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Nutrition Protocols for the Prevention of Cardiovascular Disease

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Cardiovascular disease is the number one cause of death in the United States, and prevention of cardiovascular disease is at the top of the public health agenda. Evidence shows that reducing the incidence of coronary heart disease with diet is possible. More than a half century of evidence from epidemiologic, experimental, and clinical trials pinpoints a positive correlation between lifestyle and dietary factors as they relate to blood lipids, blood pressure, and coronary heart disease risk, and a number of evidence-based nutrition guidelines have emerged. The National Cholesterol Education Program's Therapeutic Lifestyle Changes diet includes unsaturated fats, fiber, and plant sterols/stanols. The whole foods approach incorporates increased consumption of

fruits, vegetables, whole grains, and fish; and the American Heart Association guidelines emphasize functional foods like soy protein, nuts, and alcohol. These guidelines display the rationale for nutrition intervention as a primary prevention for cardiovascular disease. In addition, body weight, body mass index, waist circumference, and waist-to-hip ratio are examined as risk factors for cardiovascular disease. This article highlights key nutrients and lifestyle factors in preventing cardiovascular disease and identifies practical applications for clinicians. (*Nutr Clin Pract.* 2008;23:468-476)

Keywords: cardiovascular diseases; nutrition therapy; prevention and control; heart disease; adult

Cardiovascular disease (CVD) includes all diseases that affect the heart and blood vessels, such as coronary heart disease (CHD), coronary artery disease, dyslipidemia, and hypertension.¹ CVD remains the number one cause of death in the United States, and prevention of CVD is at the top of the public health agenda. The vast body of evidence shows that reducing the incidence of CHD with diet is possible.² The rationale for nutrition intervention, according to the National Cholesterol Education Program Adult Treatment Panel III (ATP III), is to reduce the risk factors for coronary atherosclerosis with primary prevention approaches—of which dietary factors play a prominent role.³ In 1999, ATP III recommended the Therapeutic Lifestyle Changes (TLC) diet and lifestyle (Table 1).

More than a half century of evidence from epidemiological, experimental, and clinical trials has pinpointed a positive correlation between lifestyle and dietary factors as they relate to blood lipid levels, blood pressure, and CHD.¹ Dietary patterns or the whole foods approach has emerged as a viable solution for CVD by affecting not only blood lipids but also nonlipid biomarkers, such as

inflammation. The last decade of evidence has revealed the central role of inflammation in all phases of the atherosclerotic process.⁴ Western dietary patterns, which are high in red and processed meat, sweets and desserts, potatoes and French fries, and refined grains, have been found to warm up inflammation, whereas prudent dietary practices, which are higher in fruits, vegetables, legumes, whole grains, poultry, and fish, have been found to cool it down.² In other words, dietary patterns high in refined starches, sugar, and saturated and trans fatty acids and poor in natural antioxidants and fiber from fruits, vegetables, and whole grains have been found to predispose susceptible people to increased incidence of CHD.² This article highlights the role of key nutrients and lifestyle factors in preventing CVD and identifies practical applications for clinicians.

Dietary Fiber

Extensive research and epidemiologic data have shown that dietary fiber has an inverse relationship to CVD risk, and thus dietary fiber is a functional food of the TLC diet.⁵ There are 2 types of dietary fiber, soluble and insoluble. Research shows that insoluble fiber has little to no effect on lowering cholesterol; however, soluble fiber does have an effect. As a class, soluble fibers such as oat bran, pectin, guar gum, and psyllium have been shown to reduce serum low-density lipoprotein (LDL) cholesterol by modest amounts, ranging

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Table 1. Therapeutic Lifestyle Changes Diet:
A Multifaceted Lifestyle Approach to Reduce
Risk of Coronary Heart Disease

Nutrient	Amount
Saturated fat	<7% of total calories
Polyunsaturated fat	Up to 10% of total calories
Monounsaturated fat	Up to 20% of total calories
Total fat	25%-35% total calories
Carbohydrate	50%-60% of total calories
Fiber	20-30 g/d
Protein	15% of total calories
Cholesterol	<200 mg/d
Calories	Balance energy intake and exercise ^a to maintain healthy weight and prevent weight gain.

^a Aim for at least 30-60 minutes of physical activity most days of the week (average 5 days per week).

from 3% to 10%. Total fiber also seems to be inversely related to cardiovascular disease.⁶ The ATP III guidelines recommend an intake of 10–25 g/d of soluble fiber.³

According to fiber studies, the viscosity of soluble fiber is the mechanism behind its ability to lower cholesterol and decrease dietary fat absorption in the intestines. In addition, soluble fiber seems to alter GI transit time and delay gastric emptying, which lead to a full feeling more quickly and for a longer period of time. The major benefit from eating fiber-rich foods may be a change in dietary pattern, resulting in a diet that is lower in saturated fat, trans fatty acids, and cholesterol, and higher in protective nutrients, such as unsaturated fatty acids, minerals, folate, and antioxidant vitamins.⁷

Jenkins et al⁷ concluded that consuming a high-fiber diet with 7.2 g of psyllium and 0.75 g of β -glucan daily reduced the risk of CVD by $4.2\% \pm 1.4\%$. The study also showed that following a high-fiber diet along with the National Cholesterol Education Program step II diet decreased total cholesterol 2.4% ($P = .015$). Although researchers are examining the role of soluble fiber in blood pressure, further research may be warranted. A 2005 meta-analysis of studies on fiber examined soluble fiber and its effect on blood pressure; the report showed modest reductions in systolic blood pressure of 1.13 mm Hg (95% confidence interval, -2.49 to 0.23) and in diastolic blood pressure of 1.26 mm Hg (-2.04 to -0.48). The greatest reductions in blood pressure tended to be in older adults (>40 years) and in hypertensive populations rather than in younger, normotensive ones.⁸ A 2001 study revealed a positive correlation between consumption of 16 g of total fiber with 7 g of soluble fiber coming from oats and a hypocaloric diet with significant reductions in mean systolic blood pressure over a 6-week period. The findings also revealed decreases in serum levels of total and LDL cholesterol. The results showed that adding fiber to a diet

intended for weight loss to reduce CVD risk improved not only blood pressure but also the overall blood lipid profile.⁹

