Stearic acid (systematic name, octadecanoic acid, \( \text{CH}_3(\text{CH}_2)_{16}\text{COOH} \)) is a long-chain fatty acid consisting of 18 carbon atoms without double bonds. Although it is classified as a saturated fatty acid (SFA), both biochemically and for purposes of nutrition labeling and dietary recommendations, data accumulated during the past 50 years indicate that stearic acid (C18:0) is unique among the SFAs in the food supply (1-4). Unlike other predominant long-chain SFAs – palmitic (C16:0), myristic (C14:0), and lauric (C12:0) acids - which increase blood cholesterol levels - stearic acid has been shown to have a neutral effect on blood total and low density lipoprotein (LDL) cholesterol levels (1-5).

Stearic acid’s neutral effect on blood total and LDL cholesterol levels implies that this long-chain SFA may not increase the risk for cardiovascular disease. For this reason, it has been suggested that stearic acid not be grouped with other long-chain SFAs, although to date this recommendation has not been implemented in dietary guidance or nutrition labeling.

**Intake and Sources**

Average intake of stearic acid is 5.7 g/day (8.1% of total fat) for women and 8.2 g/day (8.4% of total fat) for men, according to data from NHANES (National Health and Nutrition Examination Survey) 2001-2002 (6). For females and males aged 20 years of age and over, dietary stearic acid intake accounts for 25.7% of SFA intake (2.3% of total calories) and 17.9% of SFA (2.8% of total calories), respectively (6). Intake of stearic acid is preceded only by palmitic acid which accounts for 54.2% of SFAs (5.8% of total calories) for females and 54.5% of SFAs (6.0% of total calories) for males (6).

Major food sources of stearic acid for adults are meat/poultry/fish, grain products, and milk/milk products (1, Table 1). Fats rich in stearic acid include cocoa butter (typically consumed as chocolate), mutton tallow, beef tallow, lard, and butter (1,7). In meat (beef, pork, lamb, veal), stearic acid ranges from approximately 9% to 16% of total fat (7, Table 2). In lean ground beef (95% lean, 5% fat), stearic acid makes up 16% of total fat. A skinless, roasted chicken breast contains less stearic acid, with levels approximating 8% of total fat (7, Table 2).

When expressed as a percentage of beef’s total saturated fat, approximately one-third is stearic acid (7). A 100 g portion of cooked lean beef (composite, trimmed, retail cuts, all grades, 0” trim) contains 3.54 g saturated fat, of which 1.14 g or 31% is stearic acid (7, Table 2). In 95% lean ground beef, 37% of saturated fat is stearic acid (7, Table 2).
History

Knowledge of the effects of fatty acids on blood lipids in humans can be traced back to the early studies of Ahrens et al. (8), Keys et al. (9), Hegsted et al. (10,11), and Yu et al. (12). These investigators found that SFAs with chain lengths longer than 10 carbons generally raised blood cholesterol levels; polyunsaturated fatty acids (primarily linoleic acid) lowered blood cholesterol levels; and monounsaturated fatty acids (primarily oleic acid) had either a neutral or mildly hypocholesterolemic effect on blood cholesterol levels. These investigators also showed that the SFA stearic acid did not raise blood total or LDL cholesterol (i.e., the “bad” cholesterol) levels. Using data from studies in which adults were fed controlled, whole-food diets, Yu et al. (12) developed predictive equations to evaluate the effect of stearic acid on blood lipid levels. The analyses revealed that stearic acid, unlike the other long-chain SFAs, had no effect on blood total, LDL, and high density lipoprotein (HDL) (i.e., the “good cholesterol”) cholesterol levels in adults (12).

Despite subsequent findings supporting stearic acid’s neutral effect on blood total and LDL cholesterol levels (14,15), relative to lauric, myristic, and palmitic acids, stearic acid has been shown to exert a neutral or hypocholesterolemic effect on blood cholesterol levels in experimental animals (14,15).

