challenging; pragmatically, sufficient whole grain content was defined by the American Heart Association 2020 Impact Goals as the fiber content of whole wheat, ie, at least 1.1 g of naturally occurring dietary fiber per 10 g of carbohydrate in the grain product.
§Because of alcohol-related accidents, homicides, and suicides, especially among younger adults, alcohol use has an overall net adverse effect on population mortality. Thus, alcohol use is not advisable as a population-based strategy to reduce cardiovascular risk. For adults who already drink alcohol, no more than moderate use should be encouraged.
§Higher levels of physical activity may be needed to promote prolonged negative energy balance, ie, sustained weight loss.
Figure 1. Relationships of consumption of different foods with incidence of coronary heart disease (CHD), stroke, and diabetes in meta-analyses of prospective cohort studies (PCs). Adapted with permission from Mozaffarian.29 Dashes indicate not reported; CVD, cardiovascular disease; and RR (95% CI), relative risk (95% confidence interval).
This evidence suggests that benefits might be derived from (1) a more complex set of micronutrients, phytochemicals, and fiber found in fruits and vegetables; (2) potentially enhanced bioavailability of these nutrients in their natural state; and/or (3) replacement of less healthful foods in the diet. In long-term observational studies, greater fruit and vegetable consumption are each associated with lower incidence of coronary heart disease (CHD), and greater fruit consumption is associated with lower incidence of stroke (Figure 1). The results of RCTs of physiological measures and prospective cohorts of disease outcomes together provide strong concordant evidence that fruit and vegetable consumption lowers CVD risk (Table 2). Potential differences in health effects contributed by specific types of fruits, vegetables, or their juices require further investigation.

Whole Grains

Although no single accepted definition of whole grain exists, whole grains generally comprise bran, germ, and endosperm from the natural cereal. Bran contains soluble and insoluble dietary fiber, B vitamins, minerals, flavonoids, and tocopherols; germ contains numerous fatty acids, antioxidants, and phytochemicals. Endosperm provides largely starch (carbohydrate polysaccharides) and storage proteins. The type and extent of processing appear to modify the health effects of grain and carbohydrate consumption (Figure 2). For example, removal of bran and germ reduces dietary fiber that has important benefits, including lowering of BP and cholesterol levels; increases bioavailability and rapidity of digestion of remaining endosperm, which increases glycemic responses; and eliminates minerals, micronutrients, and other phytochemicals that may have additional independent health benefits (see “Carbohydrate Amount and Quality”).

In RCTs, consumption of whole grains improves glucose-insulin homeostasis and endothelial function and possibly reduces inflammation and improves weight loss. Consumption of whole grain oats reduces low-density lipoprotein (LDL) cholesterol without reducing high-density lipoprotein cholesterol or raising triglycerides. Consistent with physiological benefits, greater whole grain consumption is associated with lower incidence of CHD, DM, and possibly stroke (Figure 1). The higher dietary fiber in whole grains contributes to these benefits. In RCTs, increased dietary fiber reduces serum triglycerides, LDL cholesterol, blood glucose, and BP. Emerging evidence supports additional independent contributions to health from other characteristics of whole grains, including slower digestion (lower glycemic responses) and higher content of minerals, phytochemicals, and fatty acids. Thus, similar to fruits and vegetables, health effects of whole grains may result from synergistic effects of multiple constituents that are unlikely to be matched by supplemental fiber alone, added bran, or isolated micronutrients, as well as from dietary substitution for more highly refined/processed carbohydrates and starches that may themselves induce adverse cardiometabolic effects (see “Carbohydrate Amount and Quality”).

Improved nutritional assessment techniques and databases are needed to further quantify and evaluate potential contributions to health of each component of whole grains. Evidence-based yet pragmatic definitions of what constitutes a whole grain food (Figure 2) are also essential to reduce public confusion and allow consumers to choose healthy grains wisely. As one example, to help monitor national progress toward achieving dietary goals, the AHA 2020 Impact Goals developed a pragmatic definition of whole grains based on the fiber content of whole wheat, ie, ≥1.1 g of naturally occurring dietary fiber per 10 g of carbohydrate in the grain product (eg, bread, cracker, etc).

Fish

Fish and other seafood contain several healthful constituents, including specific proteins, unsaturated fats, vitamin D, selenium, and long-chain omega-3 polyunsaturated fatty acids (PUFAs), which include eicosapentaenoic acid (EPA; 20:5 omega-3) and docosahexaenoic acid (DHA; 22:6 omega-3). In humans, EPA and especially DHA are synthesized in low amounts (<5%) from their plant-derived precursor, α-linolenic acid (18:3 omega-3). Thus, tissue levels of EPA plus DHA are strongly influenced by their direct dietary consumption. Average EPA plus DHA contents of different seafood species vary by >10-fold. Fatty (oily) fish such as anchovies, herring, farmed and wild salmon, sardines, trout, and white tuna tend to have the highest concentrations.

In vitro and animal experiments suggest that fish oil has direct antiarrhythmic effects, but trials to establish direct antiarrhythmic effects in patients with preexisting arrhythmias have been inconsistent. In human trials, fish oil lowers triglyceride levels, increases diastolic BP, and resting heart rate. Observational and RCT evidence suggests that fish or fish oil consumption may also reduce inflammation, improve endothelial function, normalize heart rate variability, improve myocardial relaxation and efficiency, and, at high doses, limit platelet aggregation.

Consistent with these physiological benefits, habitual fish consumption is associated with lower incidence of CHD and ischemic stroke, especially risk of cardiac death (Figure 1), among generally healthy populations. Compared with no fish consumption, consumption of ~250 mg/d EPA plus DHA from fish is associated with 36% lower CHD mortality. Fish and fish oil are among only a handful of dietary factors for which both long-term observational studies and RCTs of CVD outcomes have been performed (Table 2). Four of 8 large RCTs of fish or fish oil, including participants with and without prevalent heart disease, documented significant reductions in CHD events. Several of these RCTs had relevant limitations. In meta-analyses of RCTs, fish oil supplementation significantly reduced cardiac mortality, including fatal CHD and sudden cardiac death. Overall, these findings are concordant with long-term observational studies of habitual fish intake in generally healthy populations and with physiological benefits of fish or fish oil in intervention...
Consumption of commercially fried fish or fish sandwiches has not been linked to benefits, possibly because of lower EPA plus DHA content or other added ingredients.39,51–54

studies (Table 2). Whether benefits of eating fish can be fully reproduced by fish oil supplements is not yet established.
Evidence for possible adverse cardiovascular effects of methylmercury found in a few fish species is limited and conflicting; if present, such effects do not appear to outweigh the net cardiovascular benefits of fish consumption.10,54a