A recent study examined the positive results of β -glucan on cholesterol by setting out to determine whether concentrated oat β -glucan that was separated from the whole oat grain (and thus was considered a “functional fiber”) had similar effects as dietary oat β -glucan that was not separated from the whole oat grain. The study showed that intake of 6 g of concentrated oat β -glucan significantly reduced serum total cholesterol (-0.3 ± 0.01 mmol/L) and LDL cholesterol (-0.3 ± 0.01 mmol/L) levels over a 6-week period. The overriding premise of this research was to find new ways to incorporate concentrated β -glucan from oats into food products other than cereals and to find acceptable ways for the public to increase consumption of soluble fiber.¹⁰ An additional result of the study’s findings was that the Institute of Medicine (IOM) suggested a change in fiber terminology from soluble and insoluble to viscous and fermentable to describe the physiochemical properties of fiber. This may help to better determine which fibers are the most effective at lowering cholesterol. Ten years ago, in a response to a petition submitted by the Quaker Oats company, the U.S. Food and Drug Administration (FDA) approved the first food-specific health claim for foods containing whole oat sources of soluble fiber (oats, oat bran, and oat flour) and reduced risk of CHD.¹¹ The FDA-approved health claim states that 3 g of soluble fiber daily from oatmeal (1½ cups cooked oatmeal), in a diet low in saturated fat and cholesterol, may reduce the risk of heart disease.¹¹ Since then many epidemiologic studies have proven that high levels of whole-grain consumption (≥ 3 servings per day) are associated with reductions in risk for CVD.¹¹

A recent review article¹² examined the existing research studies to determine whether various water-soluble fibers had the same hypocholesterolemic effects. β -Glucan, psyllium, pectin, and guar gum have all been shown to reduce serum LDL cholesterol levels. This literature review concluded that well-controlled intervention studies were adequate in supporting the claim that the various types of water-soluble fibers are effective in lowering serum LDL cholesterol concentrations without affecting high-density lipoprotein (HDL) cholesterol or triglyceride concentrations.¹²

The IOM guidelines recommend a fiber intake of 14 g per 1000 kcal.¹³ Clinical practice guidelines of the ATP III,³ the American Heart Association (AHA),¹⁴ and the American Diabetes Association¹⁵ recommend increased intake of soluble fiber as an integral component of LDL cholesterol-lowering therapy. The target dose of total dietary fiber should be 25 g/d. For hypercholesterolemic or dysglycemic patients, this amount can be increased to 50 g/d or 10-25 g of soluble fiber per day. This should first be accomplished by increasing dietary intake of fresh fruits and vegetables (5 servings per day) and whole grains (6-11 servings per day).^{7,16} Table 2 highlights recommendations for preventing flatulence as dietary fiber intake increases.

Table 2. Prevention of Flatulence
With Increasing Dietary Fiber

It can take a few months for the GI tract to adapt to high-fiber foods or a vegetarian eating style. The following tips may help to reduce intestinal gas during the transition to a diet high in fruit, vegetables, bran, whole grains, and beans.

- Increase the intake of raw and high-fiber foods gradually (5- to 10-g increments).
- Keep quantities small until GI tolerance improves.
- Drink sufficient fluid—at least 8-12 glasses a day—because high fiber intakes increase fluid needs. Beverages with caffeine or alcohol don't count because they increase fluid loss.
- Foods that are cooked or canned may be better tolerated than raw or fresh foods. Fruits, vegetables, or beans that are mashed, blenderized, or pureed may also be easier to digest—mix or blend them into baked goods, sauces, or other foods.
- Rinse canned beans before using. Boil and then soak dry beans for 4 hours; drain the soaking water and replace it with fresh water for cooking.
- Maintain regular exercise.
- If needed, consult your doctor or pharmacist for special products that decrease the production of intestinal gas. Experiment with different types to find the brand that suits you best.

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If attempts to incorporate fruits and vegetables fail, soluble fiber supplementation may be necessary to obtain the recommended 11 servings of fruits and vegetables per day. If supplementation is warranted, 1 serving of psyllium powder twice daily before meals is the most efficient form of fiber therapy. One should mix the powder with at least 8 oz of fluid, drink quickly, and follow with another 8 oz of fluid. To avoid drug malabsorption issues, fiber supplements should be taken 1 hour before or 2 hours after taking prescription medications.¹⁶ Various fiber supplements are available on the market. The most widely researched are those containing psyllium powder. Other soluble fibers, such as flaxseed and fenugreek, may have similar effects as psyllium; however, the research is limited to make definitive recommendations on any fiber supplements other than psyllium. Again, this may be where newer terminology for the different fibers is warranted.¹⁰

Role of Dietary Fat in Vascular Health

Saturated Fat, Trans Fat, and Dietary Cholesterol

Intakes of dietary saturated fatty acids, trans fatty acids, and cholesterol have been shown to increase serum total cholesterol and LDL-cholesterol levels in a dose-dependent manner; therefore, recommendations specify reducing

dietary saturated fat, trans fats, and cholesterol, and limiting fat to 20%-35% of energy.¹ The TLC diet is low in saturated fat (<7% energy) and cholesterol. (<200 mg/d). Of all types of fatty acids, trans fatty acids have the strongest effect on raising ratios of serum total cholesterol to HDL,¹⁷ a known predictor of CHD risk.¹⁸ Trans fats account for 2.6% or 5.3 g/d of total energy intake in U.S. populations.¹⁹ The AHA's Diet and Lifestyle Recommendations¹⁴ include limiting trans fats to ≤ 1 g/d, a decrease from past consumption levels.