Studies in humans also indicate that stearic acid is uniquely different from other long-chain SFAs and that it does not raise blood total and LDL cholesterol levels (1-3,5,13-19). A meta-analysis of 35 controlled trials found that when stearic acid replaced carbohydrate in the diet it had a neutral effect on blood lipid and lipoprotein levels (13). Researchers in the Netherlands reported that when intakes of stearic acid, oleic acid, or linoleic acid (7% of energy) were consumed by healthy adults for five weeks, there were no significant differences between the diets in serum lipid and lipoprotein levels (i.e., serum LDL and HDL cholesterol levels, very low density lipoprotein particle sizes, and lipoprotein subclasses) (20). Other studies reviewed by Kris-Etherton (1) reveal that lipid and lipoprotein levels are not affected when stearic acid is substituted for carbohydrate and oleic acid at 5% and 13% of energy intake, respectively. At higher stearic acid intakes, plasma total and LDL cholesterol levels tend to decrease when compared to baseline levels and diets in which palmitic acid is substituted for stearic acid (1). The effects of stearic acid on HDL cholesterol and triglyceride levels are inconsistent (1,5).

Animal and Human Studies

Animal studies demonstrate that individual SFAs differ in their effects on blood total and LDL cholesterol levels (14,15). Relative to lauric, myristic, and palmitic acids, stearic acid has been shown to exert a neutral or hypocholesterolemic effect on blood cholesterol levels in experimental animals (14,15).

Despite findings of stearic acid’s neutral effects on blood total and LDL cholesterol levels, stearic acid’s benefits with
respect to cardiovascular disease are unknown. However, findings that some foods such as chocolate and lean red meats do not increase the risk of cardiovascular disease despite their SFA content may be explained in part by their high levels of stearic acid (21-25). For example, the observation that lean red meat (beef) and lean white meat (chicken, fish) are equally effective in reducing total and LDL cholesterol in adults fed lipid-lowering diets (22-25) may be attributed in part to red meat’s higher content of stearic acid compared to that in chicken or fish.

**Possible Mechanisms**

Why stearic acid does not raise blood cholesterol levels has been the subject of considerable research and several possible explanations have been proposed (2,3,17). Although early investigations in experimental animals suggested that the absorption of stearic acid is less efficient than that for other SFAs such as lauric, myristic, and palmitic acids, subsequent studies in humans have shown that the absorption of stearic acid (94%) is only slightly lower than that of other SFAs (97% to > 99%) (2,3,26). Thus, reduced stearic acid absorption does not appear to explain differences in plasma lipid and lipoprotein responses to stearic acid compared to other saturated or unsaturated fatty acids. Another hypothesis is that stearic acid is rapidly converted in the body to the monounsaturated fatty acid, oleic acid, which does not affect blood cholesterol levels (2,3). However, in humans this conversion appears to be limited, as approximately 9% to 14% of dietary stearic acid is converted to oleic acid (2,27).

An alternative hypothesis to explain stearic acid’s neutral effect on blood LDL cholesterol is that stearic acid, unlike hypercholesterolemic SFAs, does not suppress LDL-receptor activity in the presence of dietary cholesterol (2). Another possible explanation is that dietary stearic acid reduces cholesterol absorption, perhaps by altering the synthesis of bile acids and consequently the solubility of cholesterol (28,29). Although this effect of stearic acid on cholesterol absorption has been shown in laboratory animals, it has yet to be demonstrated in humans. Clearly, the question of what biological mechanism(s) underlies stearic acid’s regulation of cholesterol metabolism remains to be resolved.

**Effects on Other Cardiovascular Disease Risk Factors**

Questions have arisen regarding the effects of stearic acid on other risk factors for cardiovascular disease such as thrombosis (blood clot), inflammation, and blood pressure (1). A heart attack (and stroke) is typically preceded by the formation of a blood clot that blocks the coronary artery. Early studies suggested that stearic acid adversely affects platelet function and blood clotting (2,3,30). However, more recent investigations in humans show that diets enriched in stearic acid have a neutral or beneficial effect on thrombotic tendency (1,17,18,31,32).

