



CHANGING PERSPECTIVES ON FAT, SATURATED FAT & HEART DISEASE

SUMMARY

A multitude of genetic and environmental factors influence the risk of coronary heart disease (CHD), a leading cause of death and disability in the U.S. Lifestyle modifications, particularly dietary changes, are the cornerstone of CHD prevention. Major diet and health reports released during the past several decades have advised the healthy American adult population to consume a diet containing 30% or less of calories as fat. However, more recent guidelines recommend a range of total fat intake of 20% to 35%. This increased flexibility in dietary fat recommendations is due in part to recognition that very low fat diets (<20% of calories) may lead to nutrient shortcomings and potentially be harmful with respect to CHD risk, for example, by decreasing high density lipoprotein (HDL) cholesterol (i.e., the "good" cholesterol) and increasing blood triglyceride levels.

Dietary guidelines have consistently advised healthy American adults to reduce saturated fat intake to 10% or less of calories. However, some recent advice calls for reducing saturated fat intakes to as low as possible while consuming a nutritionally adequate diet. Yet, the level to which saturated fat intake can be reduced without potential adverse health effects remains to be established.

Not all saturated fats increase blood cholesterol levels and saturated fats may increase some markers associated with reduced risk of CHD.

Numerous studies have demonstrated that restricting total and saturated fat is often accompanied by an increase in dietary carbohydrate, which reduces low

density lipoprotein (LDL) cholesterol (the "bad" cholesterol), but also decreases HDL cholesterol, increases triglyceride levels, and may lead to a LDL subclass distribution associated with increased risk of CHD.

Individuals vary greatly in their blood lipid responses to dietary changes in fat and saturated fat, and for some individuals reducing dietary fat/saturated fat may not be beneficial. Some of these differences may be explained by diet-gene interactions. Intake of a low fat, high carbohydrate diet results in a greater reduction in LDL cholesterol in a minority of healthy normolipidemic individuals with a predominance of small, dense LDL cholesterol (subclass pattern B which is associated with increased risk of CHD) than in the majority of those with large, buoyant LDL particles (subclass pattern A). Moreover, a substantial proportion of pattern A individuals respond to a low fat/low saturated fat diet with a shift from larger to smaller LDL and increased expression of atherogenic phenotype B.

An overzealous attempt to reduce total and saturated fat may lead some individuals to eliminate nutrient dense foods containing these nutrients. For example, omitting dairy foods can lead to shortcomings in several essential nutrients. Also, the fat in dairy foods contains components (e.g., conjugated linoleic acid) which have been demonstrated to have beneficial health effects. Further, intake of other nutrients (e.g., calcium, potassium, magnesium) in dairy foods may help reduce CHD risk.

With evolving knowledge of the role of total and saturated fat in CHD risk and of the genetic basis for individual responses to dietary changes, recommendations for dietary total and saturated fat can be expected to become more individualized. **D**

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INTRODUCTION

Coronary heart disease (CHD) is a leading cause of death and disability in the United States (1). Major modifiable risk factors for this disease include high blood pressure, tobacco smoking, high blood total cholesterol, elevated low density lipoprotein (LDL) cholesterol, low high density lipoprotein (HDL) cholesterol, physical inactivity, obesity/overweight, and type 2 diabetes mellitus (2,3). Non-modifiable risk factors include advancing age, race (e.g., African American), and genetics. Lifestyle modifications, particularly dietary changes, are the cornerstone of CHD prevention. Dietary recommendations for reducing CHD risk focus on decreasing total and saturated fat, primarily because of their ability to increase blood total and LDL cholesterol levels (2,3). However, some scientists question whether everyone should restrict their intake of total and saturated fat to lower their risk of CHD and, specifically, whether there is a beneficial role for a reasonable proportion of saturated fat in the overall diet (4-7).