Role of ω -3 Fatty Acids in CVD Prevention

Two ω -3 fatty acids, docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA), found in cold-water fish such as mackerel, salmon, herring, trout, sardines, and tuna, have been shown to decrease the risk of sudden cardiac death in animal studies and in epidemiologic, metabolic, and small clinical trials.²⁰ These long-chain ω -3 fatty acids from fish oil have been shown to reduce serum triglyceride levels and favorably affect platelet function.²¹ Alpha-linolenic acid (ALA) is a shorter chain ω -3 fatty acid found in plant sources, including flaxseed, walnuts, canola oil, and soybeans. ALA may protect against CVD by interfering with the production of proinflammatory eicosanoids.¹⁶ ALA can be converted to EPA but only in small amounts (2%-5%) in humans; even less is converted to DHA (<1%).¹ One of the 3 dietary strategies to prevent CHD recommended by Giugliano et al² was to increase consumption of ω -3 fatty acids from fish or plant sources. The rationale for the strategy to increase ω -3 fats may be associated with reduced generation of a proinflammatory milieu or anti-inflammatory activity.²²

The Chicago Western Electric Study cohort of 1822 free-living men aged 40-55 years reported that men consuming >35 g fish per day had a significantly decreased relative risk of death from CHD.²³ In studies examining how blood levels of DHA and EPA affect cardiovascular health, the Cardiovascular Health Study, which examined free-living adults >65 years old, found that a higher concentration of combined plasma DHA and EPA was associated with a lower risk of fatal ischemic heart disease.²³

There is limited research evaluating the relationship between ALA and risk of CHD. According to Van Horn et al,¹ information is needed about the efficacy of marine and plant-derived ω -3 fatty acids in women and in high-risk populations. In addition, research to determine optimal dietary intake of ω -3 fatty acids (EPA, DHA, and ALA) and the ratio of ω -6 to ω -3 fatty acids is needed.¹

An FDA-approved qualified health claim, recommending up to 3 g/d of ω -3 fatty acids to reduce the risk of CHD, stated, "Supportive but not conclusive research shows that consumption of EPA and DHA ω -3 fatty acids may reduce the risk of CHD."²⁴ The AHA recommends ≥ 2 servings (~4 oz per serving) of oily fish per week and

inclusion of foods and oils rich in ALA, such as walnuts and soy or other vegetable oils.²⁵

Role of Plant Sterols and Stanols in CVD Prevention

Plant sterol and stanols, otherwise known as phytosterols, are the most researched functional food. Data collected for decades, starting in the 1950s, show that plant sterols and stanols have cholesterol-lowering effects.²⁶ These plant-based compounds have FDA-approved health claims supporting their role in lowering both total and LDL cholesterol and play a key role in the ATP III, AHA, and American Diabetes Association guidelines.^{3,14,15}

The average American diet provides about 200 mg of plant sterols/stanols daily, whereas vegetarian diets may provide upward of 500 mg/d.¹⁵ Plant sterols/stanols are found naturally in many plant foods, including nuts, vegetable oils, fruits, and vegetables.^{27,28} The 2 most abundant plant sterols are campesterol and sitosterol. Plant stanols are found in nature in smaller amounts, with sitostanol and campestanol being the major plant stanols.²⁹ Plant sterols have chemical structures that are similar to cholesterol. The mechanism of how plant sterols affect the absorption of cholesterol is not known; however, it is believed that both the plant sterols and stanols compete with the absorption of dietary and biliary cholesterol in the intestinal lumen and replace it in the mixed micelles, thereby reducing cholesterol absorption in the intestine. Studies have shown that a minimum amount of 800 to 1000 mg/d of etherified forms of plant stanols or sterols must be added to the diet to produce a significant reduction in cholesterol absorption and LDL cholesterol levels.^{15,30}

An extensive review was recently published that summarized the nearly 20 research studies determining the amounts of sterols or stanols required to reduce serum cholesterol levels.¹ The studies showed that between 0.8 and 3 g/d of plant sterols were needed to lower both serum total cholesterol and LDL cholesterol levels by 4% to 17%. Some studies also showed that plant sterol/stanol esters seem to have an additive effect to statin therapy, therefore allowing for greater reductions than the 2 therapies alone.³¹⁻⁴⁷

Miettinen et al³¹ examined 102 mildly hypercholesterolemic adults and showed reductions of serum total cholesterol levels of 10.2% and serum LDL cholesterol levels of 14.1% when the participants consumed 1.8-2.6 g/d of sitostanol-esters enriched margarine for 1 year. In later studies, reductions in serum levels of total cholesterol (7.2%) and in LDL cholesterol (12.4%) ($P < .001$) were found in a smaller group of 36 mildly hypercholesterolemic adults who consumed 2 g/d of sterols in a fortified orange juice over an 8-week period.⁴³

Polagruto et al⁴⁸ examined reductions in serum levels of total cholesterol and LDL cholesterol as well as reductions

in the ratio of total to HDL cholesterol. The research showed that consuming a phytosterol-enriched snack bar with 1.5 g of phytosterols over a 6-week period resulted in a 4.7% reduction in total cholesterol ($P < .01$), a 6% reduction in LDL cholesterol ($P < .01$), and a 7.4% reduction in the ratio of total cholesterol to HDL lipoprotein ($P < .001$).⁴⁸ Plana et al⁴⁹ focused only on LDL cholesterol reduction of a plant sterol-enriched fermented milk and, similar to previous investigators, found an average reduction of serum LDL cholesterol concentration of 12.2% after 3 weeks and 10.6% after 6 weeks when participants consumed 1.6 g of free sterols over 42 days.

