The Rationale for Controlling Dietary Lipids in the Prevention of Coronary Heart Disease

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Despite the acknowledged association between blood cholesterol levels and coronary heart disease, many people throughout the Americas remain uncertain about the importance of that association and available countermeasures. This article reviews the subject’s historical background, results of recent studies, and current recommendations of leading expert groups in the United States. It also examines ways in which the body’s cholesterol level is influenced by changes in the dietary intake of calories, saturated and unsaturated fats, and cholesterol, and recommends the adoption of specific dietary measures as a way of markedly improving public health.

A

n association between blood cholesterol levels and the severity of atherosclerosis was first proposed over 100 years ago. Since then, the results from various types of epidemiologic (longitudinal, cross-sectional, migration, and autopsy) studies and clinical trials have supported a cause-and-effect relationship between blood cholesterol, atherosclerosis, and coronary heart disease.

In order to understand how dietary factors may increase blood cholesterol levels, it is important to know some basic facts about fats, fatty acids, and cholesterol—which are collectively referred to as “lipids.”

Fat is present in most foods, regardless of whether the foods are of plant or animal origin. The amount of fat present varies, ranging from trace amounts in leafy vegetables to predominant amounts in food ingredients such as cooking oils. Dietary fats provide the most concentrated source of energy in the diet, each gram of fat contributing slightly more than nine kilocalories (kcal). Animal tissues, dairy products, and oils extracted from certain seeds and plants provide most of the fats in the diet—oils being defined as fats that are usually liquid at room temperature.

The principal breakdown products of fats are fatty acids and glycerol. A fatty acid may be saturated or unsaturated. A saturated fatty acid contains no double bonds, and all available carbon positions are “saturated” with hydrogen atoms. The degree of unsaturation is determined by the number of carbon atoms within the fatty acid that are double-bonded to each other. A fatty acid with a single double bond is called “monounsaturated,” while a fatty acid with two or more double bonds is usually referred to as “polyunsaturated.” Fats from plant as well as...
animal sources usually contain both saturated and unsaturated fatty acids. However, fats from animal sources usually contain more saturated fatty acids than those from plant sources.

Cholesterol differs from fats and fatty acids in that it occurs in significant amounts only in animal tissues and is present in relatively small quantities. Indeed, when measured in animal foods it is typically measured in milligrams per serving, while fat is measured in grams per serving.

Cholesterol, which is synthesized by the body, also differs chemically and functionally from fat. While both are lipids, cholesterol is a complex substance belonging to a class of chemical compounds called "sterols," while fats belong to a class called "glycerides." Fats are associated mainly with the body's production and storage of energy; in addition, they supply the body with insulation and help cushion some of our internal organs. In contrast, cholesterol plays an essential role in the synthesis of cell membranes, steroid hormones, and bile acids. On the negative side, elevated blood levels of cholesterol can produce adverse health effects of great consequence.

**DIET AND HEART DISEASE**

Coronary heart disease is the single most common cause of death and disability in 31 of PAHO's 35 member countries. Indeed, it accounts for more deaths annually than any other disease, including all forms of cancer combined, and the trend seems to be increasing. Many young and highly productive people have symptomatic coronary heart disease, while others have asymptomatic disease that has not yet been diagnosed.

Besides being limiting, costly, and often painful to the individuals involved, coronary heart disease constitutes an important direct and indirect drain on national economies. Moreover, as populations tend to age and become more urbanized, individual exposure to the risks of coronary heart disease can be expected to increase. Consequently, the public health prognosis regarding coronary heart disease in the Americas is not good, and preventive measures are seriously needed.

For many individuals, the severity of atherosclerosis increases in a linear fashion with rising plasma cholesterol levels. According to Grundy (1), when 60% of a coronary artery's surface is covered with raised atherosclerotic lesions—or fibrous plaques that contain significant amounts of cholesterol and its fatty acid esters—one crosses a critical threshold for increased risk of clinical coronary heart disease, as shown in Figure 1.

