

Prudent Diet and Preventive Nutrition From Pediatrics to Geriatrics: Current Knowledge and Practical Recommendations

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“A man is what he eats” (German proverb). Food provides not only the essential nutrients for life but also other bioactive compounds for the promotion of health and the prevention of disease.^{1–3} The results of 50 years of intensive worldwide research support the conclusion that diet is the major environmental cause of atherosclerosis and cardiovascular diseases (CVD), especially in genetically susceptible individuals.⁴ A high-caloric diet, combined with limited physical activity, contributes to dyslipidemia, insulin resistance, diabetes, and obesity. All these abnormalities increase the risk of CVD. Over the past few decades, the prevalence of obesity has doubled in adults, and quadrupled in teenagers in the USA. A similar pattern is emerging in India, where an epidemic of coronary artery disease (CAD) and diabetes is under way, with no signs of a downturn. Whereas the rates of CAD have declined by 60% in the US, the rates have increased by 300% in India over the past 30 years.⁵ The public and physicians are constantly bombarded with confusing and conflicting dietary advice. This review analyzes the important recent developments in the fields of diet and nutrition for the prevention and treatment of CVD and diabetes, with particular attention to Asian Indians.

Facts and Myths about Cholesterol, Fats, and Meats

The modern understanding of the role of nutrition in heart disease began in 1903 when Anitschkow and Chalатов found that a diet of meat, milk, and egg produced atherosclerosis in rabbits. A decade later, serum total cholesterol (TC) level was found to be the agent responsible. Contrary to common belief, the contribution of dietary cholesterol to serum TC is small (<10 mg/dl). The average adult on a western diet consumes about 300 mg of cholesterol daily, which is about the size of 3 toothpicks, and hardly 3 cal. Nonetheless, high intakes of dietary cholesterol increase the number of circulating low-density

lipoprotein (LDL) particles.⁶ Dietary cholesterol is found only in the animal kingdom; 3 oz of beef, lamb, or pork contains 75 mg of cholesterol. Most of the cholesterol in poultry is in the skin, and some in dark meat. One cup of milk has 33 mg, 2 egg yolks have 560 mg, and 100 g of brain has 2000 mg of cholesterol. One hundred grams of shrimp contain about 150 mg of cholesterol but <1 g of saturated fat. The recommended dietary intake of cholesterol and various types of fat is given in Table 1.^{1,6–12} The contribution of dietary saturated fat to serum TC is very large—10 times greater than that of dietary cholesterol. Fats are substances consisting of a combination of fatty acids, which are classified as saturated (SAFA), monounsaturated (MUFA), polyunsaturated (PUFA), and transunsaturated (TRAFA), depending on the location and number of double bonds.¹³ It is not often appreciated that the quality of the fat is more important than the quantity of fat consumed. The National Cholesterol Education Program (NCEP) recommends an intake of total fat of 25%–35%, MUFA up to 20%, PUFA up to 10%, and SAFA <7% of the total energy¹⁴ (Table 1). Although many affluent Asian Indians consume 50% of energy from fat, the average consumption is about half this amount (20%–25% of the energy). Increasing the MUFA intake to 20%, and total fat intake to 35% of the energy appears to be appropriate for Asian Indians because of the beneficial effects on high-density lipoprotein (HDL) and triglycerides (TG). The NCEP dietary guidelines for PUFA and SAFA seem appropriate for Asian Indians without any modification.

Saturated fatty acids, the arch villain of atherosclerosis: Excessive consumption of SAFA is the principal dietary culprit contributing to elevated serum TC level, which is the primary determinant of atherosclerosis.^{15,16} Differences in CAD mortality worldwide are explained by differences in SAFA intake and the resulting serum TC levels in 40 countries, except for France, Finland, and India.^{16–18} Intake of SAFA suppresses the LDL-receptor activity and decreases the clearance of LDL from the circulation, resulting in a marked elevation of its level.¹⁹

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Table 1. Recommended daily energy intake and major sources of dietary fats^{1,6-12}

	Recommended intake	Current intake (USA)	Major sources
Total fat	30%–35% (40–75 g)	34%	Dairy products and animal flesh are the major sources of dietary fat; the former contribute more than the latter.
Cholesterol	<200 mg	270 mg	Egg yolk, brain, organ meat, beef, lamb, pork, poultry (thigh and skin), shell fish, shrimp, prawn, full-fat milk (especially buffalo milk), high-fat dairy products (cream, ice cream, milk shake, cheese, curd)
SAFA	<7% (10–15 g)	12%	Beef, lamb, pork, bacon, sausage, ribs, poultry with skin, butter, ghee, <i>vanaspathi</i> (vegetable <i>ghee</i>), desserts, bakery products (cakes, biscuits, cookies, donut), cheese, ice cream, full-fat milk, tropical oils (coconut, palm kernel, and palm oils)
TRAFA	Avoid	<2%	Hard margarine, vegetable shortening, frying fats (especially those used repeatedly), bakery products (cakes, Danish pastry, donuts, crackers, rusk, biscuits, cookies, white bread), French fries, fried chicken, peanut butter, nondairy creamer, tortillas, pizza, and virtually all “crispy and crunchy foods”
MUFA	Up to 20%	14%	Olive oil, canola oil, mustard oil, peanut oil, macadamia nuts, hazelnuts, pecans, peanuts, almonds, cashew nuts, pistachio nuts, avocado, dairy products, beef, lamb, mutton, poultry
n-6 PUFA	7%–8 %	6%–8%	Vegetable oils (soybean, corn, safflower, sunflower, and cottonseed)
n-3 PUFA	2%–3%	<1%	Fatty fish (mackerel, halibut, lake trout, herring, sardines, albacore tuna, salmon), meat, poultry, vegetables (tofu, soybeans, pinto beans, flax seeds), vegetable oils (soybean, canola), salad dressing, whole grains, and DHA-enriched egg, nuts (walnuts, butternut, flax seeds, pecans)

SAFA raises the serum TC level thrice as much as PUFA, and MUFA lowers it. For example, substitution of 20% of the daily energy intake of carbohydrate by SAFA increases the TC level by 30 mg/dl, whereas PUFA and MUFA lower it by 10 mg/dl.¹³ Most of this increase is due to an increase in LDL. Although some increase in HDL also occurs, it is not sufficient to offset the atherogenicity and thrombogenicity resulting from marked elevation of LDL.^{6,20}

Our diet contains SAFA of different chain lengths with varying atherogenic properties. According to their chain lengths, SAFA can be classified as short chain (4:0–6:0), medium chain (8:0–10:0), long chain (12:0–18:0), and very long chain (20:0–24:0) fatty acids. Stearic acid (C18:0) is desaturated to oleic acid soon after its absorption, and hence does not raise the TC level.^{21,22} Therefore, its use need not be restricted and, in fact, it can be recommended.²³ SAFA with chain lengths of 12–16 have the most cholesterol-raising properties.²⁴ These are lauric acid (C12:0), myristic acid (C14:0), and palmitic acid (C16:0). These 3 fatty acids account for only 25%–30% of the total dietary fat but 60%–70% of SAFAs in western diets.²⁴

Palmitic acid is the most common fatty acid in the human diet, and the principal SAFA in both animal fats and palm oil. In a study conducted in a metabolic ward, 40% of energy as palmitic acid raised the TC by 25 mg/dl v. 15 mg/dl with lauric acid.²¹ Myristic acid is the most powerful cholesterol-raising SAFA, and increases the TC level 50% more than palmitic acid. Replacement of 20% of energy from carbohydrate with myristic acid raises the blood TC level by 46 mg/dl, compared to 30 mg/dl with palmitic acid, and 20 mg/dl with lauric acid.²⁵ Most of the rise in the TC level is due to an increase in LDL, the respective contribution from HDL being 16 mg/dl, 8 mg/d and 12 mg/dl.²⁵ The major sources of myristic acid are butter and tropical oils (Table 2).^{6,20-25} The TC-raising potential of lauric acid is 33% less than that of palmitic acid, and it is the principal SAFA in coconut and palm kernel oils, both containing 48%.²³⁻²⁵ Coconut and palm oils are also high in myristic acid (18%), and this explains why the consumption of these oils raises the LDL level in a fashion similar to that of butter (Fig. 1).^{25,26} Studies in laboratory animals indicate that coconut oil increases both TG and LDL levels;^{6,27,28} the claim

Table 2. Chemical characteristics and atherogenicity of major fatty acids^{6, 20–25}

Fatty acids	Chemical structure	Atherogenicity	Comments
	SAFA		Highly atherogenic and thrombogenic. Markedly increases LDL level. Slightly increases HDL level.
Lauric acid	C12:0	↑	Coconut oil 48%, palm oil 48%, and butter fat 3%.
Myristic acid	C14:0	↑↑↑	Most potent cholesterol-raising SAFA. Coconut 18%, palm kernel oil 18%, butter fat 18%, animal fats 1%–5%
Palmitic acid	C16:0	↑↑	Most common and reference standard of SAFA. Palm oil 45%, butter fat 26%, beef fat 26%, mutton fat 24%, chicken fat 23%, pork fat 25%, cocoa butter 26%, coconut oil 9%, and palm kernel oil 8%.
Stearic acid	C18:0	⇔	Raises HDL level without raising LDL level. Butter fat 13%, beef fat 22%, mutton fat 25%, chicken fat 6%, pork fat 12%, cocoa butter 35%, coconut oil 3%, and palm oil 4%.
	TRAFA		Increases Lp(a), TG, small, dense LDL levels. Decreases HDL level; 3-fold increase in cardiac arrest.
Elaidic acid	C18:1 n-9 trans	↑↑↑	Fried food, crispy food, cakes, biscuits, donuts, pizza, reused frying oils.
	MUFA		Significantly lowers LDL level. Raises HDL level. Lowers insulin resistance. Antiatherogenic and antithrombogenic.
Oleic acid	C18:1 n-9	↓↓	Butter fat 28%, beef fat 39%, mutton fat 33%, chicken fat 42%, pork fat 45%, cocoa butter 35%, coconut oil 7%, palm kernel oil 14%, and palm oil 39%.
	n-6 PUFA		Lowers LDL levels. Lowers HDL level to a small extent Antiatherogenic.
Linoleic acid	C18:2 n-6	↓↓	Predominant PUFA in western diets.
	n-3 PUFA		Decreases LDL, and TG levels, blood pressure, and risk of sudden death. Increase HDL level, heart rate variability. Antiarrhythmic and antithrombogenic effects
Alpha-linolenic acid (ALNA)	C18:3 n-3	⇔	Precursor to EPA and DHA. Flaxseed oil 50%, canola oil 10%, mustard oil 10%
Eicosapentaenoic acid (EPA)	C20:5 n-3	⇔	Fatty fish (sardines, mackerel, salmon)
Docosahexaenoic acid (DHA)	C22:6 n-3	⇔	Fatty fish (sardines, mackerel, salmon)

that lauric acid does not raise TC is not supported by scientific data.^{21,23} Recent studies have shown that caprylic acid (C:8) and capric acid (C:10) raise the LDL level to about 50% that of palmitic acid, and raise the TG level.^{6,23–25} Coconut oil contains 14% of these two cholesterol-raising SAFA.