Traditionally, the effects of dietary fats on CHD risk have been estimated from their impact on blood total and LDL cholesterol levels. However, fats also affect HDL cholesterol and LDL and HDL subclasses with varying atherogenic potential. The effects of decreasing total and saturated fat on these markers of CHD risk, as well as the differential effects of dietary fat/saturated fat in subgroups of the population and in individuals with a specific genetic make-up, justify some caution in the widespread application of low fat/low saturated fat diets to reduce the risk of CHD. Although the appropriate amount of dietary fat, and in particular saturated fat, for optimal health is unknown, there is increasing support for tailoring dietary fat recommendations to individuals to prevent CHD (2,7,8).

This *Digest* reviews dietary recommendations for total fat and saturated fat to reduce CHD risk; the effects of total and saturated fats on blood lipoprotein levels; differing effects of lowering total and saturated fat in subgroups of the population and in individuals with specific genetic profiles; and the importance of moderating,

not eliminating, saturated fat or foods containing saturated fat to reduce CHD risk and improve overall health.

DIETARY FAT/SATURATED FAT RECOMMENDATIONS TO REDUCE THE RISK OF CORONARY HEART DISEASE

Major diet and health reports released during the past several decades have identified excessive dietary fat, and particularly saturated fat, as a prime cause of diet-related chronic diseases such as CHD (2,3,9-14). Most of these public health recommendations for U.S. adults advise a reduction in total fat intake to 30% or less of calories to reduce the incidence of CHD. The National Cholesterol Education Program (NCEP) Step 1 diet for the general healthy population recommends restriction of total fat to 30% or less of calories (3). Likewise, the American Heart Association (AHA) advises a reduction in fat intake to 30% or less of total calories (2). However, recognition that very low fat diets (<20% of calories) may lead to nutrient shortcomings and potentially be harmful to cardiovascular health (i.e., by decreasing HDL cholesterol and increasing blood triglyceride levels) (2,14,15) has contributed to a relaxation of dietary fat recommendations. Recent dietary fat recommendations, such as those issued by the Institute of Medicine (IOM), in its *Dietary Reference Intakes for Macronutrients* (12), and the federal government's 2005 *Dietary Guidelines for Americans* (13,14) advise a range of total fat intake of 20% to 35% of calories, as opposed to a specific minimum, for all Americans age 18 years and over.

Beginning with the U.S. Senate's *Dietary Goals for the U.S.* in 1977 (9), dietary recommendations have consistently advised healthy American adults to reduce their saturated fat intake to 10% or less of calories (2,3,10-14). For adults with elevated LDL cholesterol levels or with cardiovascular disease, a lower intake of saturated fat (i.e., <7% of calories) is recommended. The IOM's report (12) recommends that intake of saturated fat be as low as possible while consuming a nutritionally adequate diet. However, the lower safe level of saturated fat or specific saturated fatty acids remains to be established (4). The IOM's report adds

Some questions are being raised regarding whether everyone should reduce their intake of total and saturated fat to lower their risk of CHD.



that “it is neither possible nor advisable to achieve 0 percent of energy from saturated fatty acids in typical whole-food diets” and that doing so could lead to “undesirable effects (e.g., inadequate intake of protein and certain micronutrients) and unknown and unquantifiable health risks” (12). As discussed below, the possibility that a certain amount of dietary saturated fatty acids or specific saturated fatty acids may be beneficial for cardiovascular health cannot be ignored (4). Also, accumulating evidence indicates that individuals vary in their blood cholesterol response to dietary changes in total and saturated fat intake (2,7,16).