When the arterial wall is injured mechanically (as a result of smoking, hyper-

![Figure 1. A diagram of the relationship between coronary atherosclerosis (based on the percentage of the surface of coronary arteries covered with raised lesions) and age at different levels of plasma cholesterol. When 60% of the coronary artery surface is covered with lesions, patients enter the zone of markedly enhanced risk for clinical coronary heart disease. From S.M. Grundy, JAMA, 28 November 1986, vol. 256, no. 20, pp. 2849–2856 (1).](image-url)
tension, diabetes mellitus, nutritional deficiencies, percutaneous transluminal coronary angioplasty, and perhaps even viral diseases), the artery becomes more susceptible to plaque development. In addition, several other reversible or controllable risk factors such as obesity and physical inactivity seem to be linked to development of atherosclerosis (1-4).

Cholesterol, which is water-insoluble, and other lipids are transported through the blood by lipid-protein complexes (called lipoproteins) of varying densities. Low-density lipoproteins (LDLs) carry two-thirds or more of the blood cholesterol. LDL delivers cholesterol to the tissues for uptake by specific receptors, which in turn inhibits cholesterol synthesis. Increased levels of LDL are related to increased risk of coronary heart disease. When there are insufficient LDL receptors, increased blood levels of cholesterol result as well as increased cellular cholesterol synthesis, with cholesterol being deposited within the artery endothelium—initiating the atherosclerotic process (1, 5). Another lipoprotein, high-density lipoprotein (HDL), is a lipid scavenger; it removes cholesterol from cell membranes and transports it to the liver for degradation as bile salts. HDL, associated with this “reverse transport” function, appears to have antiatherogenic properties; and HDL-cholesterol levels are inversely associated with coronary heart disease risk (1, 5).

EXPERT OPINIONS

There has been longstanding controversy as to whether healthy individuals should consciously lower their consumption of dietary fat and cholesterol in an effort to prevent coronary heart disease. Historic findings relating to this question include the following:

In 1979 the U. S. Surgeon General issued a report on health promotion and disease prevention entitled Healthy People (6). The report concluded, on the basis of what was then known or strongly suspected about the relationship between diet and disease, that Americans would probably be healthier if, besides making other dietary changes, they consumed less saturated fat and cholesterol. This position was reaffirmed by public advice to “avoid too much fat, saturated fat, and cholesterol” that was contained in the Dietary Guidelines for Americans published jointly by the U. S. Department of Health and Human Services and the U. S. Department of Agriculture in 1985 (7).

A conference aimed at developing a consensus on lowering blood cholesterol levels was held by the U. S. National Institutes of Health on 10-12 December 1984. The consensus panel of experts concluded in its report that “elevated blood cholesterol level is a major cause of coronary artery disease,” and that “there is no doubt that appropriate changes in our diet will reduce blood cholesterol levels.”

The panel cited epidemiologic data and over a dozen clinical trials that it said offered reasonable assurance that reducing blood cholesterol levels would afford significant protection against coronary heart disease. Accordingly, it recommended that “all Americans [except children under two years of age] be advised to adopt a diet that reduces total dietary fat intake from the [then] current level of about 40% to 30% of total calories, reduces saturated fat intake to less than 10% of total calories, increases polyunsaturated fat intake but to no more than 10% of total calories, and reduces daily cholesterol intake to between 250 and 300 mg” (4).

For some time, the American Heart Association (AHA) has recommended that most people should lower their dietary intake of lipids, particularly cholesterol and saturated fatty acids. At present the association recommends that healthy
adults derive less than 30% of their total caloric intake from fats, and that cholesterol intake be less than 100 mg per 1,000 calories, not to exceed 300 mg per day (2, 8).

In 1984 the Inter-Society Commission for Heart Disease Resources in the United States published a statement that recommended reducing dietary cholesterol to no more than 250 mg per day, reducing total fat intake to less than 30% of total caloric intake, and adjusting fat intake so that saturated fats provided no more than 8% of the total calories (9). This commission was created in 1969 to develop guidelines for optimal medical resources to prevent and treat cardiovascular disease. Its members include the AHA, the American Medical Association, the American College of Cardiology, the American College of Physicians, the American Public Health Association, and 12 other medical associations.