Replacing 5% of the daily energy intake of SAFA with MUFA and PUFA could reduce the risk of CAD by 42%.²⁹ Therefore, substituting MUFA and PUFA for SAFA and TRAFA is more effective in lowering the risk of CAD than

simply reducing the total amount of fat.³⁰ Since 1970, the total fat intake decreased from 42% to 34%, and SAFA from 18% to 12% in the USA, as a result of nationwide changes in dietary habits.^{2,31} This change in dietary fat intake is primarily responsible for the decrease in serum TC level from 220 to 200 mg/dl in the US population. This decrease in TC level is principally responsible for the dramatic reduction in CAD, during a period when the rates of obesity and diabetes doubled in Americans.³²

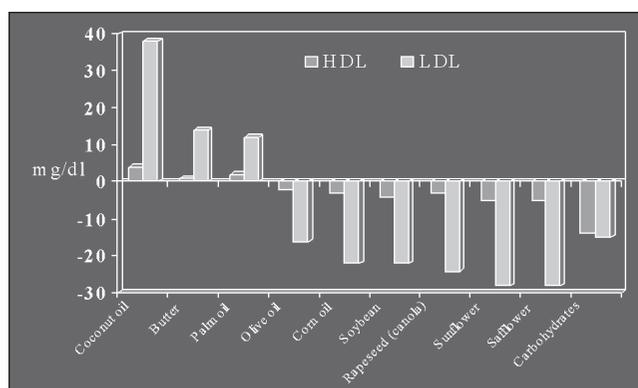


Fig. 1. Predicted change in HDL and LDL when all fat in Dutch diet is replaced by a particular oil.

Transfatty acids (TRAFA)—the hardened fat that hardens arteries fast:

TRAFA is formed during the partial hydrogenation of vegetable oils, a process that converts oils into solid or semisolid fats for subsequent use in food products. This process not only improves the texture and firmness but also markedly increases the shelf-life of food by minimizing oxidative spoilage.³³ Elaidic acid (n-9 trans 18:1) is the principal TRAFA, although several other trans isomers are also formed.²³ Such oils are used in commercial baked goods, and for cooking in most fast-food chains in western countries.³⁴ Perhaps an equally important and often neglected cause of TRAFA formation is the spontaneous hydrogenation of vegetable oils during deep-frying.³⁵ Very small amounts of TRAFA are also found in beef and dairy products (Table 1).

Consumption of TRAFA has a greater adverse effect on lipoproteins than that of SAFA.³⁶ Whereas both SAFA and TRAFA increase LDL levels considerably, TRAFA also decreases HDL levels, thereby increasing the TC/HDL ratio, the single best lipid-related risk factor for CAD.³⁷ Replacing 9% of calories from SAFA with TRAFA results in a 20% decrease in HDL level.³⁸ Other important adverse effects of TRAFA consumption include increases in lipoprotein(a) (Lp[a]), TG, and small, dense LDL levels,^{34,38–44} as well as increased platelet aggregation, endothelial dysfunction, and sudden death.³⁴ TRAFA are stronger predictors of CAD and diabetes than SAFA and carbohydrates.^{1,45–47} In the Nurses' Health Study, women in the highest versus the lowest quartile of TRAFA consumption had a 50% higher risk of CAD.⁴⁸ It is estimated that a substitution of 2% of calories from TRAFA with MUFA and PUFA results in a 53% reduction in CAD risk—a risk double that of substitution of calories from SAFA.²⁹ TRAFA consumption also markedly increases the postprandial insulin response in diabetic patients.⁴⁹ Replacing 2% of energy from TRAFA with PUFA

would lead to a 40% reduction in diabetes.⁴⁵ SAFA calories should not be replaced by TRAFA calories; doing so is like jumping from the frying pan to the deep-fat fryer.⁵⁰

The average consumption of TRAFA in the USA and Europe is low (<2% of energy or 11–27 g/day).^{51,52} However, TRAFA accounts for about 5% of fat in American diets, and 5% of fat stored in adipose tissue.^{33,50,53} Butter contains 60% SAFA, whereas stick margarine contains 16% TRAFA. The tub or soft margarine contains only 2 g of TRAFA per 15 ml. Therefore, the fat-spread of choice remains soft margarine;⁵⁴ olive oil may be an even better substitute. Although many margarines and shortenings previously contained up to 50% of TRAFA, in most western countries, these products currently have a low TRAFA content due to recent manufacturing changes.⁴⁷ However, frying fats used in fast-food outlets still contain over 30% of TRAFA. French fries sold in these outlets provide 7–8 g of TRAFA per portion. About one-third of TRAFA in the western diet comes from French fries, fried chicken, pizza, and cookies.

The TRAFA consumption is likely to be high in Asian Indians because deep-frying is a favorite mode of cooking at home as well as in restaurants. Deep-frying is associated with spontaneous hydrogenation and TRAFA formation, and repeated re-use of oils previously used for deep-frying may further increase the TRAFA content. These practices appear to be the norm rather than the exception, and may be of enormous public health importance, especially with regard to elevated Lp(a) levels, and high rates of CAD in this population. There is an urgent need to ascertain and disseminate the TRAFA content of *vanaspathi* (vegetable ghee) and frying oils used in India. As of today, we are not aware of any industrial manufacturing changes aimed at lowering the TRAFA content of Indian foods, as has been done in western countries.

MUFA, the good fat that raises the good cholesterol:

Diets high in MUFA (oleic acid C18:1) make LDL resistant to oxidation, restore LDL-receptor activity, and markedly lower LDL levels. Substitution of 20% of energy from carbohydrates with MUFA decreases TC by 10 mg/dl. The reduction in TC is 3-fold higher when MUFA replaces SAFA. For example, TC decreases by 40 mg/dl when 20% of energy from SAFA is replaced with MUFA.^{13,25} The effect on small, dense LDL is even greater. Other beneficial effects of MUFA include the favorable influence on blood pressure, endothelial activation, inflammation, and thrombogenesis.

A higher intake of MUFA lowers insulin resistance and diabetes, unlike SAFA and TRAFA, which increase it.^{45,49,55–60} Consumption of MUFA offers the unique

advantage of effectively lowering LDL levels without lowering HDL or raising TG levels. Individuals with low HDL levels have a high risk of CAD.^{61,62} Subjects with high TG, especially those with the metabolic syndrome and diabetes, are highly sensitive to the TG-raising effects of a high carbohydrate load. A high carbohydrate diet is associated with highly atherogenic, small, dense LDL particles, while high-fat diets are associated with less atherogenic, buoyant LDL particles. Thus, replacing SAFA with MUFA is more effective in preventing CAD than reducing the total fat intake, especially in Asian Indians, a population with high rates of prevalence of the metabolic syndrome and diabetes. The NCEP III has recommended up to 20% of total calories from MUFA (Table 1). This recommendation seems particularly appropriate for Asian Indians.³²

In Mediterranean countries, the high intake of MUFA in the form of olive oil is inversely related to CAD.¹⁵ The Nurses' Health Study and other studies of almost 300 000 Americans showed that a diet rich in MUFA in the form of canola oil also reduces the risk of CAD.^{29,63,64} Contrary to common belief, energy-controlled, high-MUFA diets do not promote weight gain, and are more acceptable than low-fat diets for weight loss in obese subjects. The addition of MUFA should be at the expense of SAFA and carbohydrates. Since all fats are high in calories (9 cal/g), failure to decrease the energy from carbohydrates and SAFA would invariably result in weight gain, and mitigate most of the beneficial effects of MUFA.

Meat and dairy products, which are also rich in SAFA, provide most of the MUFA in western diets. Olive oil and canola oil are good sources of MUFA (Table 3),⁶⁵ canola oil appears to be even better as it contains less SAFA and more PUFA, especially alpha-linolenic acid (ALNA). Mustard oil is high in MUFA but also high in erucic acid, which is known to have toxic effects on the heart. Canola oil is genetically engineered mustard oil without erucic acid. Nuts and avocado are excellent sources of MUFA and are recommended, provided the quantity is no more than 50–100 g/day.⁶⁶ Groundnut (peanut) products are a rich source of MUFA; they are inexpensive and widely available in India.⁶⁷

PUFA, another healthy substitute for SAFA: There are 2 series of PUFA that are deemed essential. Linoleic acid (C18:2 n-6) is the predominant omega-6 or n-6 PUFA. The predominant (parent) omega-3 or n-3 PUFA is linolenic acid (18:3 n-3).²³ Linoleic acid increases the fecal excretion of steroids, and inhibits the hepatic synthesis of apo B-containing lipoproteins. Replacing SAFA with PUFA reverses the suppression of LDL-receptor activity by cholesterol-raising SAFA (similar to that of MUFA).^{6,68}

Table 3. MUFA, PUFA, and SAFA content (%) in 100 g of various cooking oils⁶⁵

	MUFA	PUFA	SAFA
Sunflower oil, high oleic (>70%)	84	4	10
Safflower oil, high oleic (>70%)	75	14	6
Olive oil	74	8	14
Almond oil	70	17	8
Mustard oil	59	21	12
Canola oil	59	30	7
Cod liver oil	47	23	23
Peanut oil	46	32	17
Sunflower oil, linoleic (<60%)	45	40	10
Sesame oil	40	42	14
Rice bran oil	39	35	20
Palm oil	37	9	49
Cocoa butter	33	3	60
Corn oil	24	59	13
Soybean oil	23	58	14
Walnut oil	23	63	9
Sunflower oil, linoleic (>60%)	20	66	10
Cottonseed oil	18	52	26
Safflower oil, linoleic (>70%)	14	75	6
Palm kernel oil	11	2	82
Coconut oil	6	2	92

Substituting 20% of energy from SAFA with PUFA decreases the TC level by 40 mg/dl. Most of the reduction is in LDL, and the number of apo B particles.^{23,25} PUFA does not raise the TG level, and sometimes lowers it.⁶⁹ The two undesirable effects of PUFA are increased susceptibility for peroxidation, and lowering of the HDL level.^{10,70–72} HDL levels are reduced by about 1% for every 2% of MUFA or SAFA energy substituted with PUFA.^{6,70–72}

The substitution of PUFA for SAFA calories has played a major role in reducing TC levels and CAD in the USA. The CAD mortality rate declined by 60% in the past 3 decades in the USA.⁷³ About a third of the decline in CAD rates is attributed to a 6%–8% decrease in the serum TC level in the population; this, in turn, was due to an increase in the consumption of PUFA from 3% to 6%, and a decrease in SAFA consumption from 16% to 12% of the energy. The importance of PUFA is further underscored by the marked differences in PUFA consumption, which parallel the 4-fold difference in CAD rates between France and Finland.¹⁶ Vegetable oils, such as soybean, corn, safflower, sunflower, and cottonseed, are the primary sources of n-6 PUFA (Table 1). Their average consumption in the western diet is 6%–8% of energy, (17 g/day for men, and 12 g/day for women).⁷⁴