EFFECTS OF TOTAL AND SATURATED FAT ON BLOOD CHOLESTEROL AND LIPOPROTEIN LEVELS

According to the lipid or diet-heart hypothesis of CHD, dietary total fat, saturated fat, and cholesterol increase the level of blood cholesterol, which is a risk factor for CHD (17). However, as reviewed by several authors, evidence supporting the lipid hypothesis is inconclusive or contradictory (4,18-22). The failure of some studies to show that decreasing total fat, and in particular saturated fat, lowers risk of CHD may be explained by several environmental and genetic factors, including differences in the blood cholesterol-raising effect of individual saturated fatty acids and the heterogeneous nature of cholesterol-carrying lipoproteins. The effect of consuming saturated fat on CHD risk has been narrowly focused on its ability to elevate LDL cholesterol (4). However, saturated fat may influence other lipoproteins (e.g., HDL cholesterol) associated with reduced risk of CHD.

Differing Cholesterolemic Effects of Individual Saturated Fatty Acids.

Not all saturated fatty acids have the same effect on blood cholesterol levels (2,12,23,24). Stearic acid (C18:0) has a neutral effect on blood cholesterol levels, whereas other long chain saturated fatty acids such as lauric (C12:0), myristic (C14:0), and palmitic (C16:0) acids increase blood cholesterol levels. Stearic acid may influence CHD risk independently of an effect on blood cholesterol levels (25). Compared to a palmitic acid-rich diet, a stearic acid-rich

diet has been shown to have beneficial effects on thrombogenic and atherogenic risk factors (25).

Effects of Dietary Fat on Blood Lipoprotein Levels.

Numerous studies demonstrate that restricting total and saturated fat is often accompanied by an increase in dietary carbohydrate which can lead to a blood lipid profile associated with increased risk of CHD (4,7,8,13,18,26-29). A low fat, high carbohydrate diet reduces LDL cholesterol, but also decreases HDL cholesterol and ApoA1 (the major transport protein for HDL) and increases blood triglyceride levels. Low blood HDL and ApoA1 levels and elevated triglyceride levels are associated with increased risk of CHD (7). Increasing HDL cholesterol levels helps to protect against the development of CHD and possibly stroke (30). A study in healthy adults who consumed either a low fat (25% of energy), high carbohydrate (60% of energy) diet or a high fat (45% of energy), low carbohydrate (40% of energy) diet in random order for two weeks found that those on the low fat, high carbohydrate diet had lower HDL cholesterol levels, higher blood triglyceride levels, and a persistent increase in remnant lipoproteins (28)

Evidence also indicates that saturated fats raise HDL cholesterol levels (4,31,32). Moreover, individual saturated fatty acids differ in their effects on HDL cholesterol levels (24,31). A study in young men found that HDL cholesterol levels were higher after intake of myristic acid than after intake of stearic acid (31). According to a meta-analysis of 35 studies, stearic acid has a minimal effect on LDL cholesterol and no effect on HDL cholesterol, whereas other long chain saturated fatty acids such as lauric acid increase both LDL and HDL cholesterol levels (24). As one author stated, “saturated fats cannot be quite so evil because, while they elevate LDL, which is bad, they also elevate HDL, which is good” (20). Other researchers suggest that the increase in HDL cholesterol from a diet high in saturated fat compensates for the adverse effects of saturated fatty acids on LDL (33).

Effect of Dietary Fat on

Lipoprotein Subclasses. Scientists have discovered that the atherogenicity of LDL and HDL cholesterol varies with their particle size and density (7,34-37). That is,

Saturated fat increases LDL (the “bad”) cholesterol, but it also increases HDL (the “good”) cholesterol and may lead to a LDL subclass distribution associated with a lower risk of CHD.

not all LDL cholesterol is equally “bad,” nor is all “HDL” cholesterol good. To-date, seven different LDL subclasses and five different HDL subclasses based on their particle size and density have been identified (34). In the case of LDL cholesterol, a predominance of small, dense particles (so-called LDL subclass pattern B) is associated with increased risk of CHD, whereas large, buoyant LDL particles (so-called LDL subclass pattern A) are associated with a lower prevalence of CHD (7,34-42). LDL subclass pattern B is found in approximately 30 to 35% of adult males, in 5% to 10% of males <20 years of age, and in 5% to 10% of premenopausal women (7). The majority of the population has the less atherogenic LDL subclass pattern A. Individuals with LDL pattern B tend to have low HDL cholesterol, high triglyceride, higher blood sugar levels, high blood pressure, and obesity, a constellation of disorders known as metabolic syndrome and a profile associated with a two-to three-fold increase in CHD (7).