In 1982 the World Health Organization Expert Committee on Prevention of Coronary Heart Disease recommended that in countries with a high incidence of coronary heart disease, blood cholesterol levels should be lowered through progressive changes in eating patterns. These patterns should include limitation of cholesterol intake to less than 300 mg per day, limitation of total fat energy to 30% of total energy, and limitation of saturated fat energy to less than 10% of total energy (10).

CLINICAL STUDIES

In January 1984 the National Heart, Lung, and Blood Institute (NHLBI) released the results of a 7 to 10 year clinical study it had sponsored that was known as the Lipid Research Clinic's Coronary Primary Prevention Trial. These results showed that reducing serum cholesterol by means of the cholesterol-lowering drug cholestyramine (a bile acid sequestrant) resulted in a reduction in fatal and nonfatal heart attacks (11). In addition, the study results revealed a linear correlation between cholesterol reduction and a reduction in the risk of coronary heart disease.

More specifically, the double-blind placebo-controlled study dealt with 3,806 hypercholesterolemic men at high risk of developing coronary heart disease. The men who were actively treated achieved a 19% reduction in coronary heart disease risk as a direct result of an 8.5% reduction in plasma cholesterol. Thus, on the average, every 1% decline in plasma cholesterol yielded a 2% reduction in the risk of coronary heart disease (11).

Before initiation of the study, all potential participants were placed on a three-month diet that contained about 400 mg of cholesterol per day and that was designed to provide a polyunsaturated-to-saturated fatty acid ratio of approximately 0.8. This diet resulted in a 3.5% reduction in total plasma cholesterol and a 4.0% reduction in LDL-cholesterol, the lipoprotein-cholesterol complex associated with increased risk of coronary heart disease (12). NHLBI concluded that the study's findings, "taken in conjunction with the large volume of evidence relating diet, plasma cholesterol levels and coronary heart disease support the view that cholesterol lowering by diet also would be beneficial" (11).

The study, however, followed men with no previous evidence of coronary heart disease. Consequently, to learn if the actual atherosclerosis disease process could be reversed or slowed by a concomitant reduction in blood cholesterol levels, scientists from the University of Southern California's Atherosclerosis Research Institute performed a two-year trial known as the Cholesterol Lowering Atherosclerosis Study.
This study enrolled 162 nonsmoking men with normal blood pressures who had previously undergone coronary artery bypass graft surgery and who were clinically stable. These subjects were randomly assigned either to a cholesterol-lowering drug intervention group (whose members received colestipol hydrochloride plus niacin) or to a placebo group. Each group was given a cholesterol-lowering diet. The placebo group’s diet included less than 250 mg of cholesterol per day and provided 26% of the subject’s total energy intake in the form of fat (10% as polyunsaturated fat, 5% as saturated fat, and 11% as monounsaturated fat). The drug group had a much stricter diet, one allowing only half the placebo group’s daily cholesterol intake. Each subject was also given an angiogram (arterial X-ray) of his coronary, carotid, and femoral arteries and his coronary bypass grafts, both before and after two years of study participation (13).

The before-and-after results in the diet-only (placebo) group were statistically significant: Decreases were seen in the levels of total cholesterol (4%), triglyceride (5%), LDL-cholesterol (5%), and the ratio of LDL-cholesterol to HDL-cholesterol (6%). More dramatic decreases were seen in the drug intervention group’s observed levels—except in the level of the HDL-cholesterol complex, which increased by 37% (13).

Sixty-one percent of the men in the drug intervention group showed either no change or improvement of their atherosclerotic status (16.2% actually improved). Along these same lines, 39% of those in the placebo group showed either no change or improvement (2.4% actually improved). The results of this study demonstrated for the first time that nonsmoking men with normal blood pressures and prior coronary bypass surgery could actually reverse the atherosclerotic disease process through significant reductions in blood cholesterol levels (13).