Since 1999, plant sterol/stanol esters have been included in fortified foods in the United States. Significant research in the past 5 years has led to a strong growth in the manufacture of new plant sterol/stanolesterified foods. Along with the margarine spreads that were the initial vehicle for phytosterols, some orange juices, snack bars, and yogurt drinks now contain plant sterols and stanols (Table 3). Comparable to the spreads, these new products show evidence in clinical trials of producing similar hypocholesterolemic effects at the same amounts of approximately 2 g/d.³¹⁻⁴⁹ Over the last 2-5 years, cholesterol-lowering foods in the marketplace that contain either plant sterol or stanol esters have been developing at a fast rate. These phytonutrients are also available in dietary supplements. Whether in food or supplement form, these products are supposed to become effective after 2 weeks of consuming 2-3 servings per day with meals.⁴⁸ Regardless of the promising results of plant sterols and the new products introduced, research is needed to strengthen this evidence.

There are no significant contraindications to consuming plant sterols and stanols daily. On the other hand, there are concerns about specific populations, such as pregnant women, children, and elderly persons, consuming the therapeutic amounts of plant sterols; however, there is currently no research to support this concern. Research has shown that adding phytosterols to the diet may affect the absorption of some fat-soluble vitamins, specifically β -carotene.^{15,29,32} To counteract this potential vitamin malabsorption, adding an additional serving of a high-carotenoid fruit or vegetable daily is recommended.^{15,29,32}

In 2000, the FDA approved an interim final health claim which stated that foods containing either plant stanol or sterol esters may reduce the risk of CHD by lowering serum total and LDL cholesterol levels. The FDA recommends a minimum of 800 mg/d of plant sterols/stanols to help reduce the risk of CHD by lowering total and LDL cholesterol.⁵⁰ In accordance with the current evidence, the ATP III, the AHA, and the American Diabetes Association recommend 2 g/d of plant sterols/stanols as adjuncts to LDL cholesterol-lowering strategies for CVD risk reduction.^{3,14,15}

Table 3. Plant Sterol/Stanol Product List

Phytosterol-containing Food	Serving Size	Sterols or Sterol Esters Per Serving	Amount Needed to Meet Daily Recommendations
Smart Balance® omega plus butter spread (Smart Balance, Paramus, NJ)	1 T	450 mg plant sterols	3-4 T
Benecol® regular spread (McNeil Nutritionals, Fort Washington, PA)	1 T	0.85 g sterol esters	4 T
Benecol® light spread (McNeil Nutritionals, Fort Washington, PA)	1 T	0.85 g sterol esters	4 T
Promise Activ® light spread (UnileverUSA, Englewood Cliffs, NJ)	1 T	1.7 g sterol esters	2 T
Promise Activ® Supershots (UnileverUSA, Englewood Cliffs, NJ)	1 bottle	2 g plant sterols	1 bottle
Minute Maid® Premium Heart Wise™ orange juice (The Coca-Cola Company, Atlanta, GA)	8 oz	1 g sterol esters	2 cups
Hain's Rice Dream® Heartwise rice drink (Hain Celestial Group, Melville, NY)	8 oz	0.65 g sterol esters	2 cups
Nature Valley® Healthy Heart chewy granola bar (General Mills, Minneapolis, MN)	1 bar	0.4 g sterol esters	3-4 bars
CocoaVia™ chocolate bar (Mars, Hackettstown, NJ)	1 bar (20-22 g)	1.1 g sterol esters	2 bars
CocoaVia™ snack bar (Mars, Hackettstown, NJ)	1 bar (22-23 g)	1.5 g sterol esters	2 bars
CocoaVia™ chocolate-covered almonds (Mars, Hackettstown, NJ)	1 pack (28 g)	1.1 g sterol esters	2 packs
Kroger™ Active Lifestyle milk (Kroger Company, Cincinnati, OH)	8 oz	0.4 g sterol esters	2 cups
Lifetime™ low-fat cheese slice (Lifeline Food Company, Seaside, CA)	1 slice (19 g)	0.65g sterol esters	2 slices
Orowheat® whole grain and oat (Bimbo Bakeries USA, Dallas, TX)	1½ slices	0.4 g sterol esters	3 slices
Sturm Foods® instant oatmeal (Sturm Foods, Manawa, WI)	1 packet	650 mg sterol esters	2 packets
Centrum Cardio® (Wyeth Consumer Healthcare, Madison, NJ)	2 tablets	800 mg sterol esters	2 tablets
GNC Preventive Nutrition® Heart Advance™ (General Nutrition Centers, Pittsburgh, PA)	1 pill	400 mg free phytosterols	2 pills
Vegapure® Heart Choice™ (Cognis Group, LaGrange, IL)	1 pill	650 mg sterol esters	2 pills
Nature Made® Cholest-Off (Pharmavite, LLC, Mission Hills, CA)	2 pills	900 mg plant sterols	4 pills
TwinLab® Cholesterol Success™ (TwinLab Corporation, American Fork, UT)	1 pill	850 mg sterol esters	3 pills