### Nuts

Nuts contain several bioactive constituents that could improve cardiometabolic health, including extent of processing, food structure, dietary fiber content, content of bran and germ, and glycemic response to ingestion. This Figure presents a proposed taxonomy to integrate these various characteristics. Types of foods with evidence for cardiometabolic benefits are shaded green, and those with evidence for adverse cardiometabolic effects are shaded red. Intact and minimally processed whole grains (darker green; ie, greater benefits) may plausibly have greater benefits than milled whole grains (lighter green; ie, lesser benefits) because of intact food structure and lower glycemic response; refined sugars in liquid form (darkest red, ie, greatest harms) may have greater adverse effects than refined grains, starches, and sugars (lighter red; ie, lesser harms) because of particularly unfavorable effects on satiety and weight gain. † Both simple and complex refined carbohydrates induce similarly high glycemic responses following ingestion and, in amounts typically consumed in Western diets, induce de novo lipogenesis in the liver, ie, the conversion of carbohydrates to fat. Compared to glucose, fructose produces smaller blood glycemic responses but more strongly stimulates de novo lipogenesis. Animal-experimental and limited human studies suggest that fructose, which represents about half of all sugars in refined sugars such as either high fructose corn syrup or sucrose (eg, cane sugar, beet sugar), may have additional adverse effects on hepatic steatosis and insulin resistance. Corn provides reasonable fiber and modestly lower glycemic responses than many types of potatoes. Yams and sweet potatoes are not included herein because of higher nutrient contents and lower glycemic responses to ingestion.

<table>
<thead>
<tr>
<th>Type</th>
<th>Processing and Structure</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intact whole grains</td>
<td>Whole grain with the bran, germ, and endosperm from the natural cereal intact</td>
<td>Brown rice, bulgur wheat, amaranth, wheat berries</td>
</tr>
<tr>
<td>Minimally processed whole</td>
<td>Some processing is performed to improve palatability or digestibility yet the bran and</td>
<td>Stone-ground whole wheat bread, cracked wheat, steel-cut oats</td>
</tr>
<tr>
<td>grains</td>
<td>germ remain partially intact</td>
<td></td>
</tr>
<tr>
<td>Milled whole grains</td>
<td>The whole grain, including bran, germ, and endosperm, is milled to fine flour</td>
<td>Most commercially available whole grain breads, whole grain breakfast cereals, whole grain pasta</td>
</tr>
<tr>
<td>Refined grains†</td>
<td>The bran and germ are removed during processing, leaving the endosperm comprised</td>
<td>White bread, white rice, most ready-to-eat breakfast cereals, instant oatmeal, regular pasta.</td>
</tr>
<tr>
<td>Starchy vegetables†</td>
<td>Plants that have been bred or engineered to contain high levels of starch with relatively low dietary fiber and micronutrients</td>
<td>Potatoes, corn</td>
</tr>
<tr>
<td>Refined sugars†</td>
<td>Natural and industrially-produced mono- and di-, and oligosaccharides, including</td>
<td>Candies, other sugars added to foods</td>
</tr>
<tr>
<td></td>
<td>sucrose, glucose, fructose, high fructose corn syrup, maltose, dextrose, and maltodextran</td>
<td></td>
</tr>
<tr>
<td>Sweetened refined grains†</td>
<td>Refined grains with added refined sugars</td>
<td>Sweetened breakfast cereals, grain-based desserts (cakes, cookies, pies, doughnuts, sweet rolls, muffins)</td>
</tr>
<tr>
<td>Refined sugars in liquid</td>
<td>Natural and industrially-produced mono- and disaccharides in liquid form</td>
<td>Sugar-sweetened beverages, including sodas, iced teas, sports drinks, and fruit drinks</td>
</tr>
<tr>
<td>form†</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Evidence for possible adverse cardiovascular effects of methylmercury found in a few fish species is limited and conflicting; if present, such effects do not appear to outweigh the net cardiovascular benefits of fish consumption.10,54a

### Figure 2

Types of processing and structure of grains, sugars, and starches. A major research and policy gap is the absence of one accepted taxonomy to define whole grains or carbohydrate quality that incorporates their various characteristics that can influence cardiometabolic health, including extent of processing, food structure, dietary fiber content, content of bran and germ, and glycemic response to ingestion. This Figure presents a proposed taxonomy to integrate these various characteristics. Types of foods with evidence for cardiometabolic benefits are shaded green, and those with evidence for adverse cardiometabolic effects are shaded red.
only modest reductions in diastolic BP (−2 mm Hg) and LDL cholesterol (−3%). Legumes provide an overall package of micronutrients, phytochemicals, and fiber that could plausibly reduce cardiometabolic risk. Further investigation of their effects in well-conducted prospective cohorts and RCTs is required.

Meats
Several constituents of red meats could increase cardiometabolic risk, including SFA, cholesterol, heme iron, and in processed meats, high levels of salt and other preservatives. Lower consumption of red meats is consistently part of overall dietary patterns associated with lower CVD risk (see “Dietary Patterns” and Table 2). In meta-analyses of prospective cohort studies, total red meat consumption was associated with overall nonsignificant trends toward higher risk of CHD and DM (Figure 1). When different types of meat were evaluated systematically, consumption of processed meats but not unprocessed red meats was associated with higher incidence of CHD and DM. These findings suggest that adverse effects of preservatives (eg, sodium, nitrates, and phosphates) and/or preparation methods (eg, high-temperature commercial cooking/frying) could influence health effects of meat consumption. In one observational analysis, both unprocessed and processed meat consumption were associated with higher CHD risk when such consumption replaced foods with cardiometabolic benefits, such as low-fat dairy, nuts, and fish.

Dairy Foods
Some short-term RCTs have evaluated the potential benefits of dairy consumption for satiety or weight loss, with inconsistent and inconclusive findings. Multicomponent dietary interventions that included daily intake of low-fat dairy foods significantly lowered BP, lipid levels, and insulin resistance and improved endothelial function, independent of changes in weight. In such multicomponent interventions, the specific benefits of dairy foods cannot be quantified separately. Nonetheless, consistent with physiological benefits, higher dairy consumption was associated with lower risk of both stroke and DM in long-term observational cohorts (Figure 1).

The active constituents for such cardiometabolic benefits are not established. On the basis of lower content of calories, SFA, and cholesterol, together with no established nutritional advantage of whole-fat dairy, most dietary guidelines and scientific organizations recommend low-fat or nonfat dairy consumption. These guidelines generally recommend dairy foods as a source of selected nutrients (eg, calcium, vitamin D, protein, potassium, magnesium, and other vitamins) rather than based on equally or more relevant evidence for their effects as a whole food that may reduce cardiometabolic risk. In long-term observational studies, a lower risk of DM and metabolic abnormalities has been variably linked to consumption of low-fat dairy, whole-fat dairy, or both. Conjugated linoleic acid and calcium were proposed as potential mediators, but RCTs have demonstrated very small benefits or even adverse cardiometabolic effects of these factors. Dairy foods contain a complex assortment of other ingredients that could each play a role in lowering cardiometabolic risk, including specific fatty acids, proteins and peptides, vitamins, and other nutrients. Potentially varying health effects of specific dairy foods (eg, milk, yogurt, cheese, and butter) also require further study.