Contrary to previous fears, n-6 PUFA do not antagonize the anti-inflammatory effects of n-3 PUFA nor do they raise the risks of breast, colorectal, or prostate cancer in

humans.^{75,76} However, a very high n-6 PUFA to n-3 PUFA ratio may increase the thrombogenicity through increased production of arachidonic acid and thromboxane A₂. This is because linoleic and linolenic acids use the same set of enzymes for desaturation and chain elongation.⁷⁷ An n-6 PUFA to n-3 PUFA ratio of 3:1 appears to be optimum.^{78–80} Japan, which has one of the highest rates of fish consumption, has recently changed the recommendation of this ratio from 4:1 to 2:1;⁸¹ this ratio may be advisable for vegetarians).⁹

Fish, a tasty way to prevent sudden death: Fish do not die from myocardial infarction (MI), and populations that consume large amounts of marine foods have a low prevalence of CVD death.^{82–99} Replacing high-fat meat with fish is also associated with a decreased risk of CAD. The results of several large studies show that one or two fish meals per week are associated with a 30%–50% reduction in sudden death.^{84,95,98} In a meta-analysis of 11 prospective studies involving 116 764 individuals, fish consumption was inversely related to CAD death. This report suggests that 40–60 g/day of fish consumption is optimal, and results in a 40%–60% risk reduction.¹⁰⁰ Greater intake has no additional benefits, and suggests a threshold effect.^{101,102} However, a recent large study of 5103 women with diabetes showed a dose–response relationship. Consumption of fish 1–3 times per month was associated with a 40% risk reduction, and a 64% risk reduction was seen among those who consumed fish >5 times per week.^{102,103} The benefit is seen in people with and without prior heart disease.¹⁰⁴ These benefits persist as long as the fish consumption is continued.

Fish is a tasty food that contains many essential nutrients, such as selenium, iodine, vitamin D, and n-3 PUFA.^{97,105} The beneficial effects of fish are largely mediated through n-3 PUFA, which displace arachidonic acid from platelet phospholipid stores, thereby reducing the available substrate for thromboxane A₂ synthesis.^{106,107} The principal effects of n-3 PUFA are antithrombogenic and antiarrhythmic, whereas that of n-6 PUFA is antiatherogenic.^{108–117} The serum levels of n-3 PUFA are inversely related to sudden death.^{66,82,89,93,95,118–121} The consumption of n-3 PUFA decreases blood pressure, and homocysteine level, increases HDL level, and improves hemostatic factors (Table 4).^{10,75,122–132} A 30%–50% reduction in TG can be achieved by taking 3–5 g/day of n-3 PUFA.¹³⁰

The major n-3 PUFA in fish oils are eicosapentaenoic acid (EPA) (20:5 n-3) and docosahexaenoic acid (DHA) (22:6 n-3); together they constitute 26% of fish oil fatty acids.²³ The benefits from n-3 PUFA are greater with DHA

Table 4. Omega-3 fatty acids and CVD^{10,75,122–132}

- Decrease the risk of ventricular fibrillation and sudden death
 - Increase cell membrane PUFA
 - Favorably alter cardiac ion channel function and action potential
 - Decrease the ventricular fibrillation threshold
 - Increase heart rate variability
- Decrease the risk of stroke and MI
- Decrease platelet reactivity, aggregability, and the risk of thrombosis
- Reduce monocyte reactivity
- Reduce inflammatory cytokines and response
- Improve endothelial function
- Reduce the expression of vascular adhesion molecules
- Markedly lower the TG and remnant lipoprotein levels
- Decrease the growth of atherosclerotic plaques
- Decrease homocysteine levels
- Improve insulin sensitivity and reduce the risk of diabetes
- Slightly lower blood pressure

and EPA found in fatty fish, shellfish, and marine mammals than with ALNA found in canola oil, soybean oil, and walnut.¹⁰⁸ It is important to distinguish between lean and fatty fish for cardioprotection, because the content of n-3 PUFA is highest in fatty fish.¹¹⁹ Fatty fish, such as mackerel, sardine, and salmon, are widely available and inexpensive. Heating is associated with significant loss of n-3 PUFA.⁹ Frying fish is associated with an even greater loss of EPA and DHA, and may be particularly harmful if fried in SAFA.¹³³ The current intake of DHA and EPA is only 200 mg/day, and needs to be increased 5-fold to meet the dietary goals.¹¹

Both plant-based (ALNA), and fish-based (EPA and DHA) supplements have shown benefits in secondary prevention.^{89,90,134,135} In one such trial of 605 French men recovering from an MI, there was a 70% reduction in total and cardiac death during a follow-up of 27 months in those who received an experimental “Mediterranean diet” using canola oil-based margarine, enriched with n-3 PUFA.¹³⁶ In another large randomized study of 11 324 survivors of a recent MI, there was a 20% reduction in total deaths, 30% reduction in CVD deaths, and 45% reduction in sudden deaths among those who received n-3 PUFA 1g/day.⁹⁰ The totality of the data suggest that n-3 PUFA can be considered as the best antiarrhythmic agent and antifibrillatory treatment.^{106,117,133,135} Cardiologists and their patients should pay serious attention to this new paradigm in the diet–heart hypothesis, and increase the intake of fish and fish oil.^{133,137}

The amount of n-3 PUFA necessary for cardioprotection is surprisingly low. The current recommendation is to take 2–3 fish meals per week (200–300 g/week of fish). A less

attractive alternative is to consume 1000 mg/day of n-3 PUFA (contained in 3000 mg of fish oil capsules).^{10,11,74,96,133} The current intake of n-3 PUFA in the US is 1600 mg/day or 0.7% of the calories, which is about half the recommendation.¹¹ Fish is more beneficial than fish oil, but the latter may be required in most patients with CAD to obtain the required amount of n-3 PUFA.⁹⁰ Patients with CAD should consume about 1800 mg/day of n-3 PUFA (DHA and EPA) as the best insurance against sudden death.

Alpha-Linolenic acid (ALNA)—the n-3 PUFA of the plant kingdom: There is no DHA and EHA in a vegetarian diet. Vegetarians derive their n-3 PUFA almost exclusively from ALNA, which is also the major type of n-3 PUFA in omnivores.¹³⁸ There is increasing evidence for the cardioprotective effects of ALNA, albeit less than EPA and DHA.¹³⁹ In a large study involving 43 700 men, increased intake of ALNA reduced the risk of MI by 60%.⁶⁴ A similar risk reduction was also observed in the Nurses' Health Study and Multiple Risk Factor Intervention Trial (MRFIT).^{93,140} Some vegetable oils are high in ALNA (flaxseed oil 50%, canola oil 10%, mustard oil 10%, soybean oil 7%) while others are low (groundnut oil <0.5%).¹⁴¹ Walnuts are a rich source of ALNA; small concentrations are found in green leafy vegetables, corn oil, almonds, hazelnuts, cereals, pulses, millets, and spices.^{78,142} Walnuts and canola oil account for most of the ALNA in the western diets.^{78,101} The recommended intake of ALNA is 2% of energy but the current intake in the USA is 0.6% of energy.^{11,74}

ALNA is readily converted to EPA, and more slowly to DHA; the latter being the major component of phospholipid membranes of the brain and retina.⁷⁸ The beneficial effects of ALNA are less than half that of DHA and EPA, because the conversion of ALNA to the more active longer-chain metabolites is inefficient: <5%–10% for EPA, and 2%–5% for DHA.^{80,141,143} This explains why vegetarians have lower levels of n-3 PUFA than omnivores, and also higher platelet aggregability.⁷⁷ Since the biological effects of plant n-3 PUFA are significantly lower than marine n-3 PUFA, the requirements may be higher (3% of energy) for vegetarians than for nonvegetarians.^{9,74,77}

Protein: Americans eat 80–90 g/day of protein, which is twice the daily requirement, and most of this comes from meat, which is also high in SAFA. Up to 25% of daily energy from protein (but not more than 100 g/day) is permissible if the major source of protein is plant-based. Nuts are important sources of plant protein along with soy, bran, beans, and legumes. Substituting protein for carbohydrates increases HDL and lowers TG levels.^{144,145} In a meta-analysis

Table 5. Atherogenic, antiatherogenic, and neutral fatty acids in selected meats and cooking oils^{6,147}

Fat	Total SAFA (%)	Cholesterol-raising SAFA (%)	Stearic acid: cholesterol neutral SAFA (%)	Oleic acid: cholesterol-lowering MUFA (%)
Beef fat	51	29	22	39
Mutton fat	54	29	25	33
Chicken fat	30	24	6	42
Pork fat	39	27	12	45
Butter fat	66	53	13	28
Cocoa butter	61	26	35	35
Coconut oil	92	89	3	7
Palm kernel oil	74	71	3	14
Palm oil	50	46	4	39

of 38 controlled human clinical trials, consumption of soy protein (47 g/day) was associated with a significant 13% decrease in LDL, 10% decrease in TG, and a 2% increase in HDL levels.¹⁴⁶ This led to FDA approval for the use of food labels for the health claim that soy protein can reduce the risk of heart disease.

Meat: Although meat contains a significant amount of SAFA, almost half the SAFA is stearic acid, which does not raise TC levels. In addition, meat contains up to 45% of cholesterol-lowering MUFA. Furthermore, lean meat has much less SAFA than fatty cuts of meat (Table 5).^{6,147} Lean beef is an excellent source of protein and MUFA, and has less SAFA than chicken (dark meat); 6 oz of lean beef contains 3.0 g of SAFA v. a chicken thigh which contains 5.2 g of SAFA (the term loin or round signifies lean meat whereas prime or rib signifies fat cuts with very high SAFA in the USA). Chicken and lean beef (not fatty meat) have similar effects on plasma lipoproteins, and are interchangeable in a healthy diet.^{30,148,149}

Glycemic Load: A Potent Predictor of the Metabolic Syndrome and Diabetes

The source, nature, and amount of carbohydrates have a profound influence on postprandial glycemia, which in turn is directly associated with the risk of CAD in patients with diabetes.^{6,150,151} Foods containing the same amount of carbohydrate (carbohydrate exchange) may have up to a 5-fold difference in glycemic impact, depending on the differences in the digestion and absorption.^{152,153} The glycemic index is an extension of the fiber hypothesis, and was proposed in 1981 as a physiological system for the classification of carbohydrate-containing foods.^{154,155}

Carbohydrate classified by glycemic index, in contrast to its traditional classification as either simple or complex, is a better predictor of CAD in epidemiological studies.¹⁵⁶ The glycemic index is a scientific measure of the glycemic response to various foods, and is obtained from published food tables. The hierarchy of the glycemic index begins with beans, lentils, rice, spaghetti, potatoes, white bread (with refined flour), and refined grain cereals.¹⁵⁰ A high glycemic index indicates a lower quality of carbohydrate associated with low HDL levels, and low rates of satiety.^{157,158} Fruits, nonstarchy vegetables, parboiled rice, and legumes have a low glycemic index.¹⁵⁹ The glycemic index of potato is 102%, white bread 100%, whereas that of apple is 55%, and broccoli 13%. Glycemia observed after consuming dried peas is only one-third that of an equivalent amount of potatoes. Since peas are also high in fiber, their consumption needs to be encouraged, especially in patients with diabetes.^{153,160}