Genetic factors explain up to half of the variation in LDL size distribution, with environmental factors such as adiposity and diet explaining the rest (7). Studies show that diets high in saturated fat are associated with an increase in larger, less atherogenic, LDL particles (42,43). In a cross-over study of high fat (46%) or low fat (24%) diets which examined LDL subclass responses to total and specific saturated fatty acids in 103 men, total saturated fat as well as myristic acid and palmitic acid were positively associated with more buoyant, larger LDL subclass particles (i.e., a less atherogenic profile) (43). Saturated fat intake also reduced the activity of hepatic lipase, an enzyme involved in the formation of small, dense LDL cholesterol (43).

A recent cross-sectional study in 291 healthy Swedish men, who were divided into three groups based on their proportion of small, dense LDLs, found that intakes of reported fat from dairy products and individual fatty acids in milk products were associated with significantly fewer small, dense LDL particles and a more favorable LDL profile (44).

Greater understanding of gene-nutrient interactions will allow better identification of individuals who may or may not benefit from reducing total and saturated fat intake.



In the case of HDL cholesterol, studies indicate that risk of CHD is reduced when larger HDL2 subfractions predominate over the smaller, more dense HDL3 subfractions (34,41). Dietary total and saturated fat can influence HDL particle size and density (43,45). Reducing dietary total and saturated fat intake has been shown to result in a pronounced decrease in the larger size HDL2 subfractions compared to the smaller sized HDL3 subfractions (i.e., a more atherogenic profile) (45).

INDIVIDUAL BLOOD LIPID RESPONSES TO DIETARY CHANGES IN TOTAL AND SATURATED FAT

Individuals vary greatly in their blood lipid response to changes in dietary fat and saturated fat (2,4,5,7,16,46,47). Dietary fat may have unique effects on lipoproteins and CHD risk in men versus women, in individuals with metabolic syndrome versus those without this disorder, and in individuals with genetically determined lipoprotein profiles (5,7,48-50).

In a recent study in postmenopausal women with CHD who consumed a total fat intake of 25% of energy, greater saturated fat intake was associated with a more favorable lipoprotein profile (i.e., higher HDL cholesterol, higher ApoA1, lower triglycerides, and a lower ratio of total cholesterol to HDL cholesterol) and less progression of coronary atherosclerosis over a three year period compared to a lower saturated fat intake (48). In contrast, intakes of polyunsaturated fat and carbohydrate were associated with increased progression of CHD (48). The associations were independent of other known risk factors for CHD.

The researchers suggest that their findings support other studies demonstrating that men and women differ in their response to dietary changes (4,48,49). LDL cholesterol is a stronger predictor of CHD in men, whereas non-LDL lipid variables such as triglyceride levels and HDL cholesterol abnormalities are stronger predictors of CHD in women (5,48). The beneficial

effect of saturated fat intake on HDL and triglyceride levels may be especially important for women after menopause when HDL cholesterol is lower and CHD risk is concomitantly higher (48). Also, the women in the study had risk factors consistent with the metabolic syndrome (i.e., diabetes, high blood pressure, overweight, low HDL cholesterol, higher triglycerides) (5). Thus, the effect of saturated fat intake on blood lipid levels and CHD risk may differ according to the presence or absence of metabolic syndrome. In obese, insulin resistant individuals, intake of saturated fat and cholesterol in the form of four eggs/day elevated LDL cholesterol less than in lean, insulin sensitive individuals (50).