Sugar-Sweetened Beverages
Evidence from ecological comparisons and prospective cohorts supports positive associations of sugar-sweetened beverage intake with adiposity in children and adults. In the United States between 1965 and 2002, when overweight/obesity was increasing rapidly, the proportion of total dietary calories consumed from beverages increased from 11.8% to 21.0%, or ~222 calories/d per person. Most of this increase was from sugar-sweetened beverages (60%), such as sodas/colas, sweetened fruit drinks, and sports drinks, followed by alcohol (31%) and 100% fruit juices (9%); calories from other beverages (eg, milk) did not increase. The average American teenage boy drinks ~24 oz/d (300 kcal) of sugar-sweetened beverages; the average American teenage girl, ~16 oz/d (200 kcal). Most sugar-sweetened beverage intake by children occurs at home, not at school.

Limited short-term trials suggest that compared with solid foods, calories in liquid form may be less satiating and thereby increase the total amount of daily calories consumed. Sugar-sweetened beverage intake can also displace more healthful beverages, such as milk. Several RCTs of small (n=15) to moderate (n=644) sizes have demonstrated that reduced sugar-sweetened beverage intake improves weight loss or reduces weight gain in both children and adults. In one multicomponent lifestyle intervention, each 1-serving/d reduction in sugar-sweetened beverages was associated with 0.65-kg greater weight loss. In a meta-analysis of prospective cohorts, higher sugar-sweetened beverage intake was associated with higher incidence of DM and metabolic syndrome. One cohort observed a positive association between sugar-sweetened beverage intake and incident CHD. The combination of highly refined carbohydrate calories, a liquid form that may minimize satiety, absence of other beneficial nutrients/constituents, displacement of more healthful beverages, and very high intake in many population subgroups renders reduction in sugar-sweetened beverages an important dietary target for improving individual and population cardiometabolic health.

Alcohol
Alcohol use has been related to both beneficial and adverse cardiovascular outcomes. Habitual heavy alcohol intake is cardiotoxic, causing a large portion of nonischemic dilated cardiomyopathies in many nations. The ensuing ventricular dysfunction is often irreversible, even when alcohol consumption is stopped; continued drinking in such patients is associated with high mortality. Both acute binges and higher habitual intake of alcohol have also been associated with higher risk of atrial fibrillation. Conversely, in controlled trials and in the absence of weight gain, modest alcohol use raises high-density lipoprotein cholesterol, reduces systemic inflammation, and improves insulin resistance. Consistent with these effects,
compared with nondrinkers, individuals who drink alcohol moderately (up to ~2 drinks/d for men and ~1 drink/d for women) experience a lower incidence of CHD and DM. In observational studies, benefits of moderate alcohol intake could be overestimated, because the comparison category of nondrinkers often includes former drinkers and other individuals who avoid alcohol because of poor health, and because individuals burdened by negative effects of alcohol are generally underrepresented in such long-term studies, for which participation requirements favor healthier individuals. Nonetheless, the consistently lower event rates in observational studies and demonstrated physiological benefits in RCTs provide concordant evidence that moderate alcohol use confers at least some cardiometabolic benefit.

Experimental studies suggest that some nonalcohol components, such as resveratrol in wine, could have potential benefits, but evidence from both RCTs of risk factors and prospective cohort studies of clinical end points are most consistent with direct effects of alcohol itself. For example, moderate intakes of wine, beer, and liquor have each been associated with lower CHD risk in different populations. The pattern of drinking appears quite relevant, with lowest cardiovascular risk seen among individuals who drink moderately on several days of the week, rather than among irregular or binge drinkers. Because of alcohol-related accidents, homicides, and suicides, especially among younger adults, alcohol use has an overall net adverse effect on population mortality. Thus, alcohol use is not advisable as a population-based strategy to reduce CVD risk. For adults who already drink alcohol, no more than moderate use can be encouraged. Avoidance of weight gain should also be reinforced, because an average serving of alcohol contributes ~120 to 200 kcal that, as discussed previously, may be less satiating than calories from solid foods.

**Dietary Patterns**

An impressive and expanding body of evidence demonstrates that overall dietary patterns can improve health and prevent CVD (Table 2). Several healthful dietary patterns have been identified, often derived by different research approaches but sharing several key characteristics, including an emphasis on fruits, vegetables, other plant foods such as beans and nuts, and (in many patterns) whole grains and fish; with limited or occasional dairy products; and often with limited red meats or processed meats and fewer refined carbohydrates and other processed foods (Table 3). These dietary patterns are each generally consistent with food-based priorities for CVD health (Table 1). In RCTs, consumption of such dietary patterns substantially improves multiple cardiovascular risk factors (Figure 3).

**Dietary Approaches to Stop Hypertension**

DASH (Dietary Approaches to Stop Hypertension) dietary patterns emphasize fruits, vegetables, and low-fat dairy products; include whole grains, poultry, fish, and nuts; and are lower in red meat, sweets, and sugar-sweetened beverages. The original DASH diet was low in total fat (27% energy) and higher (55% energy) in carbohydrate; additional DASH diet patterns have been evaluated that exchange ~10% energy of carbohydrate for vegetable sources of monounsaturated fat or protein. In controlled feeding trials, each of these DASH diets significantly lowered BP and improved blood lipids compared with usual Western diets (Figure 3), which highlights the greater importance of specific food choices rather than macronutrient composition for maximization of CVD benefits. BP reduction was greatest when DASH diets were combined with reduced sodium intake. In multiple observational studies, greater adherence to DASH dietary patterns was associated with lower risk of CVD. Unfortunately, very few US adults, even those with elevated BP, currently eat a diet consistent with DASH dietary patterns.

**Mediterranean**

Diverse agricultural patterns, cultures, and countries bordering the Mediterranean Sea preclude defining any single Mediterranean dietary pattern or simple criteria. Mediterranean diets have also changed over time: In Crete, a Mediterranean island with historically low rates of CHD, diets now contain less fruit and olive oil and more meats than diets of earlier generations, with associated increases in serum cholesterol and adiposity among these modern residents. Many traditional Mediterranean diets share common characteristics, including an emphasis on vegetables, fruits, breads/cereal foods usually made from wheat, nuts, and olive oil; sometimes including fish and (in non-Islamic countries) wine with meals; and being lower in saturated fat, cholesterol, and meats. Ecological comparisons and prospective cohort studies consistently demonstrate inverse associations between consumption of traditional Mediterranean diets and risk of CHD, stroke, and total mortality. In RCTs, traditional Mediterranean diets improve several physiological risk factors (Figure 3).