Glycemic load is the product of the glycemic value of the food and its carbohydrate content (per serving) divided by 100. For example, carrot has a high glycemic index but a low glycemic load (Table 6).^{152,161} The overall daily dietary glycemic load is calculated by adding the glycemic loads of all the different foods consumed in a given day. Accordingly, the glycemic load can be decreased by reducing the amount of carbohydrate intake and/or by consuming foods with a low glycemic index.¹⁶² In addition to the quality and quantity of carbohydrates consumed, the glycemic load also represents diet-induced insulin demand.^{163,164} PAI-1 levels are significantly increased with high glycemic load, and decreased with low glycemic load.¹⁶⁵

Dietary carbohydrates drive TG much more than dietary fat.⁶ A high glycemic load produces only mild increments in TG levels in individuals with normal TG levels but marked elevation in those with fasting lipemia and/or obesity.^{6,166–168} A low HDL level is a strong risk factor for CAD, even when the TC level is not elevated.^{169,170} A high glycemic load produces a low HDL, particularly when substituted for MUFA or PUFA.^{23,157,166,171–179} In a prospective study of 75 521 women followed up for 10 years, those in the highest quintile of glycemic load had double the risk of CAD after adjustment for age, smoking status, total energy intake, and other risk factors ($p < 0.0001$).¹⁵⁶

More importantly, a glycemic load promotes diabetes, especially in those with insulin resistance.^{156,161,180–183} (Fig. 2)¹⁸³ This is particularly true for refined carbohydrates, sweets, white bread, and potatoes.^{45,156,183,184} Thus, a high glycemic load may be considered a risk factor of equal importance as high SAFA diet in precipitating diabetes. A low glycemic load can reduce insulin secretion in patients

Table 6. Glycemic index of common foods^{152,161}

	Glycemic index	Serving size	Carbohydrate per serving	Glycemic load
Basmati rice	58	150 g	38	22
Brown rice (South India)	50	150 g	33	16
Parboiled rice (Canada)	48	150 g	36	26
White rice (Uncle Ben's)	45	150 g	36	16
Curry rice (Japan)	67	150 g	61	41
Jasmine rice	109	150 g	42	46
White bread	70	2 slices	30	21
Uppuma	18	150 g	33	6
Upittu	68	150 g	42	28
Chapati	76	60 g	30	23
Dosai	77	150 g	39	30
Idli	77	250 g	52	40
Poori	70	150 g	41	28
Pongal	68	250 g	52	35
Millet/ragi	104	70 g	50	52
Barley	43	150 g	37	16
Tapioca	70	250 g	18	12
Kellogg's cornflakes	81	30 g	26	24
Milk, full-fat	27	250 g	12	3
Yogurt	36	200 g	9	3
Orange juice (reconstituted USA)	57	250 ml	26	15
Pineapple	59	120 g	13	7
Plums	39	120 g	12	5
Prunes	29	60 g	33	10
Raisins	64	60 g	44	28
Cantaloupe	65	120 g	6	4
Plantain, green	38	120 g	21	8
Banana, unripe	70	120 g	45	31
Banana	53	170 g	25	13
Strawberry jam	51	30 g	20	10
Laddu	27	50 g	31	8
Black-eyed beans	42	150 g	30	13
Chickpeas	10	150 g	30	3
Kidney beans (rajmah)	13	150 g	25	3
Lentils	30	150 g	17	5
Lima beans	32	150 g	30	10
Pinto beans	39	150 g	26	10
Soy beans	15	150 g	6	1
Sweet corn	59	80 g	18	11
Green peas	39	80 g	7	3
Carrot	47	80 g	6	3
Beet root	64	80 g	7	5
Potato	85	150 g	30	26
Split peas	32	150 g	19	6
Snickers bar	55	60 g	35	19
Pizza Hut supreme pan pizza	36	100 g	24	9
Coca Cola	58	250 g	26	14
Instant noodles	47	180 g	40	19
Macaroni	45	180 g	49	22
Spaghetti	68	220 g	27	19

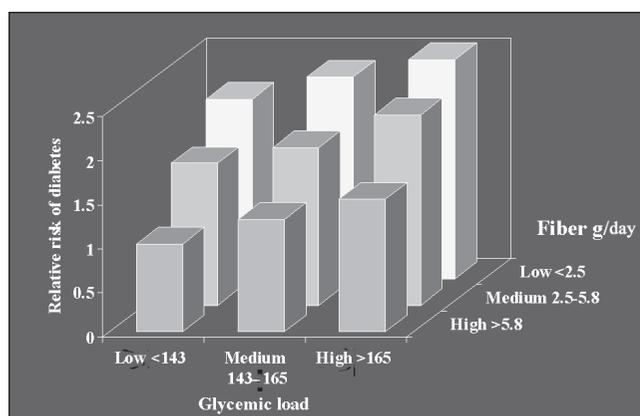


Fig. 2. Risk of diabetes in 65 173 US women during 6 years of follow-up: influence of glycemic load and fiber

with type 2 diabetes, decrease insulin requirements in type 1 diabetes, and improve glycemic control in both types of diabetes. The incremental benefit from low glycemic load is similar to that offered by pharmacological agents that also target postprandial hyperglycemia, such as alpha-glucosidase inhibitors.^{185,186} The benefit of low glycemic load on the development of diabetes is similar to MUFA, PUFA, whole grains, fiber, fruits, and vegetables.

Whole Grains: The Foundation of Healthy Food

Whole grains have been the staple food worldwide for centuries, especially among vegetarians.^{187,188} Whole grain and legume consumption not only decreases blood sugar and insulin resistance but also prevents the development of diabetes, particularly in people with the metabolic syndrome.^{185,186} Whole-grain products are a good source of fiber, minerals, as well as several vitamins, including vitamins B and E. In a 12-year follow-up of 42 898 men, the risk of developing diabetes was 42% lower in those with the highest intake of whole grains. The risk was reduced by 52% in those who also engaged in physical activity, and 87% in those who also had a low BMI.¹⁸⁹ The risk reduction was attributed to higher intakes of cereal fiber and magnesium. Intake of whole-grain cereal is inversely associated with hypertension, CAD, stroke, and CVD mortality^{190,191} (Table 7).^{192–206} In another study, 25%–30% reduction in stroke was observed with the intake of whole grains—similar in magnitude to that of statins.^{206–208} In sharp contrast, intake of refined grains increases the risk of diabetes, stroke and CVD.^{191,205–212} These prospective data highlight the importance of distinguishing whole-grain from refined-grain cereals in the prevention of CVD and diabetes.²⁰⁹ Efforts

Table 7. CVD risk reduction demonstrated with selected food groups^{192–206}

Author	CVD risk reduction (%)
Fruits and vegetables (15%–48%)	
Bazzano et al. ¹⁹²	25
Liu et al. ¹⁹³	15
Joshi-pura et al. ¹⁹⁴	30
Joshi-pura et al. ¹⁹⁵	20
Gaziano et al. ¹⁹⁶	48
Knekt et al. ¹⁹⁷	35
Nuts (19%–48%)	
Albert et al. ¹⁹⁸	48
Ellsworth et al. ¹⁹⁹	19
Hu et al. ²⁰⁰	35
Fraser et al. ²⁰¹	38
Fraser et al. ²⁰²	40
Jiang et al. ²⁰³	21 (diabetes)
Whole grains (32%–44%)	
Jacobs et al. ²⁰⁴	33
Fraser et al. ²⁰¹	44
Liu et al. ²⁰⁵	32
Liu et al. ²⁰⁶	32 (stroke)

should be made to replace refined-grain with whole-grain foods.¹⁸⁹

A whole-grain food includes all the edible parts of the grain: the bran, the germ, and the endosperm.²¹³ Grinding or milling, using modern technology, leads to the loss of many beneficial micronutrients, antioxidants, minerals, phytochemicals, fiber, and much of the germ.²¹⁴ As a result, refined grain products are devoid of most vitamins and essential fatty acids, and contain more starch.²¹⁵ Because of the loss of bran and pulverization of the endosperm, refined grains are digested and absorbed rapidly, resulting in a large increase in the levels of blood sugar and insulin.²¹⁵ The common grains consumed in the West include wheat, oats, rye, rice, barley, and corn.²¹³ In the USA, rye bread is an important source of whole grain consumption, and results in a lower glucose response than white bread.^{152,212} Whole-grain, ready-to-eat cereal contains >25% whole grain content by weight.¹⁸⁹ The recommended intake is at least 6 servings of grain (but not more than 11) with at least 3 being whole grains. The current intake of whole grains is less than half a serving/day or 15% of the grain intake. Only 2% of the 150 lb of wheat flour consumed per capita in the USA is whole-grain flour.²¹⁶ Commonly consumed refined grain foods include white rice (*idli, dosa*), refined wheat and flour (white bread), pancakes, cakes, sweet rolls, English muffins, muffins, waffles, rolls, biscuits, pizza, and refined-grain ready-to-eat breakfast cereal, and their use should be minimized.

Nuts: A Wholesome Food and Powerhouse of Healthy Fats and Nutrients

Extensive studies during the past decade have transformed the image of nuts from fattening snacks to a wholesome and heart-healthy food to be consumed daily.^{198–202,215} Nuts are rich sources of protein, antioxidants, fiber, vitamins and minerals (especially potassium and magnesium). Nuts yield 5%–10% fiber, and 12%–25% protein. The consumption of nuts is also associated with a reduced risk of CAD in several studies.^{198–202,217,218} Yet, nuts are not generally recommended as snacks because of their high fat content. Although nuts contain 45%–80% fat, most of the fats are the highly beneficial MUFA and PUFA (Table 8).⁶⁵

Nuts, particularly almonds, significantly improve lipid profiles because of the high fiber and MUFA component. The dose–response effects of almonds were compared with low-SAFA (<5% energy), whole-wheat muffins used as the control diet in a randomized crossover study involving 27 dyslipidemic men and women. Three isoenergetic supplements each (mean 423 kcal/day; 22% of energy) were consumed for 1 month. The supplement consisted of full-dose almonds (73 g/day), half-dose almonds plus half-dose muffins, and full-dose muffins. Full-dose almonds produced a highly significant decrease in the Lp(a) level (8%), LDL:HDL ratio (8%), and oxidized LDL (14%) compared to the control diet.²¹⁹ A 9% decrease in the LDL level occurred with 73 g/day of almonds, and 4% decrease with 37 g/day (handful) of almonds. This result translates to a 1% reduction in LDL for every 7 g/day of almonds, and is consistent with other studies.^{220,221} More importantly, there was no difference in body weight between the almond and muffin diet.²²² Nuts are energy-dense, and contain

160–200 cal/oz. It cannot be overemphasized that energy from nuts should replace the unhealthy calories from SAFA and refined grains to prevent weight gain.