Researchers in Scotland found that when healthy young men consumed isocaloric diets containing 38% of calories from fat and rich in stearic acid, oleic acid, or linoleic acid, these fatty acids did not significantly influence blood coagulation and fibrinolysis (33). Other researchers have reported that diets enriched in stearic acid lower fasting factor VII coagulation (which would reduce thrombosis) compared with diets rich in other long-chain SFAs or monounsaturated fatty acids (16,31). A dietary intervention study in healthy young men in Denmark found that intake of a diet high in stearic acid for three weeks reduced fasting coagulation factor VII activity by 13% and 18% compared to a high palmitic acid diet or a diet high in myristic and lauric acids, respectively (31).

Researchers in Australia found that compared to a diet high in palmitic acid, a stearic acid-rich diet (~ 7% of calories or 19 g/day) had beneficial effects on several measures of thrombosis (e.g., platelet volume, platelet aggregation, coagulation factor VII activity) in healthy males (17). In this study, stearic acid was increased by 12 g/day using stearic acid-enriched margarine. When these same investigators used a lower amount of dietary stearic acid (i.e., an increase of 3 g/day using milk chocolate) in a similar study, no differences in platelet aggregation or platelet activation activity were found (18). However, there was a trend for mean platelet volume to decrease on the stearic acid-rich diet and increase on the palmitic acid-rich diet (18). Decreased mean platelet volume represents a shift towards smaller, less reactive platelets, which are thought to be less likely to contribute to thrombus formation. Other studies indicate that dietary stearic acid is not more thrombogenic compared to other saturated (palmitic, myristic, lauric acids) or unsaturated (oleic, linoleic) fatty acids (31,32). Evidence to date indicates that diets rich in stearic acid do not increase thrombosis. However, the specific effects of stearic acid on various measures of thrombosis appear to be influenced by the experimental conditions employed (e.g., dose and dietary source of stearic acid) (18).

With respect to inflammation, one study in adult male volunteers found that high levels of stearic acid (11% of total calories) increased plasma fibrinogen levels (a marker of inflammation), whereas markers of inflammation were not affected when stearic acid was consumed at a level of 7% of total calories, which is above the current intake of 3% of total calories (1,34). Diets containing stearic acid at levels of 8% to 13% of calories appear to have no effect on blood pressure (1). In addition to the level of intake of stearic acid, the positional distribution of stearic acid on the triglyceride molecule may influence its metabolic effects. Preliminary findings indicate that, compared to
naturally occurring stearic acid in foods such as beef (i.e., on the SN-1 and SN-3 positions), synthetic stearic acid (i.e., on the SN-2 position) may have adverse effects on blood lipid and glucose levels (35).

**Summary**
A common misperception is that all SFAs are alike in terms of their cardiovascular disease risk. However, research demonstrates that individual SFAs differ in their effects on blood cholesterol levels. In contrast to the predominant long-chain SFAs in the diet that raise blood total and LDL cholesterol levels (i.e., lauric, myristic, and palmitic acids), studies consistently show that stearic acid has a neutral effect on these lipid levels in humans. Less clear are the effects of stearic acid on thrombosis, inflammation, and blood pressure (1). However, there is no evidence that intakes of stearic acid equal to or slightly higher than amounts typically consumed have any adverse effects on these cardiovascular disease risk factors.

Approximately one-third of the total SFAs in beef is stearic acid. When this is taken into account, the amount of potentially hypercholesterolemic fatty acids is similar for beef, chicken, and fish (7). Beef’s relatively high content of stearic acid may help to explain research findings indicating that lean beef is just as effective as chicken or fish in reducing blood total and LDL cholesterol levels in adults fed low fat diets (22-25).

With increased understanding of the effects of dietary stearic acid on cardiovascular disease risk factors, future dietary recommendations and nutrition labels may be better defined to positively position this unique SFA with health professionals and ultimately the consumer. Separate consideration of stearic acid would place fewer restrictions on foods and allow for more flexibility in planning diets to reduce cardiovascular disease risk. Because there is no practical way to incorporate stearic acid’s neutral effect on blood lipid levels into dietary guidance, current dietary recommendations are for total SFAs without consideration of individual SFAs (27,36,37).
References