**Vegetarian**

Several types of vegetarian diets are consumed around the world, including those consumed by pesco-vegetarians (who consume fish); lacto-ovo-vegetarians (who consume milk and eggs); and strict vegans (who consume no animal products). Potentially different cardiometabolic effects of these different vegetarian diets are not established. Few RCTs of vegetarian diets have been performed (Table 2). Two small trials (n = 58 and n = 20 subjects, respectively) demonstrated reductions in BP with vegetarian diets versus typical Western diets. Three other trials found no differences between lacto-vegetarian or vegan diets versus conventional dietary recommendations for improving weight loss, BP, blood lipids, or insulin resistance. In several observational studies, vegetarians experienced improved health outcomes compared with nonvegetarians. Several characteristics of vegetarian diets could account for these relationships, including greater inclusion of plant-based foods and fewer meats, processed meats, and other processed and fast foods. Because vegetarians are often generally more health conscious, other lifestyle characteristics, in addition to diet, could contribute to observed lower rates of CVD. Overall, vegetarian diets have been studied less extensively than DASH or Mediterranean patterns, and although the foods that are not
<table>
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<tr>
<th>Citation</th>
<th>Swain et al, 2008&lt;sup&gt;109&lt;/sup&gt;</th>
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<th>Trichopoulou et al, 2003&lt;sup&gt;110&lt;/sup&gt;</th>
<th>Nunez-Cordoba et al, 2009&lt;sup&gt;109a&lt;/sup&gt;</th>
<th>Fung et al, 2009&lt;sup&gt;110b&lt;/sup&gt;</th>
<th>Craig et al, 2009&lt;sup&gt;110c&lt;/sup&gt;</th>
<th>Wilcox et al, 2007&lt;sup&gt;111&lt;/sup&gt; and 2009&lt;sup&gt;111a&lt;/sup&gt;</th>
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<tr>
<td>Emphasizes</td>
<td>Nutrient‐dense vegetables, fruits, whole grains, fish, and low‐fat dairy products</td>
<td>Nutrient‐dense vegetables, fruits, whole grains, fish, and low‐fat dairy products</td>
<td>Nutrient‐dense vegetables, fruits, whole grains, fish, and low‐fat dairy products</td>
<td>Vegetables, beans, fruits, nuts and seeds, fish and seafood, cereals, olive oil</td>
<td>Plant foods, vegetables, fruits, breads, other cereals, potatoes, beans, nuts and seeds, olive oil, and fish</td>
<td>Plant foods, including fruits, vegetables, legumes, nuts, grains</td>
<td>Plant foods, including fruits, vegetables, legumes, nuts, grains</td>
<td>Rice, legumes, soy foods, vegetables, seaweed, and fish</td>
</tr>
<tr>
<td>Includes</td>
<td>Lean meat</td>
<td>Lean meat</td>
<td>Lean meat, red wine</td>
<td>Cheese, yogurt, red wine</td>
<td>Lean meat</td>
<td>Fish (pesco vegetarians); dairy and eggs (lacto‐ovo‐vegetarians)</td>
<td>Fruit, meat, and eggs</td>
<td></td>
</tr>
<tr>
<td>Limits (small amounts)</td>
<td>Processed meats</td>
<td>Processed meats</td>
<td>Processed meats</td>
<td>Meats, dairy</td>
<td>Red meats, sweets</td>
<td>Potatoes</td>
<td>Red meats, poultry</td>
<td>Milk products</td>
</tr>
<tr>
<td>Carbohydrates, % energy</td>
<td>8</td>
<td>48</td>
<td>48</td>
<td>ND</td>
<td>47</td>
<td>39.1</td>
<td>‡</td>
<td>79</td>
</tr>
<tr>
<td>Protein, % energy</td>
<td>15</td>
<td>15</td>
<td>25</td>
<td>ND</td>
<td>18</td>
<td>15.1</td>
<td>‡</td>
<td>13</td>
</tr>
<tr>
<td>Total fat, % energy</td>
<td>27</td>
<td>37</td>
<td>27</td>
<td>42.9</td>
<td>33</td>
<td>ND</td>
<td>‡</td>
<td>8</td>
</tr>
<tr>
<td>Saturated fat, % energy</td>
<td>6</td>
<td>6</td>
<td>6</td>
<td>13.3</td>
<td>10</td>
<td>10 (including trans)</td>
<td>‡</td>
<td>2.0</td>
</tr>
<tr>
<td>Monounsaturated fat, % energy</td>
<td>13</td>
<td>21</td>
<td>13</td>
<td>22.7</td>
<td>15</td>
<td>9.5</td>
<td>‡</td>
<td>2.3</td>
</tr>
<tr>
<td>Polyunsaturated fat, % energy</td>
<td>8</td>
<td>10</td>
<td>8</td>
<td>6.9</td>
<td>5.1</td>
<td>ND</td>
<td>‡</td>
<td>3.5</td>
</tr>
<tr>
<td>Cholesterol, mg</td>
<td>150</td>
<td>150</td>
<td>150</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>‡</td>
<td>ND</td>
</tr>
<tr>
<td>Dietary fiber, g</td>
<td>29</td>
<td>29</td>
<td>29</td>
<td>ND</td>
<td>29</td>
<td>20</td>
<td>‡</td>
<td>22</td>
</tr>
<tr>
<td>Potassium, mg</td>
<td>4450</td>
<td>4450</td>
<td>4450</td>
<td>ND</td>
<td>4589</td>
<td>ND</td>
<td>‡</td>
<td>2623</td>
</tr>
<tr>
<td>Calcium, mg</td>
<td>1181</td>
<td>1181</td>
<td>1181</td>
<td>ND</td>
<td>1028</td>
<td>ND</td>
<td>‡</td>
<td>315</td>
</tr>
<tr>
<td>Magnesium, mg</td>
<td>473</td>
<td>473</td>
<td>473</td>
<td>ND</td>
<td>396</td>
<td>ND</td>
<td>‡</td>
<td>317</td>
</tr>
<tr>
<td>Sodium, mg</td>
<td>2190</td>
<td>2190</td>
<td>2190</td>
<td>ND</td>
<td>2532</td>
<td>ND</td>
<td>‡</td>
<td>2370</td>
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</table>
### Table 3. Continued