More generally, two relationships have emerged from these and other clinical studies involving reduced blood cholesterol levels. First, the NHLBI-sponsored study discussed above showed that every 1% reduction in plasma cholesterol yielded an average 2% reduction in the risk of coronary heart disease (11). And second, the Multiple Risk Factor Intervention Trial (MRFIT), another very large study sponsored by the National Institutes of Health, data from which were applied to the Framingham Heart Study equation, showed that every 1 mg/dl reduction in serum cholesterol yielded an average 1% decrease in coronary heart disease mortality over a six-year period (14). Figure 2 shows the serum

![Figure 2](image)

Figure 2. The relationship between serum cholesterol and death from coronary heart disease in 361,662 men 35–57 years of age.

cholesterol-mortality relationship found in this study.

CURRENT CHOLESTEROL RECOMMENDATIONS

The most current U.S. cholesterol treatment recommendations for adults were published in 1988 in a report of the National Cholesterol Education Program Adult Treatment Panel (15). This panel had been convened by the NHLBI to update and build upon previous recommendations of expert committees, including its own 1984 NIH Consensus Conference on Lowering Blood Cholesterol to Prevent Heart Disease.

The panel’s report (15) does not distinguish between the sexes or between various age groups. Instead it presents two classifications for people 20 years of age and older; these are as follows:

**Initial Classification Based on Total Cholesterol**

- **< 200 mg/dl** Desirable blood cholesterol
- **200-239 mg/dl** Borderline-high blood cholesterol
- **≥ 240 mg/dl** High blood cholesterol

**Classification Based on LDL-Cholesterol**

- **< 130 mg/dl** Desirable LDL-cholesterol
- **130-159 mg/dl** Borderline-high-risk LDL-cholesterol
- **≥ 160 mg/dl** High-risk LDL-cholesterol

Other recommendations and findings of the panel include the following:

- Dietary treatment is the cornerstone of therapy to reduce blood cholesterol levels. The view that diet modification is impractical or doomed to failure is not justified. For many high-risk patients, the goals of cholesterol lowering can be achieved by dietary therapy alone.
- A cholesterol-lowering diet can be tasty, satisfying, and consistent with good nutrition. Many patients will not need to alter their eating habits radically.
- Step One of dietary treatment calls for a saturated fat intake accounting for less than 10% of total caloric intake, a total fat intake accounting for less than 30% of total caloric intake, and a dietary cholesterol intake of less than 300 mg/day. Step Two calls for further reductions lowering saturated fat intake to less than 7% of total caloric intake and reducing cholesterol intake to less than 200 mg/day (see Table 1).
- For most patients, dietary therapy should be continued at least six months before deciding whether to add drug treatment. It is important that dietary therapy not be regarded as a failure prematurely.
- Although the goal is to lower the LDL-cholesterol concentration, for convenience total cholesterol can be used to monitor the body’s response to dietary therapy.

With respect to coronary heart disease (CHD) risk factors other than LDL-cholesterol, the panel listed those shown in Table 2.

**EFFECTS ON PLASMA CHOLESTEROL LEVELS**

Reduction of blood cholesterol levels results from lowered dietary intakes of
Table 1. Dietary therapy for high blood cholesterol.

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Step 1 diet</th>
<th>Step 2 diet</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total fat</strong></td>
<td>Less than 30% of total calories</td>
<td>Less than 30% of total calories</td>
</tr>
<tr>
<td>Saturated fatty acids</td>
<td>Less than 10% of total calories</td>
<td>Less than 7% of total calories</td>
</tr>
<tr>
<td>Polyunsaturated fatty acids</td>
<td>Up to 10% of total calories</td>
<td>Up to 10% of total calories</td>
</tr>
<tr>
<td>Monounsaturated fatty acids</td>
<td>10–15% of total calories</td>
<td>10–15% of total calories</td>
</tr>
<tr>
<td><strong>Carbohydrates</strong></td>
<td>50–60% of total calories</td>
<td>50–60% of total calories</td>
</tr>
<tr>
<td><strong>Protein</strong></td>
<td>10–20% of total calories</td>
<td>10–20% of total calories</td>
</tr>
<tr>
<td>Cholesterol</td>
<td>Less than 300 mg/day</td>
<td>Less than 200 mg/day</td>
</tr>
<tr>
<td>Total calories</td>
<td>To achieve and maintain desirable weight</td>
<td>To achieve and maintain desirable weight</td>
</tr>
</tbody>
</table>

Source: United States, National Institutes of Health, National Heart, Lung, and Blood Institute (15).