Consumption of other nuts (except coconuts) is equally beneficial. For example, a 10% reduction in the LDL level can be achieved by the daily consumption of 40 g of walnuts, peanuts or pistachios, 70 g almonds, 100 g macadamia nuts, and 110 g of pecans.^{223–230} Nuts are as effective as increasing physical activity and trimming calories to increase HDL levels. Adding 2 oz or 60 g of nuts to a diet is a delicious way to decrease the TC/HDL ratio and CAD risk.^{8,14,23,231,232} Nuts also improve insulin sensitivity and prevent diabetes.²³³ In a prospective study of 83 818 women, 3206 new cases of type 2 diabetes were observed during a follow-up of 16 years.²⁰³ Consumption of nuts was inversely associated with the risk of type 2 diabetes after adjustment for age, body mass index (BMI), physical activity, smoking, alcohol use, and dietary factors (total calories, fat calories, and fiber). The risk of diabetes was reduced by 27% in those who consumed >5 oz/week of nuts or peanut butter compared to those who almost never ate these products.²⁰³ The proscription of nuts can no longer be justified. In fact, regular nut consumption as replacement for refined grains and high-fat meats is strongly recommended.^{161,234}

Fruits and Vegetables, the Natural Way to Consume Antioxidants and Flavonoids

Fruits and vegetables are rich in a myriad of nutrients and phytochemicals, including fiber, vitamins B and C, antioxidants, potassium, and flavonoids.^{190,215} Phytochemicals are bioactive nonnutrient plant

Table 8. MUFA, PUFA, and SAFA content (in g) in tree nuts and fruit (all nuts are dry roasted without salt except coconut)⁶⁵

	Calories per 100 g	Fat content per 100 g	MUFA	PUFA	SAFA
<i>Nuts</i>					
Macadamia nuts	718	76	59	1	12
Hazelnuts	646	62	47	8	5
Pecans	710	74	44	21	6
Almonds	597	53	34	13	4
Cashew nuts	574	46	27	8	9
Pistachio nuts	570	46	24	14	6
Walnuts	607	57	13	37	4
Flax seed	492	34	7	22	3
Coconut meat, creamed	684	69	3	1	61
Coconut meat, sweetened, shredded	501	35	2	0	31
<i>Fruits</i>					
Avocados, California	177	17	11	2	3
Olives	115	11	8	1	1
Avocados, Florida	112	9	5	1	2

compounds linked to a reduced risk of chronic diseases. Fruits and vegetables decrease blood pressure, homocysteine, and cancer, especially that of the GI tract.^{211,235,236} Since fruits and vegetables are rich in potassium, their liberal intake is recommended for the prevention and treatment of hypertension.²³⁷ Good sources of potassium include bananas, oranges, beans, fish, and dairy products. While you can get an overdose of potassium from pills, you cannot get an overdose of potassium from food. Moreover, dietary supplements do not have the health benefits associated with a diet rich in fruits and vegetables. For example, the antioxidant value of 100 g of apple is equivalent to 1500 mg of vitamin C.³

Several large studies, including one comprising 84 000 women and 42 000 men, have shown a significant inverse association between the consumption of fruits and vegetables and CVD mortality^{190,194,195} (Table 8). The relationship is particularly strong with vitamin C-rich fruits, green leafy vegetables, and carotenoid vegetables (carrots, broccoli, spinach, lettuce, tomatoes, and yellow squash).^{192–196,238,239} Consuming fruits and vegetables (3 times/day compared with <1 time/day) was associated with a 27% lower incidence of stroke, a 42% lower stroke mortality, a 24% lower CAD mortality, a 27% lower CVD mortality, and a 15% lower all-cause mortality after adjustment for standard CVD risk factors.¹⁹² In sharp contrast, consumption of potatoes and French fries increase the risk of CAD and stroke.^{152,215}

The landmark study of the Dietary Approaches to Stop Hypertension (DASH)²⁴⁰ has yielded tremendous insights into the benefits of increased intakes of various types of fruits and vegetables. The DASH diet is rich in vegetables, fruits, and low-fat dairy products (9 servings of fruits and vegetable combined per day).²⁴⁰ As compared with the control diet with a high sodium, the DASH diet with a low sodium intake led to a decrease in systolic blood pressure of 7 mmHg in normotensive individuals, and 11.5 mmHg in hypertensive individuals. The benefits of the DASH diet on lipoprotein levels were equally spectacular, with an 11 mg/dl decrease in LDL and a 4 mg/dl increase in HDL levels without significant effects on TG levels. Men had a greater reduction in LDL level than women, with no difference between Whites and Blacks. These results suggest that the DASH diet is likely to reduce the risk of CAD and can be recommended as an overall eating plan.²⁴¹ The current intake of fruits and vegetables is 3 servings/day each in the USA; only 23% consume the recommended 5 servings/day each.²⁴² The DASH diet is feasible in the real world, unlike the array of drastic diets which are impossible to continue for more than a few months.²⁴⁰

Flavonoids: Flavonoids are secondary metabolites that plants use to attract pollinators, repel predators, and to color flowers, leaves, and fruits.²⁴³ Important biological effects of flavonoids include the scavenging of oxygen-derived free radicals, inhibition of LDL oxidation, increase in HDL levels, and protection against CVD and several chronic diseases.^{244–248} The beneficial effects of these natural products on health were known long before the discovery of flavonoids. The major sources of flavonoids are vegetables (onions, kale, broccoli), fruits (apples, grapes, berries), olive oil, and beverages such as tea and wine.^{244,248,249} Other sources include grains, bark, roots, stems, and flowers. Flavonoids present in red wine could be partly responsible for the low CAD mortality seen in red wine drinkers (“French Paradox”). Red wine is the major source of flavonoid in France and Italy (40%), onions and apples in Finland, and olive oil in Greece.²⁵⁰ The strong taste of extra-virgin olive oil is partly caused by the abundance of flavonoids.

Antioxidants: Oxidative modification of LDL accelerates atherosclerosis whereas dietary antioxidants prevent LDL oxidation. These antioxidants include vitamin C, vitamin E, beta-carotene, selenium, flavonoids, magnesium, and MUFA.²⁵¹ It is worth emphasizing that vitamin pills are no substitute for a healthy diet. Although an earlier study suggested some benefits from antioxidant vitamin supplementation, several subsequent studies involving more than 100 000 patients have consistently failed to demonstrate any benefit. More recent studies suggest that possible harm may outweigh the benefit of these vitamins.^{252–254} In a recent study, the use of vitamins E and C reduced the lipid-lowering efficacy of statins and niacin by 50%. More importantly, the clinical event reduction was lowered from 90% to 60%.²⁵⁵ The current scientific evidence does not support any protective role of vitamins E, C, and beta-carotene supplements; their use only creates a diversion away from proven therapies.²⁵⁶ The US Preventive Service Task Force (USPSTF) recommends against the use of beta-carotene supplements.²⁵⁷ It is worth noting that the oxidative modification of LDL continues to be relevant, and people should obtain their antioxidant vitamins from food sources. (However, folic acid fortification is recommended in women who are pregnant or might become pregnant.)

Non-nutritive Food Adjuncts

Fiber: The term dietary fiber was coined to describe the plant cell wall removed during the refining process.²⁵⁸ Dietary fiber improves coagulation, fibrinolysis, insulin

sensitivity, LDL, and blood pressure levels.^{259–265} Fiber is particularly concentrated in bran. Insoluble fiber shortens the intestinal transit, resulting in less time for carbohydrate absorption.²⁶⁶ Soluble (viscous) fiber, such as beta-glucan, which is found in oat bran, delays gastric emptying, and slows the absorption and digestion of carbohydrates. These processes lead to a slower release of glucose into the circulation, resulting in a reduced demand for insulin.^{187,189,267} An intake of 16 g of total fiber is associated with a 12% decrease in CAD risk.²⁶⁸ FDA has permitted cardiovascular health claims to be made by the industry for 2 viscous fibers, beta-glucan and psyllium.²⁶⁹ Psyllium supplementation significantly lowers TC and LDL levels; it is safe and well tolerated.²⁷⁰

The benefit of whole grains appears to be mediated primarily through the greater intake of fiber, and is greater with cereal fiber than vegetable or fruit fiber.^{212,263,264,271,272} Approximately one-fourth of the fiber provided by cereal sources is water soluble.²⁶⁸ Cereal fiber consumption is associated with a 21% lower risk of incident CVD, and 30% lower risk of diabetes.^{164,188,259,273} (Fig. 2). Cereal fiber consumption may reduce the risk of CVD via the substitution effect, replacing the intake of other foods having potentially detrimental effects. In addition to cereal grains, legumes are also excellent sources of water-soluble dietary fiber. Half a cup of cooked beans contains, on an average, 6 g of total fiber and 2 g of soluble fiber.²⁷⁴

The current ADA recommendation for a healthy diet is to consume 25 g/day of fiber with about one-third from soluble fiber. In one study, type 2 diabetics consuming 50 g/day of fiber (25 g soluble, 25 g insoluble) lowered the blood sugar by 13 mg/dl, plasma TC by 7%, and TG levels by 10%.²⁷⁵ Thus, a high intake of dietary fiber, above the level recommended by the ADA, particularly of the soluble type, improves glycemic control, insulin levels, and plasma lipid concentrations in patients with type 2 diabetes.²⁷⁵

Plant sterols and stanols: Plant sterols and stanols are structural analogues of cholesterol. Low-fat plant stanol-containing margarines lower plasma LDL levels (by as much as 12%) in those with hypercholesterolemia by suppressing cholesterol absorption.^{276–279} In one randomized controlled study, the reduction in LDL with such supplements was similar to 20 mg of lovastatin (30% with statin v. 28.6% with diet).²⁸⁰ Various plant supplements have been shown to reduce LDL by 40% (Table 9).^{146,219,265,280–283}

Spices: Plants have the capacity to synthesize a diverse array of chemicals. Spices are aromatic vegetable substances, the significant function of which is food seasoning rather than nutrition. Typically, spices are the

Table 9. A portfolio of dietary factors useful for LDL reduction^{146,219,265,280–283}

		Decrease in LDL(%)
SAFA intake	<7%	10
Dietary cholesterol intake	<200 mg	5
Body weight	5 kg	5
Plant sterols/stanols	1–3 g/day	5
Soy protein	25 g/day	5
Nuts (almonds)	50 g/day	5
Viscous fiber intake	5–10 g/day	5
Total LDL reduction	Full portfolio	40

dried aromatic parts of plants, generally the seeds, berries, roots, pods, and sometimes leaves, that mainly grow in tropical countries. Common spices include turmeric, paprika, saffron, cinnamon, nutmeg, red and black pepper. In contrast, herbs used in cooking are typically composed of leaves and stems.²⁸⁴

Caffeine: Caffeine is found in coffee, tea, soft drinks, chocolate, and some nuts. Finland has one of the highest rates of per capita coffee drinking (13 kg/year).²⁸⁵ In a prospective study of 20 179 Finnish adults, coffee drinking was not associated with an increased risk of MI. However, consumption of large quantities of boiled unfiltered coffee raises cholesterol and homocysteine levels.^{285–287} In an experiment involving 10 volunteers, who consumed the equivalent lipid content of 6–7 cups of boiled unfiltered coffee daily for 6 weeks, the LDL levels increased by 33 mg/dl.^{288–290} These data suggest that daily consumption of 1–2 cups of coffee is safe with no particular health benefits or risks.