Role of Soy Protein in Prevention of CVD

The role of soy in the prevention of CVD, particularly LDL cholesterol-lowering effects, has been the subject of numerous controlled clinical studies. However, confounding variables, such as the amount and forms of soy used (eg, soy protein, soy protein isolate, soy flour, and soy oil), and, more specifically, the isoflavones comprising soy protein, genestein and daidzein, make these studies difficult to interpret.¹ Soy protein (21 g/1000 kcal) is incorporated into the portfolio diet. In 2006, a study reported findings from a 1-year trial in

which 66 individuals who adhered well to the portfolio diet (31.8% of participants) experienced reduced serum LDL cholesterol levels by 29.7%.⁵¹ However, which dietary factors contributed to the cholesterol-lowering effects were unknown. The current data for the cardiovascular benefits of soy are not conclusive. In fact, the U.S. Food and Drug Administration's approved health claim from 1999, which stated that 25 g/d of soy protein was associated with reduced risk of CHD, is no longer supported by the current research. In 2006, Sacks et al⁵² concluded in a critical summary by the AHA that isolated soy protein offered no particular advantages in lowering

lipid levels vs milk or other proteins. Modest reductions in serum LDL cholesterol levels have been achieved with soy intake, especially for subjects with hypercholesterolemia.¹

What does this mean for clinicians? Although additional research is needed, it is important to remember that soybeans are a nutritionally viable plant-based protein that may be used to reduce the saturated fat in the diet from animal protein.

Alcohol

Cardioprotective Effects of Alcohol

The cardiovascular effects of alcohol are well documented. The inverse association between moderate alcohol consumption and CHD was well established in a review of the scientific literature.⁵³ The research points to as little as 1-2 weeks of moderate alcohol consumption having a positive effect on HDL cholesterol levels. Moderate consumption, according to the Dietary Guidelines for Americans 2005, is no more than 1 drink per day for women and no more than 2 drinks per day for men—ideally taken with meals. A standard serving of alcohol is 12 oz of beer, 5 oz of wine, and 1.5 oz of 80-proof distilled spirits; each contains approximately 0.6 oz of alcohol.⁵⁴ The adverse effects of consuming large amounts of alcohol include alcoholism, liver disease, cancer, and incapacitating and fatal accidents¹; thus, moderation is recommended for individuals who choose to consume alcohol.

Although the common denominator found in the research is that moderate alcohol consumption appears to increase serum HDL cholesterol levels, it has also been found to prolong blood clot formation by lowering levels of fibrinogen and protein tissue-type plasminogen activator.⁵⁵ In addition, a negative side effect of moderate alcohol consumption (30 g/d or 2 drinks per day) is a subsequent increase in serum triglyceride levels.⁵⁶

Dietary Recommendations of Alcohol Use

Although the clinically recommended cardioprotective dietary pattern includes some alcohol, it is by no means a license for individuals to start consuming alcohol if it is not part of their current lifestyle. There are some populations for whom alcohol is not recommended, such as pregnant women, those at risk for alcoholism, or anyone engaging in activities that require attention, skill, or coordination, such as driving a vehicle or operating machinery. In addition, people with cardiomyopathy, hypertension, cardiac arrhythmias, and high serum triglyceride levels should avoid alcohol because it can contribute to the onset of pancreatitis.¹ Because alcohol use can contribute to weight gain by increasing energy intake, overweight or diabetic patients should limit or avoid alcohol consumption.⁵⁷

Functional Foods and Weight Status in CVD Risk

Over the last decade, an “inclusive food strategy” has been the cornerstone of the AHA’s dietary recommendations for combating CVD and related diseases. However, there are challenges posed by integrating a growing list of heart-healthy foods into the diet without increasing energy intake beyond that required to achieve a healthy body weight.⁵⁸ In relation to food, the AHA’s 4 goals are to achieve a healthy overall diet, achieve a healthy body weight, promote desirable blood lipid levels, and achieve desirable blood pressure levels. To meet these goals, additional foods, such as nuts, have been added to the list of heart-healthy foods.

Nuts and CVD Risk

Consumption of $\frac{3}{4}$ to 1 oz of unsalted nuts daily (almonds or walnuts are the nuts researched) is suggested to confer cardiovascular benefits.¹ In more than 86,000 women in the Nurses Health Study, the consumption of 5 oz of nuts per week resulted in significantly lower CHD risk than those who rarely ate nuts.⁵⁹ Evidence has isolated the fatty acids in the nuts as the primary cardioprotective ingredients. In a randomized, crossover trial of 28 men and women, the mean (SD) levels of total and LDL cholesterol were 6.0 (1.1) mmol/L and 4.1 (1.0) mmol/L, respectively. The patients had a mean body mass index (BMI) of 26.9 (3.2) kg/m². Participants were asked to consume a low-saturated-fat diet, which included 30 g/d of nuts (nut diet) or 1 serving of a cereal containing canola oil (cereal diet) for 2 periods of 6 weeks, separated by a 4-week washout.⁶⁰ The data revealed that a 30-g/d serving of nuts had similar lipid-lowering effects as a serving of the canola-based cereal, and the investigators concluded that foods with a similar fatty acid profile as nuts can produce comparable decreases in lipoprotein-mediated cardiovascular risk.⁶⁰ As always, the concern about nuts is the high caloric value. A serving of almonds or walnuts is about 140 kcal. These discretionary calories can add up quickly and cause unnecessary weight gain, which can lead to obesity, a risk factor for CVD.

Obesity as a Risk Factor for CVD

Obesity (BMI >30) is an independent risk factor for CHD based on evidence from a recent review of large population studies, such as the Framingham Heart Study, the Nurses Health Study, the Buffalo Health Study, and the Cancer Prevention Study II.⁶¹ Obesity research has shown that excess body weight influences CHD risk factors, such as serum lipid profile (serum LDL cholesterol, triglyceride, HDL cholesterol levels), hypertension, and insulin resistance.