Saturated fat and cholesterol. When dietary cholesterol is reduced, lowered blood levels usually will reflect this decrease.

Dietary intake of saturated fatty acids (SFA) has the most potent effect upon blood cholesterol levels, specifically LDL-cholesterol levels. Elevated SFA intake down-regulates the LDL receptor activity and also increases the rate of LDL synthesis (1, 2, 5, 16). Likewise, a positive energy balance that leads to obesity causes an overproduction of LDL and an increase in the rate of cholesterol synthesis, resulting in an exaggerated increase in blood cholesterol and LDL-cholesterol levels (1, 2, 5, 16).

When blood cholesterol levels are elevated in the mild to moderate range, dietary excesses are suspected. More severe elevations are attributed to a genetic component (3, 5). When, for example, a group of people in one geographic area consumes a fairly uniform diet, and yet wide variations in plasma cholesterol levels exist, inherited differences in LDL receptor activity are considered a major cause. Nevertheless, even when a ge-

Table 2. Risk status based on the presence of coronary heart disease (CHD) risk factors other than LDL-cholesterol.

The patient is considered to have a high-risk status if he or she has one of the following:

- Definite CHD, as indicated by the characteristic clinical picture and objective laboratory findings of either:
  - Definite prior myocardial infarction, or
  - Definite myocardial ischemia, such as angina pectoris
- Two other CHD risk factors:
  - Male sex
  - Family history of premature CHD (definite myocardial infarction or sudden death before 55 years of age in a parent or sibling)
  - Cigarette smoking (currently smokes more than 10 cigarettes per day)
  - Hypertension
  - Low HDL-cholesterol concentration (below 35 mg/dl, confirmed by repeated measurement)
  - Diabetes mellitus
  - History of definite cerebrovascular or occlusive peripheral vascular disease
- Severe obesity (≥ 30% overweight)

* Maleness is considered a risk factor in this scheme because the rates of CHD are three to four times higher in men than in women in the middle decades of life and roughly two times higher in the elderly. Hence, a man with one other CHD risk factor is considered to have a high-risk status, whereas a woman is not so considered unless she has two other CHD risk factors.

Source: United States, National Institutes of Health, National Heart, Lung, and Blood Institute (15).
netic basis exists for elevated cholesterol levels, most individuals respond to dietary changes (1-3, 15, 16).

Several prediction formulas have been developed in the United States that estimate the serum cholesterol level based on dietary intakes of polyunsaturated fatty acids (PUFAs), SFAs, and cholesterol. These formulas, developed for an average population of middle-aged North American males, illustrate numerically the profound influence of SFAs on blood cholesterol levels (18). Dietary PUFAs reduce serum cholesterol levels by half the amount that SFAs increase them. Dietary cholesterol makes a much less significant contribution to serum cholesterol levels, as indicated by Formula 2, where the effect of dietary cholesterol intake varies only with the square root of that intake.

The most commonly used formula, known as a modified Keys formula or the Keys/Anderson formula, is used when one knows the percentage of total daily caloric intake accounted for by lipids. This formula is

1. PSC (predicted serum cholesterol) 
   \[
   PSC = \phi + 164, \text{ and }
   \]

2. \[\phi = 1.26(2S - P) + 1.50Z; \text{ so } PSC = 1.26(2S - P) + 1.50Z + 164\]

where:

- \( \phi \) = a measure of the serum cholesterol elevating ability of the average individual's dietary pattern;
- \( S \) = saturated fatty acids expressed as a percentage of total daily caloric intake; only those acids with carbon chain lengths of 12, 14, and 16 are included;
- \( P \) = total polyunsaturated fatty acids expressed as a percentage of total daily caloric intake; and
- \( Z \) = the square root of cholesterol intake expressed as milligrams per 1,000 kcal of daily caloric intake.