Tea: Tea, the most widely consumed beverage in the world other than water, has been associated with lower cardiovascular risk.^{291–295} Unlike coffee, tea consumption is associated with a substantial reduction in LDL levels. Tea is rich in flavonoids. Green tea contains catechins, whereas black tea, formed from the polymerization of catechins, contains theaflavins.²⁹¹ In one recent study, theaflavin-enriched green tea extract reduced the LDL level by 16%.²⁹⁶ Tea is the major source of flavonoid intake in Japan (>80%); the Japanese consume an estimated 7 cups/day of tea compared to half a cup/day in the USA. Adding milk to tea, as is common in the UK and India, abolishes the beneficial effect of tea.²⁹⁷

Alcohol: Moderate intake of alcohol (one drink a day for women and 2 drinks a day for men) may decrease the risk of CAD.²⁹⁸ Recently, it has been shown that only one drink per week is enough to provide cardiac protection (45 ml of spirits or 350 ml of beer or 120 ml of wine); the

cardioprotection is similar for beer, wine, whiskey, brandy, vodka, rum, and drinks in equivalent amounts.^{299,300} More than 2 drinks per day does not provide any additional protection and, in fact, the net effect may be harmful until the age of 45 years in men and 55 years in women.³⁰¹ Like carbohydrates, consumption of large quantities of alcohol raises TG levels.^{6,302} Other dangers of excessive alcohol consumption includes alcohol dependence, liver disease, high blood pressure, obesity, stroke, traffic accidents, spousal abuse, suicide, and breast and other cancers. Given these risks, the American Heart Association cautions people against increasing their alcohol intake or starting to drink if they do not already do so.

Weight Gain and Weight Loss Diets

Excess calories and obesity: Diets of any type containing more energy than needed or expended will lead to obesity and dyslipidemia.³⁰³ A calorie is a calorie whether it comes from carbohydrates, fat, or protein. Excess calories of any kind will eventually be converted by insulin to body fat.³⁰⁴ A common misconception is that dietary fat of any kind is fattening, while low-fat and high-protein diets have slimming properties. It is absolutely vital that both physicians and the public understand that it is the excess calories and not diet composition that causes weight gain.^{304–309} There is no evidence of weight gain with a high MUFA diet, compared with a high carbohydrate diet, under isoenergetic conditions.^{310,311}

Obesity is not only a reflection of overnutrition but also an important contributor to the mass dyslipidemia seen in India and western countries.⁶ Obesity in general is accompanied by the increased production of apo B and a decrease in the HDL levels.^{6,312} Humans have a limited capacity to store energy as carbohydrates. When carbohydrate intake exceeds storage and oxidation capacities, the excess is converted to fat by de novo lipogenesis that leads to high TG levels.³¹³ This process is increased several-fold in people with the metabolic syndrome which, if left untreated, leads to overt diabetes (25-fold risk).³¹² Body fatness and not lean body mass is the principal determinant of diabetes and prediabetes.³¹⁴ At a given BMI, Asian Indians have 7%–10% higher body fat; accordingly, BMI <23 is termed optimum; BMI 23–25 overweight, and >25 obese in Asian Indians. Likewise, the optimum waist circumference is lower in Asian Indians than Whites with a cut-off <90 cm in men and <80 cm in women.³¹⁵ Although obesity and dyslipidemia are uncommon in less affluent societies, some individuals may be excessively sensitive to caloric excess.⁶

Fast foods rapidly produce plaques. The average American gained 9 lb in the past decade. A third of vegetable taken in the USA are either French fries or potato chips.³⁰⁵ In one study, overweight subjects who consumed fairly large amounts of sucrose (28% of energy), mostly as beverages, had increased energy intake, body weight, fat mass, and blood pressure after 10 weeks. These effects were not observed in a similar group of subjects who consumed artificial sweeteners.³¹⁶ Restricting the dietary cholesterol can achieve a 3% reduction in TC level, whereas losing weight from trimming extra calories can reduce LDL by 5% to 20%.⁸

Weight loss: The recipe for effective weight loss is a combination of motivation, physical activity and caloric restriction; maintenance of weight loss is a balance between caloric intake, and physical activity, with life-long adherence. Each pound of body fat contains 3500 cal. Therefore, a person who consumes 500 cal less than he spends each day can lose 1 lb of fat a week. Any higher weight loss is due to a more severe caloric restriction or water loss rather than fat loss. The minimum caloric intake in a medically unsupervised weight loss diet is 1500 cal/day for men, and 1200 cal/day for women. Superior long-term participation and adherence is observed in a high-fat diet rather than a low-fat diet (35% v. 20%), especially in western cultures.³⁰⁹ The greater success rate is due to higher palatability of the high-fat diet provided by mixed nuts and lean meat.³⁰⁹ Furthermore, the long-term outcome of a reduced-fat diet consumed ad libitum for weight control is dismal. In one study, compared with the control group, weight decreased in the reduced-fat diet group significantly by 3 kg in 1 year but diminished to an insignificant 1 kg by 5 years.³¹⁷ Until more information becomes available, “the prudent diet,” which is a balanced diet, is the one to follow for young and old alike.³¹⁸

Very low fat diet: Some experts have argued for a very low-fat diet (<10%).³¹⁹ Since these diets are not high-protein diets (like the Atkins diet), they are in reality very high in carbohydrate. High-carbohydrate diets (the Macrobiotic diet) increase insulin resistance and induce the metabolic syndrome. In controlled trials, low-fat, high-carbohydrate diets decreased HDL levels. Replacing 10% of energy from SAFA with carbohydrate lowers the HDL levels by 5 mg/dl, even when the carbohydrate consumed is complex.^{62,173,320,321} There is also a marked increase in TG level, which makes LDL small, dense, and more dangerous.^{173,320–322} The effect is strongest when carbohydrates replace SAFA but is also seen when carbohydrates replace MUFA and PUFA. The effect is seen

in both short- and long-term trials, and is therefore not a transient phenomenon. Therefore, replacement of SAFA must be achieved through increasing MUFA and not by carbohydrates. The adverse effects of high-carbohydrate diets (high glycemic load) in the metabolic syndrome and diabetes have not received due attention, especially in the Indian literature. The recommended carbohydrate intake is <50% of calories in people with the metabolic syndrome or diabetes (NCEP).

The allure and dangers of very low-carbohydrate, high-protein diets: High-protein diets that are extremely low in carbohydrates are touted as a new strategy for successful weight loss by many.³²³ Most such diets contain <10% carbohydrates, 25%–35% protein, and 55%–65% fat. Because the protein is provided mainly by animal sources, these diets are high in SAFA and cholesterol. Thus, these diets are truly high-fat diets masquerading as high-protein diets. Advocates of this diet often promote serious misconceptions about carbohydrates, insulin resistance, ketosis, and fat burning as the mechanisms of action for weight loss. To avoid excess load on the kidneys, the total protein intake should not exceed 100 g/day.³²⁴ More importantly, the body has an obligatory requirement for glucose of about 100 g/day, largely determined by the metabolic demands of the brain.^{324,325}

In randomized studies, the extent of weight loss was small (4 kg), and adherence to the diet was low.^{326,327} In one study, although a low-carbohydrate diet produced a 4% greater weight loss at 6 months than did the conventional diet, the differences did not persist at 1 year. Furthermore, adherence was poor, and attrition was high in both the high- and low-carbohydrate groups. Longer and larger studies are required to determine the long-term safety and efficacy of low-carbohydrate, high-protein, high-fat diets.³²⁶ Two recent studies have provided insight into high-protein diets; the initial weight loss is due to fluid loss and ketosis-induced appetite suppression. The monotony of this diet also results in involuntary caloric restriction.^{326,327}

The beneficial effects on blood lipids and insulin resistance are due to the weight loss, and not the change in caloric composition. Such diets increase LDL but decrease TG levels, in sharp contrast to high-carbohydrate diets, which increase TG, and decrease HDL levels. Although these diets may not be harmful for most healthy people over a short period of time, there are no long-term scientific studies to support their overall efficacy and safety. Markedly atherogenic profiles have also been reported in children with ketogenic diets. At 6 months, the high-fat ketogenic diet significantly increased plasma LDL levels by 50 mg/dl,

triglycerides 58 mg/dl, apo B 49 mg/dl, and non-HDL cholesterol 63 mg/dl. The mean HDL-cholesterol levels decreased significantly.³²⁸ These lipid abnormalities in children are more than likely to translate into a high risk of heart disease as young adults.

High-protein diets also do not provide the variety of foods needed to continue the diet on a long-term basis. High-protein diets are not recommended, and are perhaps dangerous because they restrict most healthful foods that provide essential nutrients, especially fruits and vegetables. Individuals who follow these diets are therefore at risk for compromised vitamin and mineral intake, as well as potential cardiac, renal, bone, and liver abnormalities overall.³²⁴ The consumption of a very low-carbohydrate diet for 6 weeks delivers a high acid load to the kidney, increases the risk of stone formation, decreases body calcium, and may increase the risk of bone loss and fractures.³²⁹ A high-protein diet is the ultimate antithesis of the prudent diet. It is important to realize that diets are not for 6 weeks, 6 months or 6 years, but for a lifetime. Although most quick-fix diets have a short-term success rate >90%, the long-term failure rate is 100%.

Healthy and Contaminated Vegetarian Diets

Omnivores or nonvegetarians outnumber vegetarians 10 to 1 in western cultures. Vegetarians include vegans who do not consume any animal products, ovo-vegetarians who consume egg, lacto-vegetarians who consume milk, ovo-lacto-vegetarians who consume egg and milk, and semi-lacto-vegetarians who eat small amounts of meat (<1 time/week). Ironically, most self-defined vegetarians in western countries consume red meat and poultry, albeit infrequently, and in very small quantities. In a recent survey, only 1% of self-reported vegetarians did not eat meat in the USA, whereas about 6% of Americans who do not consume any meat did not identify themselves as vegetarians.^{330–332}

Western vegetarians generally consume a healthier diet than omnivores; healthy foods such as soy, nuts, legumes and vegetables replace meat.³³³ They generally have twice the fish consumption of nonvegetarians.³³⁰ This is not the case with Indian vegetarians who shun fish. US vegetarians eat more whole-grain products, dark green and deep yellow vegetables, whole-wheat bread, brown rice, soy milk, tofu, meat substitutes, legumes, lentils, garbanzos, walnuts, and pecans.³³⁰ However, they eat the same amount of food as omnivores (1000 kg/year) but are usually thinner.³³⁴ A healthy vegetarian diet is characterized by more frequent

consumption of fruits and vegetables, whole grains, legumes and nuts, resulting in higher intakes of dietary fiber, antioxidants and phytochemicals.³³⁵ Thus a vegetarian diet contains a portfolio of natural products that can improve both the carbohydrate and lipid abnormalities in diabetes.¹⁸⁷

Vegetarians eat about two-thirds of SAFA, and one-half of cholesterol as omnivores; vegans consume one-half of SAFA and no cholesterol.^{9,336} Cholesterol levels among western vegetarians are 15–25 mg/dl lower than omnivores.^{337–340} Vegans have very low levels of LDL.^{341,342} Nuts, viscous fibers (from oats and barley), soy proteins, and plant sterols in vegetarian diets improve serum lipid levels.³³⁷ Furthermore, substituting soy or other vegetable proteins for animal proteins reduces the risk of developing nephropathy in type 2 diabetes.