BMI and Waist Circumference's Correlation to CVD Risk

In the Health Professionals Follow-Up Study, males with a BMI >30 had 4 times greater likelihood of CVD death than those with a BMI <23.⁶² In other large population studies, such as the National Health and Nutrition Examination Survey II, increases in BMI levels have been associated with increased levels of serum total cholesterol, non-HDL cholesterol, LDL cholesterol, and triglycerides⁶³—all CVD risk factors. Age and BMI appear to play a role, as BMI is consistently associated with increased risk for CHD and mortality among those younger than 65 years.⁶³ However, in people >65 years, BMI is not significantly related to risk of CHD or CVD events.^{64,65}

Research has also shown that abdominal adiposity or waist circumference is related to overall or CVD mortality.⁶² The lower the waist circumference in men <65 years of age, the lower the mortality risk. In men, studies have shown that a waist circumference <38 inches poses less of a CVD risk than a circumference >38 inches.⁶⁶ Research also reveals that CVD risk in women increases proportionately with increases in waist circumference. In a study of 44,702 women, a higher risk (3.06) was reported with waist circumferences >38 inches compared with waist circumferences <30 inches.⁶⁷ Thus, the current recommendation leans toward using waist circumference or waist-to-hip ratio as a primary predictor of CHD and risk for CVD death rather than BMI, especially in those ≤65 years of age.¹

Conclusion

The vast body of scientific evidence shows that diet plays a primary role in the prevention of CVD. Various protocols for nutrition intervention support increased intake of fruits and vegetables (9-11 servings per day) and dietary fiber (25 g/d) as well as regular consumption of ω-3 fatty acids from cold-water fish at least 2 times per week, plant sterol/stanols (2 g/d), and nuts (1 oz/day). Substituting soy protein for animal protein to reduce saturated fat in the diet may be cardioprotective. In addition, moderate alcohol intake of 1-2 drinks per day with meals is recommended (for individuals who choose to consume alcoholic beverages). Overall lifestyle behaviors, such as energy balance, weight status, BMI, and waist circumference, are important risk factors that deserve consideration in the scope of cardiovascular health and wellness.

References

1. Van Horn L, McCoin M, Kris-Etherton PM, et al. The evidence for dietary prevention and treatment of cardiovascular disease. *J Am Diet Assoc.* 2008;108:287-331.
2. Giugliano D, Ceriello A, Esposito K. The effects of diet on inflammation: emphasis on the metabolic syndrome. *J Am Coll Cardiol.* 2006;48:677-685.
3. Executive summary of the third report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *JAMA.* 2001;285:2486-2497.
4. Libby P, Ridker PM, Maseri A. Inflammation and atherosclerosis. *Circulation.* 2002;105:1135-1143.
5. Hu FB, Willett WC. Optimal diets for prevention of coronary heart disease. *JAMA.* 2002;288:2569-2578.
6. Brown L, Rosner B, Willett WW, Sacks FM. Cholesterol-lowering effects of dietary fiber: a meta-analysis. *Am J Clin Nutr.* 1999;69:30-42.
7. Jenkins DJ, Kendall CW, Vuksan W, et al. Soluble fiber intake at a dose approved by the U.S. Food and Drug Administration for a claim of health benefits: serum lipid risk factors for cardiovascular disease assessed in a randomized controlled crossover trial. *Am J Clin Nutr.* 2002;75:834-839.
8. Strepel MT, Arends LR, van't Veer P, Grobbee DE, Geleijnse JM. Dietary fiber and blood pressure: a meta-analysis of randomized placebo-controlled trials. *Arch Intern Med.* 2005;165:150-156.
9. Saltzman E, Das SK, Lichtenstein AH, et al. An oat-containing hypocaloric diet reduces systolic blood pressure and improves lipid profile beyond effects of weight loss in men and women. *J Nutr.* 2001;131:1465-1470.
10. Queenan KM, Stewart ML, Smith KN, Thomas W, Fulcher RG, Slavin JL. Concentrated oat beta-glucan, a fermentable fiber, lowers serum cholesterol in hypercholesterolemic adults in a randomized controlled trial. *Nutr J.* 2007;6:6.
11. Andon MB, Anderson JW. State of the art reviews: the oatmeal-cholesterol connection: 10 years later. *American Journal of Lifestyle Medicine.* 2008;2:51-57.
12. Theuvsen E, Mensink RP. Water-soluble dietary fibers and cardiovascular disease. *Physiol Behav.* 2008;94:285-292.
13. *Dietary Reference Intakes: Energy, Carbohydrate, Fiber, Fat, Fatty Acids, Cholesterol, Protein, and Amino Acids.* Washington, DC: National Academy of Science and the Institutes of Medicine; 2002.
14. Lichtenstein AH, Appel LJ, Brands M, et al. Diet and lifestyle recommendations revision 2006: a scientific statement from the American Heart Association Nutrition Committee. *Circulation.* 2006;114:82-96.
15. Franz MJ, Bantle JP, Beebe CA, et al; American Diabetes Association. Evidence-based nutrition principles and recommendations for the treatment and prevention of diabetes and related complications. *Diabetes Care.* 2003;26(suppl 1):S51-S61.
16. Szapary PO, Conway ME. Functional foods in the prevention of cardiovascular disease. In: Carson J, Burke FM, Hark L, eds. *Cardiovascular Nutrition: Disease Management and Prevention.* Chicago, IL: American Dietetic Association; 2004:219-226.
17. Mensink RP, Zock PL, Kester AD, Katan MB. Effects of dietary fatty acids and carbohydrates on the ratio of serum total to HDL cholesterol and on serum lipids and apolipoproteins: a meta-analysis of 60 controlled trials. *Am J Clin Nutr.* 2003;77:1146-1155.
18. Shai I, Rimm EB, Hankinson SE, et al. Multivariate assessment of lipid parameters as predictors of coronary heart disease among postmenopausal women: potential implications for clinical guidelines. *Circulation.* 2004;110:2824-2830.
19. Allison DB, Egan SK, Barraj LM, Caughman C, Infante M, Heimbach JT. Estimated intakes of trans fatty and other fatty acids in the U.S. population. *J Am Diet Assoc.* 1999;99:166-174.
20. Kang JX, Leaf A. Prevention of fatal cardiac arrhythmias by polyunsaturated fatty acids. *Am J Clin Nutr.* 2000;71(1 suppl):202S-207S.
21. Kris-Etherton P, Daniels SR, Eckel RH, et al. Summary of the scientific conference on dietary fatty acids and cardiovascular health:

- conference summary from the nutrition committee of the American Heart Association. *Circulation*. 2001;103:1034-1039.
22. Connor WE. Importance of n-3 fatty acids in health and disease. *Am J Clin Nutr*. 2000;71(1 suppl):171S-175S.
 23. Daviglus ML, Stamler J, Orenca AJ, et al. Fish consumption and the 30-year risk of fatal myocardial infarction. *N Engl J Med*. 1997;336:1046-1053.
 24. U.S. Food and Drug Administration. FDA Announces Qualified Health Claims for Omega-3 Fatty Acids 2004. <http://www.fda.gov/bbs/topics/news/2004/new01115.html>
 25. Davis BC, Kris-Etherton PM. Achieving optimal essential fatty acid status in vegetarians: current knowledge and practical implications. *Am J Clin Nutr*. 2003;78(3 suppl):640S-646S.
 26. Cater N. Historical and scientific basis for the development of plant stanol ester foods as cholesterol-lowering agents. *Eur Heart J*. 1999;1:S36-S44.
 27. Cater NB. Plant stanol ester: review of cholesterol-lowering efficacy and implications for coronary heart disease risk reduction. *Prev Cardiol*. 2000;3:121-130.
 28. Ntanos FY, Duchateau GS. A healthy diet rich in carotenoids is effective in maintaining normal blood carotenoid levels during the daily use of plant sterol-enriched spreads. *Int J Vitam Nutr Res*. 2002;72:32-39.
 29. Miettinen TA, Gylling H. Non-nutritive bioactive constituents of plants: phytosterols. *Int J Vitam Nutr Res*. 2003;73:127-134.
 30. Katan MB, Grundy SM, Jones P, et al. Efficacy and safety of plant stanols and sterols in the management of blood cholesterol levels. *Mayo Clin Proc*. 2003;78:965-978.
 31. Miettinen TA, Puska P, Gylling H, Vanhanen H, Vartiainen E. Reduction of serum cholesterol with sitostanol-ester margarine in a mildly hypercholesterolemic population. *N Engl J Med*. 1995;333:1308-1312.
 32. Blair SN, Capuzzi DM, Gottlieb SO, Nguyen T, Morgan JM, Cater NB. Incremental reduction of serum total cholesterol and low-density lipoprotein cholesterol with the addition of plant stanol ester-containing spread to statin therapy. *Am J Cardiol*. 2000;86:46-52.
 33. Hallikainen MA, Sarkkinen ES, Uusitupa MI. Plant stanol esters affect serum cholesterol concentrations of hypercholesterolemic men and women in a dose-dependent manner. *J Nutr*. 2000;130:767-776.
 34. Nestle M. Genetically engineered "golden" rice unlikely to overcome vitamin A deficiency. *J Am Diet Assoc*. 2001;101:289-290.
 35. Maki KC, Davidson MH, Umporowicz DM, et al. Lipid responses to plant-sterol-enriched reduced-fat spreads incorporated into a National Cholesterol Education Program Step I diet. *Am J Clin Nutr*. 2001;74:33-43.
 36. Tikkanen MJ, Hogstrom P, Tuomilehto J, Keinanen-Kiukaanniemi S, Sundvall J, Karppanen H. Effect of a diet based on low-fat foods enriched with nonesterified plant sterols and mineral nutrients on serum cholesterol. *Am J Cardiol*. 2001;88:1157-1162.
 37. Davidson MH, Maki KC, Umporowicz DM, et al. Safety and tolerability of esterified phytosterols administered in reduced-fat spread and salad dressing to healthy adult men and women. *J Am Coll Nutr*. 2001;20:307-319.
 38. Ntanos FY, Homma Y, Ushiro S. A spread enriched with plant sterol-esters lowers blood cholesterol and lipoproteins without affecting vitamins A and E in normal and hypercholesterolemic Japanese men and women. *J Nutr*. 2002;132:3650-3655.
 39. Noakes M, Clifton P, Ntanos F, Shrapnel W, Record I, McInerney J. An increase in dietary carotenoids when consuming plant sterols or stanols is effective in maintaining plasma carotenoid concentrations. *Am J Clin Nutr*. 2002;75:79-86.
 40. Vanstone CA, Raeini-Sarjaz M, Parsons WE, Jones PJ. Unesterified plant sterols and stanols lower LDL-cholesterol concentrations equivalently in hypercholesterolemic persons. *Am J Clin Nutr*. 2002;76:1272-1278.
 41. Quilez J, Rafecas M, Brufau G, et al. Bakery products enriched with phytosterol esters, alpha-tocopherol and beta-carotene decrease plasma LDL-cholesterol and maintain plasma beta-carotene concentrations in normocholesterolemic men and women. *J Nutr*. 2003;133:3103-3109.
 42. Hendriks HF, Brink EJ, Meijer GW, Princen HM, Ntanos FY. Safety of long-term consumption of plant sterol esters-enriched spread. *Eur J Clin Nutr*. 2003;57:681-692.
 43. Devaraj S, Jialal I, Vega-Lopez S. Plant sterol-fortified orange juice effectively lowers cholesterol levels in mildly hypercholesterolemic healthy individuals. *Arterioscler Thromb Vasc Biol*. 2004;24:e25-e28.
 44. Jauhiainen T, Salo P, Niittynen L, Poussa T, Korpela R. Effects of low-fat hard cheese enriched with plant stanol esters on serum lipids and apolipoprotein B in mildly hypercholesterolaemic subjects. *Eur J Clin Nutr*. 2006;60:1253-1257.
 45. Gylling H, Rajaratnam RA, Vartiainen E, Puska P, Miettinen TA. Changes in serum level and metabolism of cholesterol with plant stanol esters in postmenopausal women with and without coronary artery disease. *Menopause*. 2006;13:286-293.
 46. Goldberg AC, Ostlund RE Jr, Bateman JH, et al. Effect of plant stanol tablets on low-density lipoprotein cholesterol lowering in patients on statin drugs. *Am J Cardiol*. 2006;97:376-379.
 47. Castro Cabezas M, DeVries JH, Van Oostrom AJ, Iestra J, van Staveren WA. Effects of a stanol-enriched diet on plasma cholesterol and triglycerides in patients treated with statins. *J Am Diet Assoc*. 2006;106:1564-1569.
 48. Polagruto JA, Wang-Polagruto JF, Braun MM, Lee L, Kiwk-Urbe C, Keen CL. Cocoa flavanol-enriched snack bars containing phytosterols effectively lower total and low-density lipoprotein cholesterol levels. *J Am Diet Assoc*. 2006;106:1804-1813.
 49. Plana N, Nicolle C, Ferre R, et al. Plant sterol-enriched fermented milk enhances the attainment of LDL-cholesterol goal in hypercholesterolemic subjects. *Eur J Nutr*. 2008;47:32-39.
 50. Food labeling: health claims; soluble dietary fiber from certain foods and coronary heart disease: final rule. *Fed Regist*. 2006;71:29248-29250.
 51. Jenkins DJ, Kendall CW, Faulkner DA, et al. Assessment of the longer-term effects of a dietary portfolio of cholesterol-lowering foods in hypercholesterolemia. *Am J Clin Nutr*. 2006;83:582-591.
 52. Sacks FM, Lichtenstein A, Van Horn L, et al. Soy protein, isoflavones, and cardiovascular health: an American Heart Association Science Advisory for professionals from the Nutrition Committee. *Circulation*. 2006;113:1034-1044.
 53. Rimm EB, Klatsky A, Grobbee D, Stampfer MJ. Review of moderate alcohol consumption and reduced risk of coronary heart disease: is the effect due to beer, wine, or spirits. *BMJ*. 1996;312:731-736.
 54. 2005 Dietary Guidelines Committee report. U.S. Department of Health & Human Services. <http://www.health.gov/DietaryGuidelines/dga2005/report/>.
 55. Mukamal K, Rimm EB. Alcohol's Effects on the Risk for Coronary Heart Disease. National Institute on Alcohol Abuse and Alcoholism. <http://pubs.niaaa.nih.gov/publications/arh25-4/255-261.htm>.
 56. Mukamal KJ, Rimm EB. Alcohol's effects on the risk for coronary heart disease. *Alcohol Res Health*. 2001;25:255-261.
 57. Franz MJ, Monk A, Barry B, et al. Effectiveness of medical nutrition therapy provided by dietitians in the management of non-insulin-dependent diabetes mellitus: a randomized, controlled clinical trial. *J Am Diet Assoc*. 1995;95:1009-1017.
 58. Kris-Etherton PM, Etherton TD, Carlson J, Gardner C. Recent discoveries in inclusive food-based approaches and dietary patterns for reduction in risk for cardiovascular disease. *Curr Opin Lipidol*. 2002;13:397-407.
 59. Rimm EB, Willett WC, Hu FB, et al. Folate and vitamin B6 from diet and supplements in relation to risk of coronary heart disease among women. *JAMA*. 1998;279:359-364.