Regarding the coefficient 1.26 in equation 2, another coefficient (1.35) had previously been used. However, this included stearic acid and fatty acids with fewer than 12 carbon atoms. It now appears that only saturated fatty acids with 12, 14, and 16 carbon atoms affect serum cholesterol levels. To date, the reason why stearic acid does not behave like lauric, myristic, and palmitic acids is unknown. However, stearic acid does increase plasma triglyceride levels (18).

Another form of the Keys/Anderson formula may be used when the daily intakes in grams of saturated and polyunsaturated fatty acids are known, rather than the proportional share of these lipids in total caloric intake. This formula, which is typically more convenient to use, is

\[
\phi = \frac{1.134(2Sg - Pg)}{\Sigma E} = 1.5Z
\]

where:

- \( Sg \) = total daily intake of saturated fatty acids in grams;
- \( Pg \) = total daily intake of polyunsaturated fatty acids in grams;
- \( \Sigma E \) = total daily kcal intake; and
- \( Z \) = the square root of cholesterol intake in mg per 1,000 kcal of daily caloric intake.

It should be noted that \( Z \) can be expressed as

\[
Z = \sqrt{\frac{1,000 \Sigma \text{Chol}}{\Sigma E}}
\]

where \( \Sigma \text{Chol} \) = daily dietary cholesterol intake in mg and \( \Sigma E \) = total daily kcal intake.

By knowing the relationships shown in these equations, one can predict how dietary alterations such as decreases in SFAs and increases in PUFAs can be expected to affect the blood cholesterol level.
Another formula, the Hegsted formula, compares two diets when the dietary percentages of saturated and polyunsaturated fat calories are known (19, 20). This formula, which can be used to predict likely changes in the serum cholesterol level resulting from changes in dietary lipid intake, is as follows:

\[
\text{delta Chol} = 2.16 \text{delta S} - 1.65 \text{delta P} + 0.0677 \text{delta C} - 0.5
\]

where:
- \( \text{delta Chol} \) = the change in the serum cholesterol level;
- \( \text{delta S} \) = the difference in the percentage of calories provided by saturated fatty acids in each of the two diets;
- \( \text{delta P} \) = the difference in the percentage of calories provided by polyunsaturated fatty acids in each of the two diets; and
- \( \text{delta C} \) = the difference in dietary cholesterol intake in decigrams (dg) per day.

This formula is best used when dietary cholesterol is not the major variable. Otherwise, it grossly overpredicts the change in serum cholesterol (19). In addition, one can also predict how a reduction in total daily calories is likely to affect the blood cholesterol level. The prediction depends upon the following relationship: For each 1 unit reduction in the \( \phi \) score of the habitual dietary pattern, assuming this is sustained for a period of two to four weeks, the serum cholesterol level can be expected to fall by 1 mg/dl (18).

It has been estimated that if a diet high in cholesterol, such as one with an intake of 300 mg/1,000 kcal per day, is altered so that the cholesterol intake is reduced by 50% without making any dietary change in fat or in total calories, it can be expected that the blood cholesterol level would fall by about 7.6 mg/dl.

Similarly, if a diet where 18% of the total calories come from saturated fat is changed to reduce the saturated fat intake by 50%—replacing the lost calories with simple carbohydrates so that the total dietary calories are kept constant—then the expected decrease in the blood cholesterol level should be about 23 mg/dl, or about three times that obtained by the dietary cholesterol reduction. As this suggests, when both effects are combined, an impressive expected reduction of about 30 mg/dl in the blood cholesterol level is obtained (19). It must be remembered, however, that these values come from group data, and that any one individual’s values may vary.