<table>
<thead>
<tr>
<th>Foods/day†</th>
<th>DASH (Higher Carbohydrate)</th>
<th>DASH (Higher Monounsaturated Fat)</th>
<th>DASH (Higher Protein)</th>
<th>Traditional Mediterranean (Greece)*</th>
<th>Traditional Mediterranean (Spain)</th>
<th>Mediterranean (US)</th>
<th>Vegetarian</th>
<th>Traditional Japanese</th>
<th>Traditional Okinawan</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vegetables, total, servings</td>
<td>4.4</td>
<td>6.3</td>
<td>5.4</td>
<td>7.5</td>
<td>2.8</td>
<td>4.4</td>
<td>‡</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>Dark green</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>‡</td>
<td>&lt;0.1 (seaweed)</td>
<td>&lt;0.1 (seaweed)</td>
</tr>
<tr>
<td>Red orange</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>‡</td>
<td>0.5 (Asian sweet potatoes)</td>
<td>6.6 (Asian sweet potatoes)</td>
</tr>
<tr>
<td>Other</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>‡</td>
<td>1.3; plus 0.3 (pickled vegetables)</td>
<td>0.9 (pickled vegetables)</td>
</tr>
<tr>
<td>Starchy</td>
<td>0.2</td>
<td>0.4</td>
<td>0.3</td>
<td>0.9</td>
<td>ND</td>
<td>No potatoes</td>
<td>‡</td>
<td>0.3 (other potatoes)</td>
<td>&lt;0.1 (other potatoes)</td>
</tr>
<tr>
<td>Fruits and juices, total, servings</td>
<td>6.6</td>
<td>4.8</td>
<td>3.8</td>
<td>4.7 (fruits and nuts, excluding juices)</td>
<td>3.5 (fruit and juice plus 0.3 (dried fruit, nuts))</td>
<td>3.2</td>
<td>‡</td>
<td>0.2</td>
<td>&lt;0.1</td>
</tr>
<tr>
<td>Grains, total, oz</td>
<td>5.3</td>
<td>4.3</td>
<td>5.0</td>
<td>5.3</td>
<td>2.5</td>
<td>ND</td>
<td>‡</td>
<td>2.4 plus 1.7 (rice)</td>
<td>1.1 plus 0.9 (rice)</td>
</tr>
<tr>
<td>Whole grains, oz</td>
<td>3.9</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>1.6</td>
<td>‡</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>Dairy products, total (servings)</td>
<td>2.0</td>
<td>1.9</td>
<td>2.3</td>
<td>1.0</td>
<td>2.1</td>
<td>ND</td>
<td>‡</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>Whole-fat</td>
<td>0.1</td>
<td>0.1</td>
<td>0.2</td>
<td>1.0</td>
<td>0.8</td>
<td>ND</td>
<td>‡</td>
<td>&lt;0.1</td>
<td>&lt;0.1</td>
</tr>
<tr>
<td>Low-fat</td>
<td>1.9</td>
<td>1.8</td>
<td>2.1</td>
<td>&lt;0.1</td>
<td>1.3</td>
<td>ND</td>
<td>‡</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>Other protein sources, oz</td>
<td>Meat</td>
<td>0.9</td>
<td>1.0</td>
<td>1.1</td>
<td>3.5</td>
<td>3.6</td>
<td>2.4</td>
<td>0</td>
<td>0.4</td>
</tr>
<tr>
<td>Poultry</td>
<td>1.6</td>
<td>1.8</td>
<td>2.6</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>0</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>Eggs</td>
<td>0.4</td>
<td>0.2</td>
<td>1.9</td>
<td>0.6</td>
<td>1.9</td>
<td>ND</td>
<td>‡</td>
<td>0.3</td>
<td>&lt;0.1</td>
</tr>
<tr>
<td>Fish/seabed</td>
<td>1.3</td>
<td>1.3</td>
<td>1.5</td>
<td>0.8</td>
<td>2.4</td>
<td>1.5</td>
<td>‡</td>
<td>2.1</td>
<td>0.6</td>
</tr>
<tr>
<td>Legumes</td>
<td>1.8</td>
<td>1.0</td>
<td>3.0</td>
<td>&lt;0.1</td>
<td>0.4</td>
<td>0.3</td>
<td>‡</td>
<td>0.4 (including soy)</td>
<td>0.3 (including soy)</td>
</tr>
<tr>
<td>Nuts and seeds</td>
<td>1.1</td>
<td>1.1</td>
<td>1.1</td>
<td>See fruit above</td>
<td>See fruit above</td>
<td>0.5</td>
<td>‡</td>
<td>&lt;1 g</td>
<td>&lt;0.1</td>
</tr>
<tr>
<td>Soy products</td>
<td>0.2</td>
<td>0.1</td>
<td>1.2</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>‡</td>
<td>See legumes</td>
<td>See legumes</td>
</tr>
</tbody>
</table>

(Continued)
Table 3. Continued

<table>
<thead>
<tr>
<th>Dietary Pattern</th>
<th>DASH (Higher Carbohydrate)</th>
<th>DASH (Higher Monounsaturated Fat)</th>
<th>DASH (Higher Protein)</th>
<th>Traditional Mediterranean (Greece)*</th>
<th>Traditional Mediterranean (Spain)</th>
<th>Mediterranean (US)</th>
<th>Vegetarian</th>
<th>Traditional Japanese</th>
<th>Traditional Okinawan</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oils and solid fats, total, g</td>
<td>46</td>
<td>70</td>
<td>31</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>‡</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>Margarines and spreads</td>
<td>7</td>
<td>22</td>
<td>4</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>‡</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>Salad dressing, mayonnaise</td>
<td>20</td>
<td>10</td>
<td>17</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>‡</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>Cooking oils, except olive oil</td>
<td>11</td>
<td>13</td>
<td>2</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>‡</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>Olive oil</td>
<td>8</td>
<td>25</td>
<td>8</td>
<td>40.3</td>
<td>19.0</td>
<td>ND</td>
<td>‡</td>
<td>ND</td>
<td>ND</td>
</tr>
<tr>
<td>Added sugar, candy, g</td>
<td>19</td>
<td>25</td>
<td>20</td>
<td>24.3</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>7.7</td>
<td>3.4</td>
</tr>
<tr>
<td>Alcohol, g</td>
<td>ND</td>
<td>ND</td>
<td>ND</td>
<td>7.9</td>
<td>7.1 (Red wine)</td>
<td>7.3</td>
<td>ND</td>
<td>30.0 (flavors and alcohol)</td>
<td>7.8 (flavors and alcohol)</td>
</tr>
</tbody>
</table>

This table was adapted, with several additions and modifications, from the 2010 Report of the Dietary Guidelines Advisory Committee on the Dietary Guidelines for Americans, US Department of Agriculture, Agricultural Research Service, available at http://www.cnpp.usda.gov/DGAs2010-DGACReport.htm.10 DASH indicates Dietary Approaches to Stop Hypertension; ND, not described.

*Values are based on the average (mean) consumption levels in the population, not on the optimal consumption levels for a traditional Mediterranean diet.

†Adjusted to an average 2000 kcal/d diet.