With the exception of tropical oils, calories from plant sources are negatively correlated with CAD mortality, whereas calories from animal sources are positively correlated.¹⁶ Olive oil, fresh fruits, and vegetables are protective against heart disease, and seem to play a greater role in the French paradox than wine.¹⁶ Greater consumption of whole milk and other animal products were important contributors to Finland having the highest rates of CAD.¹⁶ In a prospective study of 4671 vegetarians and 6225 nonvegetarians, followed up for 10–12 years, BMI, TC, and CAD mortality was substantially lower among vegetarians than in the nonvegetarians. Other studies also suggest a protective effect of vegetarianism against many diseases. Vegetarians in western countries, but not in India, enjoy remarkably good health, exemplified by low rates of obesity,^{334,343} diabetes,³⁴⁴ CAD^{345–347} and cancer,³³⁷ and a 3–6 year increase in life expectancy.^{333,348} It is not clear whether this is due to abstinence from meat or to a greater consumption of heart-healthy food.³⁴⁹

Indian vegetarianism, a form of “contaminated vegetarianism”: Most Asian Indians are lacto-ovo-vegetarians, unlike western vegetarians. About 50% of Asian Indians are vegetarians, but their lipoprotein levels, and rates of diabetes and CAD are no different from those of nonvegetarians.^{350,351} This phenomenon is due to contaminated vegetarianism, wherein vegetarians manage to consume excessive amounts of SAFA and TRFA. In the CADI study, Asian Indian physicians in the USA followed a heart-healthy diet, with 32% energy from total fat, and 8% from SAFA, which is the recommendation by the NCEP.³⁵⁰ This appears to be an exception rather than the rule. In a Canadian study, Asian Indians consumed more fried foods and high-fat dairy products, such as full-fat milk than White

Canadians.³⁵² Although the intake of fat is 20%–25% energy in most Asian countries, many affluent Asian Indians consume >50% of their calories from fat.

Indian vegetarians consume liberal amounts of bakery products, butter, *ghee*, cheese, ice cream, curd, and other dairy products to overcompensate for not using meat. Contrary to popular belief, dairy products are the major source of SAFA, even in the western diet. It is worth highlighting that SAFA intake from high-fat dairy products increases LDL levels 3 times as much as it raises the HDL level.³⁵³ Meat is expensive, and consumed in very small quantities by Indian omnivores because of cultural and financial reasons. This is in sharp contrast to an annual per capita consumption of 124 kg meat and 23 kg fish by Americans.³⁵⁴ Prolonged cooking of vegetables, as is practised in India, virtually destroys every nutrient before it is consumed. A major problem overlooked in the Indian diet is the high glycemic load, resulting in high TG and low HDL levels.³⁵⁵ There appears to be a threshold for carbohydrate consumption with an intake >280 g/day often resulting in atherogenic dyslipidemia.³⁵⁰

Deep-frying and reuse of frying oil: Deep-frying, a common form of cooking among Asian Indians, is associated with spontaneous hydrogenation, and the formation of TRFA. Reuse of oil used for deep-frying has been shown to produce endothelial dysfunction.³⁵⁶ Repeated reuse of such oil is exceedingly common among Asian Indians.³⁵⁷ HDL inhibits LDL oxidation primarily through its paroxonase activity; reuse of frying oil reduces paroxonase activity, and thus reduces the ability of HDL to prevent LDL oxidation.^{356–360} Fats that have been heated for prolonged periods in air contain many dangerous products from oxidation and breakdown of lipids. These compounds include hydroxy peroxides, aldehydes, polymers, hydroxy fatty acids, hydroperoxy epoxides, and hydroperoxy alkenals.³⁶¹ In one study, fast-food restaurant cooking oil, just before the weekly change, was compared to unused oil. The repeatedly used oil had 4 times higher peroxide levels, 7 times higher carbonyl levels, and 17 times higher levels of acids.³⁵⁷

Ghee: *Ghee* is one of the most important sources of dietary fat and a common cooking medium.^{362,363} Use of *ghee* for deep-frying is considered gourmet among Asian Indians. *Ghee* or clarified butter is anhydrous milk fat, and is rich in MUFA (32%) and SAFA (62%), most of which are cholesterol-raising (myristic acid 17%, palmitic acid 26%). It is perhaps more harmful than butter due to the added presence of cholesterol oxides, which are generated during

its preparation by prolonged heating of butter.^{362–364} Liberal dietary exposure to cholesterol oxides from *ghee* is a likely contributor to the high frequency of CAD among Asian Indians.³⁶⁴ There are conflicting data on the risk of heart disease with *ghee*.^{365,366} We are unaware of any biological explanation as to why Asian Indians can be immune to the unfavorable effects of butter and/or *ghee*. In addition to milk *ghee*, vegetable *ghee* (*vanaspathi*) is also immensely popular in Indian cooking, which exerts similar adverse effects through its high TRFAFA content.

Tropical oils: The term tropical oils refers to coconut, palm kernel, and palm oils. These oils contain a very high percentage of SAFA, unlike other vegetable oils such as rapeseed oil (mustard oil), sesame oil, and rice bran oil, which are low in SAFA and high in MUFA (Table 3). Tropical oils are more atherogenic and thrombogenic than mutton and beef fat; the latter contains <5% myristic acid compared to 18% in coconut and palm kernel oils.¹⁰⁴ In fact, these oils contain more TC-raising SAFA than animal fats—coconut oil 89%, palm kernel oil 71%, and palm oil 46% compared to <30% for butter fat, beef fat, pork fat, and chicken fat (Table 5).^{6,147} Tropical oils account for <2% of energy (<4 g/day) in the USA, but 25% or more in many other countries.^{147,367} Tropical oils are also found in commercially baked cakes, biscuits, cookies, and “snack foods”. In Mauritius, a regulated change in the SAFA content by substituting soybean oil for palm oil resulted in a dramatic 32 mg/dl fall in TC level, and underscores the crucial role of cooking oils in population levels of TC.³⁶⁸

Coconut oil: Coconut oil contains mostly cholesterol-raising SAFA (8% caprylic, 6% capric, 45% lauric, 17% myristic, and 8% palmitic acid).³⁶⁹ Rabbits fed a commercial chow diet containing 0.5% cholesterol and 14% coconut oil developed more severe dyslipidemia and atherosclerosis than rabbits fed the same diet containing olive oil instead of coconut oil. The average plasma TC level was 2-fold, and TG level 20-fold higher in the coconut oil-fed rabbits than in the olive oil-fed rabbits.²⁸ Cox et al.^{369,370} have reported the cholesterol-raising effects of coconut oil to be similar to that of butter. In a comparative study of diets rich in beef fat versus coconut oil, the plasma TC, LDL, and HDL responses were lower with beef fat than coconut oil, commensurate with the lower proportion of cholesterol-raising SAFA in beef (29%) than coconut oil (89%)³⁷¹ (Table 5). A Malaysian study in which 22% of the energy intake was substituted with coconut oil found an increase of 40 mg/dl in TC, 29 mg/dl in LDL, 36 mg/dl in TG, and 4 mg/dl in HDL levels.³⁷² The impact on LDL and HDL by using

various fats as the sole source of fat in a Dutch population is shown in Fig. 1. Note the marked increase in LDL in contrast to HDL with the use of coconut oil.

Kerala, renowned for the universal and liberal consumption of coconut milk and oil, not only has the highest level of TC in India, but also the highest rate of CAD.³⁷³ The proportion of subjects with high TC (>239 mg/dl) in Kerala is almost double that of the USA. (32% v. 18%).³⁷⁴ This is in sharp contrast to the Japanese among whom only 6% have high TC.³⁷⁵ In Sri Lanka, which also has a very high rate of CAD, about 80% of the fat in the habitual diet comes from coconut.^{134,374,376}

Consumers need to be educated about the atherogenic and antiatherogenic effects of various cooking oils, as well as animal and vegetable *ghee*. There is little awareness, and even controversy, about the atherogenic effects of certain foods and oils, especially in regions where the production, sale, and consumption of such oils have a profound impact on the regional economy.

Prudent Diet for All Ages and the Entire Population

The traditional Mediterranean diet is characterized by abundant plant foods (vegetables, breads, pastas, beans, nuts, and seeds). Fresh fruit is the typical daily dessert, and olive oil is used as the principal source of fat. Dairy products (principally cheese and yogurt), fish, and poultry are consumed in low-to-moderate amounts. Red meat and egg are consumed in low amounts (0–4 eggs weekly). Wine is consumed in low-to-moderate amounts, normally with meals. This diet is typically high in total fat (35%–45%) but low in SAFA (7%–8% of energy). Greater adherence to the traditional Mediterranean diet is associated with a significant reduction in total mortality.³⁷⁷ The 6 beneficial components of this diet have recently been elucidated. They are vegetables, legumes, whole-grain cereal, fish, fruit, and nuts, which form the basis for the “prudent diet”³⁷⁷ (Table 10).^{199–203,225–227,231–236}

According to the new paradigm, dietary pattern rather than individual nutrients appears to be more important. Recent research suggests the existence of a food synergy in which the beneficial effects of healthy foods are magnified when several different types of foods are consumed.²¹³ Hu et al. have developed the concept of “prudent diet” (modified from the Mediterranean diet).^{30,378–380} The “prudent diet” has a higher intake of vegetables, fruits, legumes, whole grains, fish, and poultry, whereas the “western diet” is characterized by a higher intake of red meat, processed meat, refined grains, sweets, desserts, French fries, and

Table 10. The benefits and sources of selected heart-healthy foods^{199–203, 225–227, 231–236}

Category	Benefits	Sources/preferred foods
Whole grains	Good source of fiber, minerals, and vitamins E and B Delay gastric emptying and blunts postprandial insulin response Improve insulin sensitivity ↓ Insulin secretion ↓ Insulin resistance, and diabetes Improve lipids (↑ HDL) ↓ Hypertension ↓ CVD incidence and CVD mortality	Whole wheat, oats, rye, barley, corn, brown rice and wild rice Whole-grain oatmeal, whole-grain, pumpernickel, rye or dark bread Whole-grain, ready-to-eat cereal Cooked cereal Pasta Popcorn Bran Buckwheat, cracked wheat, wheat germ
Fruits and vegetables	Rich in fiber, flavonoids, and antioxidants ↓ Blood pressure ↓ LDL ↑ HDL ↓ Homocysteine ↓ CVD and mortality ↓ Cancer, especially GI tract ↓ All-cause mortality	Green leafy vegetables Carotenoid vegetables (carrots, broccoli, spinach, lettuce, tomato, and yellow squash) Citrus (vitamin C-rich) fruits Temperate fruits (cantaloupe, apple, pear) Tropical fruits (mango, papaya, pineapple, jackfruit, guava, banana)
Viscous fiber	↓ LDL ↓ Postprandial glycemia	Cereal grains (oats—beta glucan) Legumes, Psyllium (Metamucil)
Soy protein	↓ LDL	Soy bean
Plant sterols and stanols	↓ LDL	Low-fat plant stanols containing margarines
Nuts	↓ LDL ↑ HDL	See Table 6

high-fat dairy products.^{378,380} The “prudent diet” is associated with a 24% decreased risk of CVD compared to a 46% increased risk with the western diet.^{215,380}

Consumers are bombarded on a daily basis with the Babel of nutritional breakthroughs.³⁸¹ Food companies advertize their products, nutrition researchers publicize their latest results, and the media are more interested in a controversial story than in scientific facts.³⁰ Trivial reports are often publicized as major breakthroughs by the media, and cause confusion among consumers. It is difficult for most journalists and consumers to tell the difference between a major research finding and a trivial report.³⁸¹

The dangers of the current western diet and the contaminated vegetarian diet, and the remarkable benefits of the prudent diet need to be disseminated among cardiologists, physicians, and the public. This diet can be sustained lifelong but needs to be adapted to Indian ingredients and cooking methods. Several countries have developed dietary guidelines to reduce nutritional information anarchy. The Indian consensus on the prudent diet should incorporate scientific facts, and the cultural preferences appropriate for different parts of India. Such

information needs to be adopted by the scientific community, and adapted by the food industry.