SATURATED AND UNSATURATED FATTY ACIDS

As previously mentioned, and as the Keys equations indicate, dietary saturated fatty acids are the leading determinant of the blood cholesterol level. Recall that the SFA/PUFA relationship is \( 2S - P \), where \( S \) and \( P \) equal the daily intake of saturated and polyunsaturated fatty acids. For a reference man maintaining a constant weight with an intake of 2,430 kcal per day, a positive \( 2S - P \) value represents the amount by which his serum cholesterol level would be raised by the abundance of saturated fatty acids. Likewise, a negative \( 2S - P \) value represents the amount by which his serum cholesterol level would be reduced. Theoretically, then, a PUFA intake twice as high as the saturated fatty acid intake would reduce the expression \( 2S - P \) to zero and would leave the blood cholesterol level unaffected by fatty acid intake (22). (Such a PUFA increase in the diet is not recommended, and is also impractical.)

The way in which the most common dietary PUFA, linoleic acid, lowers serum cholesterol has yet to be determined. One suggestion is that it has little or no effect on the LDL receptor and that it...
lowers LDL-cholesterol by substituting for saturated fatty acid intake. Other studies dispute this finding and claim that it does have its own independent effect. It has also been found that besides lowering LDL-cholesterol, linoleic acid lowers HDL-cholesterol (1, 3, 5, 8, 15).

Linoleic acid belongs to the omega-6 class of PUFAs. (The other class of PUFAs is omega-3, the omega number indicating the number of carbon atoms between the first double bond and the methyl end of the fatty acid.)

PUFAs currently account for about 6% of the calories in the U.S. diet, and many groups now recommend that it should be increased to 10% (1, 2, 4, 9, 10, 15).

Previously, linoleic acid had been enthusiastically recommended as a cholesterol lowering agent. This PUFA is found abundantly in several vegetable oils—notably safflower, sunflower seed, soybean, and corn oil. These oils, while high in PUFAs and low in saturated fatty acids, are also very rich in total calories. Increasing their share in the diet requires a compensatory decrease elsewhere; otherwise, the resulting weight gain would facilitate a rise in blood cholesterol. In this regard, the recommendation is that the increased PUFAs replace some of the saturated fats.

More recently, however, concern has been expressed about the long-term effects of linoleic acid. No population group appears to have consumed more than 10% of its calories in the form of linoleic acid over a long time period, and the literature on linoleic acid has reported potential problems (1, 14, 16). For example, it has been reported that linoleic acid is carcinogenic in laboratory animals, that it may increase one’s risk of gallstones if used in large amounts, and that it may suppress the immune system. Also, ingestion of linoleic acid in large quantities is known to alter the cell membranes. Therefore, because we do not yet know all the possible consequences resulting from long-term, high-dose consumption of this most common dietary PUFA, most experts feel justified in recommending that the intake of PUFAs not exceed 10% of the daily caloric intake.

The other class of PUFAs, the omega-3 fatty acids, are found abundantly in fish oils. The two most common of these highly polyunsaturated fatty acids in the diet are eicosapentaenoic acid and docosahexaenoic acid.

Recent claims that omega-3 oils are useful in the prevention of coronary heart disease appear premature. Actually, their value in reducing total serum cholesterol levels is unpredictable, contrary to claims now being made in the promotion of commercial supplements. Several studies indicate that the omega-3 oils may not lower LDL-cholesterol any more than the omega-6 PUFAs, and also indicate that they appear to lower protective HDL-cholesterol levels. Other studies have found them to raise LDL-cholesterol levels (1, 15). By inhibiting the synthesis of very low-density lipoproteins (VLDL), omega-3 oils do reduce VLDL levels, and they are effective in reducing plasma triglyceride levels.

Omega-3 oils also appear to have an inhibitory effect on platelet aggregation and stickiness, thereby delaying clotting time and the potential for thrombosis (1, 15). This, in fact, may be their most important feature in protecting against coronary heart disease. Fish oils may also have anti-inflammatory properties that tend to protect blood vessel walls against lipid deposition damage (5).

Large long-term intakes of the omega-3 fatty acids have yet to be evaluated for adverse effects on various bodily systems. Therefore, organizations providing expert advice do not recommend that the general public use high-dose supplements of fish oils to lower cholesterol levels or protect against coronary heart dis-
ease. One concern is that large doses of omega-3 oils might cause hemorrhagic complications—especially in persons using large doses of aspirin or other anticoagulants.