‡Vegetarian diets are defined only by what is not consumed (ie, meats and, variably, dairy, eggs, and/or fish). Thus, although many vegetarians tend to be health-conscious and may consume higher amounts of fruits, vegetables, nuts, and legumes, what actually is consumed can vary considerably and no typical vegetarian diet can be reliably defined.
Carbohydrate Amount and Quality

Among the most important new insights related to diet and cardiometabolic health is the growing evidence characterizing the importance of carbohydrate quality. Although carbohydrates have traditionally been classified as simple (eg, monosaccharides and disaccharides) versus complex (eg, starch and glycogen), several additional characteristics are relevant in determining cardiometabolic effects. These include dietary fiber content; bran and germ content; food structure (eg, intact, minimally processed, refined, or liquid); and potentially glycemic responses or induction of hepatic de novo lipogenesis following ingestion (Figure 2).

Although the most relevant dimensions of carbohydrate quality, along with a corresponding taxonomy, remain uncertain, it is clear that several aspects of carbohydrate quality influence cardiometabolic health. The effects of dietary fiber are best established, including lowering of serum triglycerides, LDL cholesterol, blood glucose, and BP.31,32 Health effects of bran and germ and food structure have been discussed (see “Whole Grains”). Emerging and compelling evidence suggests that foods with higher glycemic responses can adversely affect blood glucose control and triglycerides and perhaps LDL cholesterol levels, inflammation, endothelial function, and fibrinolysis.33,120–123 Further investigation is needed to establish the clinical relevance and potential independence of such effects from dietary fiber or bran/germ content. Animal experiments and limited human studies suggest that fructose—50% of the sugar in sucrose and 45–55% in high fructose corn syrup—may induce hepatic steatosis and insulin resistance more strongly than glucose.132a Consumption of refined sugars in liquid form appears especially problematic for weight gain (see “Sugar-Sweetened Beverages”). Emerging evidence suggests that the effects of carbohydrate quality may be most relevant in the immediate postprandial period and among individuals more predisposed to insulin resistance.129–132

Whichever metrics are used, it is evident that carbohydrate quality influences cardiometabolic health. Because multiple characteristics appear to be relevant, these health effects are unlikely to be replicated by simple extraction of individual factors (eg, fiber) or nutrients (eg, magnesium) and consumption of these as supplements or food additives.16,17 Important health benefits of consuming energy from higher-quality carbohydrate foods may also relate to substitution, ie, the replacement of more highly refined, lower-quality carbohydrates. Thus, simply eating more whole grains, or consuming individual constituents as supplements, is unlikely to produce the same benefits as substituting whole-grain, higher-quality carbohydrates for refined, lower-quality carbohydrates (Figure 2).

Fat Amount and Quality

Traditionally, dietary recommendations have advised lower total fat intake, primarily because of higher caloric density of fats compared with protein or carbohydrate. However, the type of fats consumed appears to be far more relevant for cardiometabolic health than the proportion of calories consumed from total fat.61,133–135 Calorie for calorie, industrially produced trans fats from partially hydrogenated vegetable
oils have the strongest adverse relations with CHD risk, and consumption of such fats should be as close to zero as possible. Conversely, seafood-derived omega-3 PUFAs have strong inverse relations with CHD mortality, and a recent World Health Organization Expert Consultation recommended that all adults consume at least 250 mg/d EPA plus DHA from seafood. Some evidence supports cardiovascular benefits of plant-derived omega-3 fats, but compared with naturally occurring omega-3 from seafood, the potency of plant-based omega-3 appears weaker, and mixed findings preclude strong conclusions.

On the basis of animal experiments, ecological comparisons, and RCTs documenting effects on LDL cholesterol, greater SFA consumption has traditionally been associated with increased CHD risk. Conversely, 3 recent meta-analyses of all available prospective observational studies found that individuals consuming the highest amounts of SFA had a similar incidence of CVD events as those consuming the lowest amounts. Although the reasons for these conflicting results remain controversial, growing evidence suggests that cardiometabolic effects of SFA consumption are modified by which nutrients (or foods) are decreased or increased in exchange for SFA (Table 2). For example, in most prospective observational studies, SFA consumption was compared with the average background diet, which in these studies was often heavily composed of less healthful refined carbohydrates that induce their own adverse cardiometabolic effects (see above).

Several lines of evidence support this explanation. In pooled individual-level data from 11 observational cohorts, consumption of PUFA was associated with lower CHD risk than SFA, as would be expected. However, SFA consumption, traditionally considered highly atherogenic, had relatively similar associations with CHD risk as total carbohydrate, the most common (calorically predominant) background macronutrient. Similar effects of SFA and carbohydrates on the ratio of total to HDL cholesterol have been documented, and may contribute to this finding. Overall carbohydrate quality and fiber content (as well as underlying predisposition to insulin resistance) would be expected to modify the relative effects of SFA vs. carbohydrate. In a recent observational analysis, SFA consumption was associated with a trend toward higher CHD risk than low-glycemic, higher-fiber (eg, whole grain) carbohydrates and with lower CHD risk when it replaced high-glycemic (eg, highly refined) carbohydrates. On the basis of these findings and high proportions of refined carbohydrates in most diets, a focus on reducing SFA consumption, traditionally a major public health goal, needs to be paired with decreasing consumption of refined carbohydrates, starches, and sugars. More explicitly, recommendations to replace SFA with PUFA (see below) and replace refined carbohydrates/starches and sugars with less refined, higher-fiber carbohydrate foods are relevant to reducing cardiometabolic risk.

The importance of prioritizing healthier food choices is especially clear (Table 1). Although different individual SFAs (eg, palmitic acid or stearic acid) have different effects on blood lipids, current evidence is insufficient to determine whether these individual SFAs have differential effects on disease outcomes. The health effects of many other individual fatty acids also require further study. Limitations in dietary assessment methodology persist, and advances in such methods in combination with use of objective biomarkers will enable more definitive investigation of these and related questions.

Cardiovascular effects of total PUFAs, largely derived from certain vegetable oils (eg, soybean, safflower, and corn), have been evaluated extensively. PUFA consumption improves levels of blood lipids and lipoproteins, is associated with lower CVD event rates in prospective cohort studies, and reduces clinical events as a replacement for SFA in RCTs. Consumption of monounsaturated fatty acids has been shown to favorably affect levels of blood lipids and lipoproteins, improved insulin resistance in some but not

other RCTs,146–148 and was related to lower CVD risk (when consumed from vegetable oils) in ecological studies.149 However, because consumption of monounsaturated fatty acids has been shown to increase atherosclerosis in nonhuman primates150 and has mixed relations with clinical CVD events in long-term cohort studies, some caution is needed.61,142 Also, because monounsaturated fat is derived from both animal (eg, meats and dairy) and vegetable (eg, olive oil, and canola) sources, health effects may plausibly vary depending on the types of foods consumed. For example, potential adverse effects of saturated fat, cholesterol, heme iron, or preservatives in meats could counter cardiometabolic benefits,65 whereas potential effects of phenolic compounds in olive oil could augment benefits.151 Thus, consumption of vegetable oils, including those that contain PUFA, plant-derived omega-3 fats, and MUFA, is an important feature of dietary patterns that reduce CVD risk (Table 1).