Current Knowledge on Preventive and Therapeutic Nutrition

Randomized, controlled clinical trials, meta-analysis, and systematic reviews are considered the ultimate tests of the benefits of therapeutic interventions. Such reviews have shown a 24% reduction in major coronary events in dietary trials lasting >2 years.³⁸² The TC/HDL ratio is the single best lipid predictor of CVD. This ratio is determined by 3 partly opposing dietary factors—the proportion of energy from SAFA, which raises TC; the proportion of energy from total fat, which raises HDL; and the excess in total energy intake, which produces obesity and secondarily lowers HDL.²⁰ The greatest reduction in CVD risk is achieved by LDL-lowering by reducing SAFA intake. Decreasing SAFA intake is best accomplished by reducing the intake of high-fat dairy products, and increasing fiber-rich foods. A diet incorporating lean beef, skinless chicken, and fatty fish has been shown to improve the lipid profile by 5%–10%.³⁸³

The preferred replacement for SAFA is MUFA or PUFA

and not carbohydrates (Table 11). Replacing SAFA with carbohydrates decreases the LDL levels but makes LDL small, dense, and more dangerous by increasing the TG levels.³⁰ Substituting carbohydrates with MUFA decreases the LDL level, and increases the HDL level. PUFA and MUFA increase insulin sensitivity, and decrease the risk of type 2 diabetes.^{45,384–386} Substantial evidence indicates that diets using MUFA and PUFA as the predominant form of dietary fat, an abundance of fruits and vegetables, and adequate n-3 fatty acids can offer significant protection against CAD, stroke and diabetes (Table 8). Adequate consumption of fruits and vegetables provides most of the necessary antioxidants, and are preferable to dietary supplements in the form of pills. Replacing a high glycemic with a low glycemic index, and reducing the glycemic load can reduce the risk of diabetes¹⁶¹ (Table 6).

Nuts, once deemed unhealthy because of their high fat content, have become an important part of diets designed to control weight, lower blood pressure and cholesterol, and achieve secondary prevention of CAD, besides adding variety, texture and flavor to dishes.^{145,211,215,387} Unless a beneficial effect is clearly demonstrated by well-designed scientific studies, the liberal use of butter, *ghee*, palm oil,

and coconut (oil and milk) should be discouraged. However, in diets with a negligible intake of fish, meat, milk, and dairy fat, the modest use (<7% of energy) of such oils may be preferable to no fat at all.

Practical Recommendations

Better food habits can help reduce the risk of diabetes, MI, stroke, and death. A healthy eating plan means choosing the right foods to eat, and preparing them in a healthy way. A healthy diet involves a decrease in the use of refined grains, tropical oils, egg yolks, animal, dairy, and hydrogenated fats, and an increase in the consumption of whole grains, vegetables, nuts, legumes, and fruits.⁷ Increasing the MUFA intake up to 20% of energy, as a replacement for SAFA and carbohydrates, may help prevent and treat the metabolic syndrome, diabetes, and CVD. Such a strategy can also significantly reduce the need for lipid-optimizing drugs. Since meat contains one-third MUFA and one-third cholesterol–neutral stearic acid, its consumption can also be incorporated into a healthy diet, provided lean cuts are used, and the quantity limited to 150 g/day.⁶ Practical recommendations on diet are given in Table 12.

Table 11. Summary of current knowledge on diet

- Quality of fat is more important than the quantity of fat consumed.
- SAFA is the principal determinant of elevated LDL levels. Reduction in energy intake from SAFA is the cornerstone of dietary modification. Dairy products provide more SAFA than meat, even in western diets.
- Contrary to common belief, MUFA and PUFA can significantly lower LDL levels. Replacement of 20% of energy from SAFA with MUFA or PUFA decreases TC level by 40 mg/dl.
- The atherogenicity and thrombogenicity of tropical oils are several times higher than meat.
- Increase in LDL level from lean meat is no higher than chicken, and need not be eliminated from a healthy diet.
- n-3 PUFA found in fatty fish is antithrombotic, antiarrhythmic, and prevents sudden death.
- The adverse effects from TRAEA consumption are greater than those from SAFA, because of increase in LDL and Lp(a) levels, and decrease in HDL level.
- Both quality (glycemic index) and quantity (glycemic load) of carbohydrates are important determinants of insulin resistance and the metabolic syndrome. A high glycemic index or load portends an inferior quality of carbohydrates.
- Although carbohydrates do not raise LDL levels, a high glycemic load is the major determinant of postprandial lipemia.
- Vegetarian diet is unhealthy if it contains large amounts of SAFA and TRAEA.
- Nuts, fruits, vegetables and whole grains can each reduce the risk of CVD by 15%–45%.
- Nuts are wholesome and nutritious food.
- The risk from alcohol outweighs the benefit in men <45 years and women <55 years of age.
- Drinking coffee does not increase the risk of heart disease but consumption of large amounts of unfiltered coffee can increase TC levels.
- Tea is rich in antioxidants and flavonoids, and is associated with a reduced risk of CAD; however, most of the beneficial effects are neutralized with the addition of milk.
- A calorie is a calorie, whether derived from carbohydrates, protein or fat.
- Obesity is a reflection of overnutrition (caloric excess), and a major cause of dyslipidemia.
- Caloric excess, irrespective of the composition of food, can raise LDL and lower HDL levels as a secondary effect of obesity.
- Weight reduction seen in high protein diets is due to the involuntary caloric restriction from the monotony of the diet rather than the diet composition.

Table 12. Dietary recommendations

- A minimum carbohydrate intake of 100 g/day is necessary but should not exceed more than 300 g/day.
- Indians with or predisposed to the metabolic syndrome and diabetes should limit carbohydrate intake to <50% of energy.
- Most carbohydrate calories should be from whole grains and low glycemic index foods. A total fat intake of 30%–35% is preferable to a very high carbohydrate diet.
- Reduce SAFA intake to <7% of the daily energy (10 g for women and 15 g for men), and cholesterol intake to <200 mg/day.
- Substitute excess SAFA and carbohydrate calories with MUFA ($\leq 20\%$) and PUFA ($\leq 10\%$).
- Substitute full-fat with low-fat milk and dairy products.
- Choose lean meat or skinless poultry, and limit the amount to <150 g/day.
- Minimize the intake of TRAFA by avoiding fried or crispy foods.
- Use cooking oils with beneficial effects on lipids and avoid deep-frying, especially with previously used oils.
- Consume 2–3 fish meals (200–300 g) per week; avoid frying to maintain the benefits.
- Those who are unable to consume fish may take the equivalent of 1g/day of EPA and DHA (3g of fish oil); 2 g/day is needed for those with heart disease or high TG.
- Protein intake, up to 25% of energy, is permissible if most of the protein is from plant sources.
- Control caloric intake to achieve and maintain optimum weight and waistline.
- Increase physical activity to >60 min every day.
- Reduce intake of salt to <6 g/day.
- Eat a variety of foods, including whole grains, nuts, legumes, fruits, and vegetables.
- Consume nuts, up to 60 g/day, as a substitute for unhealthy foods.
- Increase intake of fruits to ≥ 5 servings/day (500 g/day), and use whole fruits instead of juices.
- Increase intake of vegetables to ≥ 5 servings/day (500 g/day), and avoid prolonged cooking of vegetables.
- Limit alcohol intake to 1–6 drinks/week.

Conclusions

People eat specific foods because of their taste, easy availability and affordability, but are often unaware of the health benefits and risks. Dietary modifications remain the cornerstone of both the treatment and prevention of diabetes and CVD, the twin epidemics of the twenty-first century.^{234,388–391} A prudent diet together with regular physical activity, avoidance of smoking, and maintenance of a healthy body weight may prevent the majority of diabetes and CVD in the Indian population.³⁰ Aggressive dietary interventions may reduce CVD events to a similar magnitude as that achieved with statins. Compared with medical or surgical interventions, nutritional intervention is low-risk, low-cost, and readily available.²⁵⁹ A variety of whole grains, not refined grains, as well as various types of fruits and vegetables should be the main form of carbohydrates.²⁴² Prolonged cooking of vegetables should be avoided. It is important to realize that the vegetarian diet is healthy only when it is low in SAFA, and the predominant energy is from foods with a low glycemic index.²¹⁵ The best way to counter the perils of contaminated vegetarianism is by substituting full-fat dairy products with low-fat dairy products. Cooking oils containing high SAFA should be replaced with those containing high MUFA. Deep-frying, especially with previously used oils, should be discouraged. Nuts are healthy, wholesome foods, and their use should be encouraged as a replacement for unhealthy calories.

A diet rich in fish has multiple benefits, including raising HDL, and lowering TG levels, and preventing sudden death.³⁸³ Consumption of fish is preferable to taking a large number of fish oil capsules. There is increasing evidence that dietary and lifestyle modifications begun in childhood are likely to have benefits later in life.³⁹² Therefore, these dietary guidelines are applicable to all Asian Indians >2 years of age, and not just those with diabetes or heart disease.

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MRC Human Nutrition Research, Elsie Widdowson Laboratory, Cambridge, UK. Abstract. Objective: To review the evidence on diet and nutrition relating to osteoporosis and provide recommendations for preventing osteoporosis, in particular, osteoporotic fracture.

Approach: Firstly, to review the definition, diagnosis and epidemiology of osteoporosis, to discuss the difficulties in using bone mineral density to define osteoporosis risk in a world-wide context and to propose that fragility fracture should be considered as the disease endpoint. Secondly, to provide an overview of the scientific data, the stre