Differences among individuals in their blood lipid responses to dietary fat/saturated fat may be explained by diet-gene interactions, such as those affecting LDL subclass distributions (e.g., pattern A or B) (51-54). When the effects of high fat (46% of energy) versus low fat (24% of energy) diets on blood lipoproteins were investigated in healthy normolipidemic men, only a minority of men with phenotype B on a high fat diet achieved a two-fold greater decrease in LDL cholesterol with dietary fat reduction than did men with phenotype A (7,53,54). In men with phenotype B, the reduction in LDL cholesterol was largely associated with a decrease in the number of LDL particles, which would be expected to reduce the risk of CHD (7). In contrast, in the majority of men with phenotype A on the high fat diet, reductions in dietary fat led to a decrease in the cholesterol content of LDL particles with minimal change in particle number, which would be expected to have little effect on CHD risk (7). Moreover, 41% of pattern A men responded to a low fat diet with a shift from larger to smaller LDL and expression of atherogenic phenotype B (7). With a further reduction in dietary fat to levels as low as 10% of energy, an increasing number of men with phenotype A have been shown to convert to phenotype B (55).

A potential genetic basis for the unique effects of low fat diets on LDL levels in pattern B subjects has been demonstrated in studies of



An overzealous attempt to reduce total and saturated fat may result in nutrient shortcomings and unintended consequences with respect to CHD risk and overall health.

premenopausal women (56) and children (57). Although multiple genes likely explain the differences in blood lipid responses to dietary changes, variants in the apolipoprotein E (apoE) genotype have been shown to interact with the quantity and quality of dietary fat to modify LDL subclass (7,23,58-60). The apoE4 allele is associated with a more atherogenic lipoprotein profile than apoE2 or apoE3. Some evidence indicates that apoE4 carriers (found in approximately 14% of the population) are more responsive to changes in dietary fat and cholesterol than carriers of apoE2 or apoE3 alleles (7,23,58). Identification of genes involved in blood lipid responses to dietary changes may lead to individualized dietary interventions to reduce CHD risk.

CONCLUSION

Much remains to be learned about the relationship between fat/saturated fat and CHD, and in particular how other factors (e.g., nutrients, genetics) may interact to increase or decrease CHD risk in populations or individuals.

Although health professionals recommend avoiding extremes in dietary fat intake (8), an overzealous attempt to reduce total and saturated fat may lead some individuals to eliminate nutrient dense foods containing these nutrients. Omitting specific food groups or specific foods from the diet can lead to shortcomings of essential nutrients (61). Individuals who prefer higher fat milks may switch to less nutritious beverages, thereby jeopardizing their intake of many essential nutrients in dairy foods. Data from the 1994-1996, 1998 Continuing Survey of Food Intakes by Individuals (62), and affirmed by the 2005 *Dietary Guidelines* (14), reveal that intake of total dairy and milk is associated with improved intakes of essential nutrients (e.g., calcium, magnesium, potassium, zinc, iron, vitamin A, riboflavin, and folate) without an adverse effect on dietary fat or cholesterol intake. Also, the fat in dairy foods contains components such as conjugated linoleic acid (CLA), sphingolipids, and butyric acid, which have been demonstrated to have health

benefits (4,18). For example, emerging scientific evidence indicates that CLA and its precursor vaccenic acid (a naturally occurring trans fatty acid) in milk fat may protect against the development and progression of atherosclerosis (18,63-66). Further, dairy foods contain other nutrients such as calcium, potassium, and magnesium which may help reduce the risk of CHD either directly or indirectly (67-70).

As knowledge of the role of total and saturated fat in CHD risk and overall health evolves and more information becomes available on how individuals differ in their dietary responses, dietary recommendations for total and saturated fat can be expected to become more targeted and individualized. In the meantime, moderating total fat intake and keeping intake of saturated fats (as well as trans fats and cholesterol) low by making smart choices from every food group is recommended for overall health, including protection from CHD (14). **D**

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