60. Chisholm A, McAuley K, Mann J, Williams S, Skeaff M. Cholesterol lowering effects of nuts compared with a Canola oil enriched cereal of similar fat composition. *Nutr Metab Cardiovasc Dis.* 2005;15:284-292.
61. Rashid MN, Fuentes F, Touchon RC, Wehner PS. Obesity and the risk for cardiovascular disease. *Prev Cardiol.* 2003;6:42-47.
62. Baik I, Ascherio A, Rimm EB, et al. Adiposity and mortality in men. *Am J Epidemiol.* 2000;152:264-271.
63. Denke MA, Sempos CT, Grundy SM. Excess body weight: an underrecognized contributor to high blood cholesterol levels in white American men. *Arch Intern Med.* 1993;153:1093-1103.
64. Folsom AR, Stevens J, Schreiner PJ, McGovern PG. Body mass index, waist/hip ratio, and coronary heart disease incidence in African Americans and whites. Atherosclerosis Risk in Communities Study Investigators. *Am J Epidemiol.* 1998;148:1187-1194.
65. Ellekjaer H, Holmen J, Vatten L. Blood pressure, smoking and body mass in relation to mortality from stroke and coronary heart disease in the elderly: a 10-year follow-up in Norway. *Blood Press.* 2001;10:156-163.
66. Rexrode KM, Buring JE, Manson JE. Abdominal and total adiposity and risk of coronary heart disease in men. *Int J Obes Relat Metab Disord.* 2001;25:1047-1056.
67. Rexrode KM, Carey VJ, Hennekens CH, et al. Abdominal adiposity and coronary heart disease in women. *JAMA.* 1998;280:1843-1848.