Sodium
Animal experiments, migration studies, ecological studies, longitudinal observational studies, RCTs, and meta-analyses confirm that higher salt (sodium chloride) consumption raises BP.152 Elevated BP is estimated to account for nearly two thirds of strokes and half of all CHD events.153 Individual large-scale trials and meta-analyses of trials have documented conclusively that several different BP-reduction therapies prevent stroke and CHD.154 This compelling body of evidence has led numerous organizations to conclude that salt reduction, through its effects on BP, should likewise prevent stroke and CHD.10,13 A meta-analysis of prospective observational studies supports a direct relationship between increased salt intake and CVD events.155 Limited evidence from trials, including long-term follow-up of salt-reduction trials, is also consistent with lower CVD event rates after salt reduction.156,157

In North America and Europe, 75% of sodium intake is derived from packaged, preprepared, or restaurant foods, with the rest being naturally occurring or added at home.158 In Asian countries, most sodium is from soy sauce or is added at home.158 Given ubiquitous sources of sodium in American and similar diets (Figure 4),159 policy-level approaches are well-suited for reduction efforts. Projected benefits are enormous. A national effort that reduced US salt intake by 3 g/d (~1.2 g/d less sodium) could annually prevent between 60 000 and 120 000 CHD events and 32 000 to 66 000 strokes, saving 194 000 to 392 000 quality-adjusted life-years and $10 to $24 billion in healthcare costs.160 Smaller reductions in salt intake are also estimated to produce large population benefits.160,161 Overall, the totality of evidence supports aggressive policy-level approaches to lower sodium intake as a means to prevent the CVD consequences of elevated BP. Optimal BP reduction occurs when sodium reduction is combined with a healthier food–based dietary pattern such as the DASH-type diet (Table 1).

Dietary Supplements
Use of dietary supplements, often at high or pharmacological doses, is commonplace, despite the absence of convincing evidence that supplements have health benefits. Several supplements have been evaluated in observational studies and RCTs as potential therapies to prevent CVD or other conditions (Table 4).46,84,162–166 Evaluated doses have often exceeded usual or even recommended dietary intakes, often under the presumptions that the greater the intake, the greater the benefit, and that risk of harm was negligible. However, substantial evidence has accrued that most of these supplements have few CVD benefits and that certain supplements, including β-carotene, calcium, and vitamin E, can even be harmful. Presently, only omega-3 PUFA can be recommended as a supplement for CVD prevention; optimal doses and target populations require further study. Overall, the current evidence does not support supplementary vitamin or

Table 4. Summary of Evidence of the Effects of Vitamin Supplements on Cardiovascular Disease

<table>
<thead>
<tr>
<th>Vitamin</th>
<th>Effect on CVD Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Beta-carotene</td>
<td>Some cohort studies have linked low serum levels or low dietary intake of beta-carotene with higher CVD risk. RCTs of beta-carotene supplements document no benefit in the general population and increased risk of lung cancer in patients who were at high risk of lung cancer.162</td>
</tr>
<tr>
<td>Calcium</td>
<td>Calcium supplementation with vitamin D3 is recommended as a means to prevent osteoporosis, especially in women. A recent meta-analysis of RCTs suggests that calcium supplementation may increase the risk of myocardial infarction.44</td>
</tr>
<tr>
<td>Folic acid, vitamin B6, vitamin B12</td>
<td>Emerging evidence from observational studies suggests that low serum vitamin D levels are associated with higher risk of CVD events. RCTs of vitamin D supplementation have not shown conclusive reductions in risk of CVD.164 Additional trials using higher doses of vitamin D supplementation are ongoing.</td>
</tr>
<tr>
<td>Vitamin E</td>
<td>Several prospective cohort studies have linked vitamin E consumption or supplementation with lower risk of CHD. RCTs have failed to show reductions in CVD events with supplemental vitamin E, and 2 meta-analyses suggest that high-dose vitamin E supplements may increase total mortality.162,164</td>
</tr>
<tr>
<td>Fish oils</td>
<td>Multiple cohort studies have documented an inverse relationship between fish intake and subsequent CHD, in particular CHD death.39 A meta-analysis of larger and multiple smaller RCTs, largely in higher-risk populations, demonstrated a reduction in total mortality with fish oil supplementation.39 Four of 8 large RCTs among patients with and without preexisting CVD demonstrated reductions in CHD events; several of these trials had potentially relevant limitations.46</td>
</tr>
<tr>
<td>Multivitamins</td>
<td>Although some cohort studies have observed lower CVD risk with multivitamin supplements, 5 RCTs, rated to be of fair to poor quality, have not documented any clear CVD benefit of multivitamin use in mixed populations.166</td>
</tr>
</tbody>
</table>

CVD indicates cardiovascular disease; RCT, randomized controlled trial; and CHD, coronary heart disease.
other supplement approaches, including food fortification, to duplicate the cardioprotective benefits of consumption of healthful foods.

**Energy Balance**

Energy balance (calories consumed versus expended) is the principal determinant of weight gain and adiposity. Basal and activity-related energy expenditure are determined by physical activity, body size, muscle mass, age, and sex. Appropriate energy consumption (total dietary calories per day) is determined by energy expenditure and goals for weight loss, gain, or stability. Some people may consume relatively high calories but have neutral or negative energy balance because of high expenditure, whereas others may consume relatively few calories but have positive energy balance because of low expenditure. Thus, change in weight (supplemented with waist circumference), rather than direct measurement of energy expenditure or consumption, is the most practical metric to assess energy balance.

Whether macronutrient composition (percent energy from total fat, carbohydrate, or protein) has an effect on energy balance was tested recently in RCTs. In early studies not designed to test weight loss, participants assigned to low-fat diets who also received intensive dietary counseling experienced greater short-term weight loss than control subjects who received no dietary counseling.\(^{167,168}\) However, RCTs designed to investigate weight loss and having equal-intensity interventions in each dietary arm have demonstrated that macronutrient composition has little independent effect on weight loss.\(^{169–172}\) Adherence to any weight loss diet, regardless of its macronutrient composition, was the best determinant of success.\(^{169–172}\) Long-term ad libitum dietary trials\(^{168}\) and prospective cohort studies\(^{173–175}\) support that macronutrient composition has little effect on long-term weight gain.

In contrast, behavioral factors, including lower intakes of fruits, vegetables, and whole grains; larger portion sizes; and greater intakes of sugar-sweetened beverages, processed snacks, energy-dense foods, fast food meals, and possibly trans fat,
appear to have a greater influence on body weight.87a,88,136,176–181 Thus, these features of dietary quality appear to impact dietary quantity.