However, public consumption of fresh fish should be encouraged because fish is an excellent substitute for meat. Even though some fish contain almost as much dietary cholesterol as many cuts of meat, fish are very low in saturated fatty acids. Also, population studies indicate that in those geographic areas where fresh fish consumption is very high, the risk of coronary heart disease is greatly reduced.

Regarding monounsaturated fatty acids, the primary monounsaturated fatty acid in the U.S. diet is oleic acid. This acid is found primarily in olive oil, rape-seed oil (canola oil), and peanut oil. Monounsaturates had long been thought "neutral"—neither raising nor lowering blood cholesterol levels. More recent evidence indicates that when oleic acid is substituted for saturated fatty acids in the diet, it produces the same degree of LDL-cholesterol reduction as linoleic acid. However, oleic acid leaves the HDL-cholesterol level unchanged, whereas linoleic acid reduces it—at least initially (5, 15).

Population studies have shown that in Mediterranean areas such as Greece and southern Italy, where olive oil intakes are quite high, the incidence of coronary heart disease is relatively low. Of course, it may be that the high oleic acid intakes are simply substituting for saturated fatty acids. Experts currently recommend that monounsaturated fatty acids, mainly oleic acid, should account for about 10-15% of total daily calories (15).

OTHER CONSIDERATIONS

Mention should also be made of other dietary considerations. Obesity has been cited as an independent factor in hyperlipidemia, and weight loss is one of the most effective techniques for reducing blood cholesterol levels in overweight individuals. Weight reduction slows down rapid rates of cholesterol and lipoprotein synthesis; and in many people it lowers LDL-cholesterol, raises HDL-cholesterol, and reduces plasma triglycerides. In some extremely sensitive individuals, high-risk LDL-cholesterol levels will return to normal values merely with a reduced caloric intake, weight reduction, and then weight maintenance at a desired body weight. Therefore, caloric restriction and maintenance of desirable weight is especially recommended for overweight individuals at high risk (1, 15, 22).

Grundy (1) has called attention to other high-risk mechanisms not yet fully identified that alter cholesterol and lipoprotein metabolism but that are not reflected in the measurements of plasma cholesterol levels. Following ingestion of dietary cholesterol, for example, the cholesterol is transported in the gut by chylomicrons—another type of lipoprotein. After the triglycerides are split off from the chylomicrons, cholesterol-rich "chylomicron remnants" remain. These particles, along with other postprandial lipoproteins (beta-VLDL apolipoprotein A—similar to LDL), are all suspected of being atherogenic. Also, a high dietary fat intake may promote cholesterol absorption, cholesterol synthesis, and the growth of atherosclerotic lesions. It may also increase blood pressure and shift platelet metabolism toward the clotting portion of the equation (1, 2).

An additional concern about fat consumption has surfaced in recent years. Epidemiologic studies have repeatedly shown an association between dietary fat and the occurrence of cancer at several sites. While the data are not entirely consistent, and hence the relationship between dietary fat and cancer is not clear,
the U.S. National Academy of Sciences' Committee on Diet, Nutrition, and Cancer recommends that consumption of both saturated and unsaturated fats in the average U.S. diet be reduced (23).

CONCLUSION

In sum, we now have strong, consistent data from biochemical, clinical, genetic, pathologic, experimental, dietary, and epidemiologic studies, as well as from clinical trials, that conclusively demonstrate the roles of blood cholesterol, its lipoprotein carriers, and the carrier receptors' availability in causing atherosclerosis and coronary heart disease. Beyond a doubt, the collective evidence shows that by reducing elevated levels of total cholesterol and LDL-cholesterol, the risk of both fatal and nonfatal myocardial infarction is reduced. Therefore, vigorous treatment through dietary adjustment, weight reduction, and drug administration (if necessary) is recommended for individuals of all ages whose cholesterol levels are elevated. It has also been recommended that everyone receiving enough food (except children less than two years old) should limit their intake of total and saturated fat and cholesterol and slightly increase their polyunsaturated fat intake in order to lower blood cholesterol levels. Wherever food intake levels are adequate, the health of the general population will benefit markedly from a prudent eating pattern of this kind.

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