Other individual, social, and environmental factors are also linked to energy imbalance.87a,182–184 These include television watching and lower average sleep duration, especially among children/adolescents; socioeconomic status and race/ethnicity; local environments, such as the presence of fast food restaurants, grocery stores, crime safety, parks or open spaces, and walking or biking paths; and influences of advertising, social norms, and work and home dynamics. Ultimately, these influences act through changes in diet or activity to influence weight change. For example, effects of television watching and poor sleep may be mediated predominantly by dietary changes.185–190 Although the quality of available time-trend data is suboptimal, the recent obesity pandemic appears temporally related to increased energy consumption in developed nations80 and to both increased consumption and decreased expenditure in some developing nations.3

A sustained energy imbalance of ~50 to 100 extra kcal/d may be enough to produce the weight gain observed in many individuals.191,192 The often small size of this “energy gap” makes unintentional gradual weight gain easy, but also means that small improvements in energy balance, sustained over the long term, could prevent or even reverse adiposity in many individuals and populations.87a,193

### Changing Behavior

A growing body of literature supports several effective strategies for successful behavior change.194–197 Evidence for individual-level approaches was reviewed recently (Table 5).197 Targeted goals are most effective for individuals.196,197 Clinical providers can help patients prioritize the most relevant foods and dietary patterns (Table 1) and perform simple office-based assessments to inquire about and help set dietary goals.198 Clinic-based strategies are facilitated by healthcare systems changes, including scheduled visits for individual/group education; sustained in-person, telephone, or electronic feedback; and quality and reimbursement guidelines that support behavior-change efforts.199,200

Although individual-based strategies can successfully improve an individual’s diet quality, public health strategies at community, state, and national levels are essential to produce a substantial and sustained population impact.196,201 Several approaches are effective (Table 6).152,196,202–214 Given the scope of prevailing adverse dietary patterns, population approaches are critical to achieving the AHA’s 2020 Strategic Goals.7 Overall, although more research on the best translational strategies is needed, sufficient evidence presently exists to implement several individual- and population-level approaches to improving diet.

### Conclusions

High-quality evidence is now available from multiple complementary research paradigms on the cardiometabolic effects of specific dietary factors, offering new and expanded insights into dietary priorities for preventing CVD and DM. Consumption of specific foods and dietary patterns appear especially beneficial (Table 1) and warrant a focus on foods and diet patterns appear especially beneficial (Table 1) and warrant a focus on foods and overall dietary patterns rather than individual nutrients or supplements. This focus should also help counter food industry manipulation of nutrient targets (eg, promotion of highly processed, “nutrient-fortified” foods/beverages). An approach based on foods and dietary patterns can also minimize paradoxical choices that may arise from single-nutrient approaches, such as avoiding nuts or vegetable oils because of concerns about SFA or total fat content while instead consuming processed low-fat deli meats, highly refined cereals/breads, and fat-free sugary desserts.6 Levels of selected individual nutrients in foods, such as sodium and industrially produced trans fats, can be efficiently reduced by strong policy strategies. Appropriate energy balance is also essential, which will require sustained approaches that target dietary quality (types of foods/beverages consumed), portion sizes, television watching, physical activity, and local environments.

Although gaps in knowledge remain, a substantial knowledge base informs relevant dietary priorities and behavior-change strategies for improving cardiometabolic health. Effective approaches are required to harness the interest and

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**Table 6. Evidence-Based Strategies for Promoting Population-Level Dietary Change to Reduce Cardiovascular Disease Risk**

<table>
<thead>
<tr>
<th>Strategy</th>
<th>Example</th>
</tr>
</thead>
<tbody>
<tr>
<td>Policies directly targeting selected nutrients to increase or reduce levels in foods</td>
<td><em>Slow down</em></td>
</tr>
<tr>
<td>Direct mandates or bans</td>
<td></td>
</tr>
<tr>
<td>Coordinated, multicomponent media, education, and labeling</td>
<td></td>
</tr>
<tr>
<td>Changes in the food environment</td>
<td><em>Nutrition labels</em></td>
</tr>
<tr>
<td>Increasing availability of healthier foods, eg, in cafeterias, grocery stores, or neighborhoods</td>
<td><em>Smarter labeling</em></td>
</tr>
<tr>
<td>Changes in food pricing by means of subsidies or taxation</td>
<td></td>
</tr>
<tr>
<td>Point-of-purchase prompts and education</td>
<td></td>
</tr>
<tr>
<td>Media and other educational strategies</td>
<td><em>Food labeling</em></td>
</tr>
<tr>
<td>Intensive mass media campaigns promoting one simple message</td>
<td></td>
</tr>
<tr>
<td>National logos or simple branding to identify healthier foods*</td>
<td></td>
</tr>
<tr>
<td>Provision of focused, targeted messages as part of a larger multicomponent strategy</td>
<td><em>Nutrition labels</em></td>
</tr>
<tr>
<td>Regulation of advertising to particular at-risk groups</td>
<td></td>
</tr>
<tr>
<td>Posting of information on food labels or menu boards†</td>
<td></td>
</tr>
<tr>
<td>Social and community supports†</td>
<td></td>
</tr>
<tr>
<td>Coordinated educational and environmental efforts in schools, workplaces, community centers, and religious centers</td>
<td><em>Nutrition labels</em></td>
</tr>
<tr>
<td>Group sessions and self-monitoring strategies</td>
<td></td>
</tr>
<tr>
<td>Multicomponent strategies†</td>
<td></td>
</tr>
<tr>
<td>Combinations of upstream policy measures, midstream environmental approaches and media campaigns, and downstream community approaches</td>
<td><em>Nutrition labels</em></td>
</tr>
</tbody>
</table>

*Such interventions experience greatest success when multiple stakeholders (community members, local organizations, policy makers) are involved throughout planning, implementation, and sustainability.196,214†Evidence that this strategy alters consumer behavior is still relatively limited, but this strategy appears to at least contribute to food industry choices in terms of what is offered.*

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energy of policy makers and the food industry to improve diet throughout the world. Although many diet-related prevention strategies are likely to be highly cost-effective for society, most policy makers and medical systems remain focused on downstream treatments that use drugs, devices, and surgeries. New strategic initiatives are needed to draw together all key stakeholders—the public, healthcare providers, food industry, and policy makers—to identify remaining barriers and develop operational plans to translate our considerable knowledge on the health effects of foods and dietary patterns and on behavior change into actual improvements in diet.

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Eating Patterns and Coronary Heart Disease Risk in Early Life: The Childhood and Adolescent Determinants of Adult Coronary Health Study


current interest in the role of eating patterns and Coronary Heart Disease (CHD) risk. The Childhood and Adolescent Determinants of Adult Coronary Health Study (CARDIA) is a longitudinal study with a cohort of 5,113 black and white adults aged 18-30 years at baseline. Financial support: NIDDK and NHLBI.


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Components of a Cardioprotective Diet: New Insights
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