Fatty Acids

Nomenclature

Fatty acids are organic acids characterized by long unbranched aliphatic tails (4-28 carbons) attached to a carboxyl group (Gunstone 1996; Lichtenstein 2005). Most naturally occurring fatty acids possess an even number of carbon atoms. The carbon chain may contain several unsaturated or exclusively saturated bonds. Thus, fatty acids differ in chain length, degree of saturation, location of the double bond(s) along the chain, and whether the orientation of hydrogen atoms adjacent to the double bond is *cis* or *trans*. Fatty acids with no double bonds are referred to as saturated fatty acids (SFA) while those with one double bond are monounsaturated fatty acids (MUFA). Fatty acids with two or more double bonds are termed polyunsaturated fatty acids (PUFA). PUFA are most often categorized into two groups distinguished by a difference in the location of the first double bond from the methyl end of the acyl chain, namely, n-6 and n-3. Linoleic acid (18:2n-6) and alpha-linolenic acid (18:3n-3) are representatives of these two groups. Each contains 18 carbon atoms but differs in the number of double bonds (18:**2**n-6 vs 18:**3**n-3) and the location of the first double bond (18:2**n-6** vs 18:3**n-3**). In this report, unless marked, all unsaturated fatty acids are assumed to be in the *cis* configuration.

Sources and Physiological Functions

Good sources of PUFA include salmon, tuna, mackerel, sardines, walnuts, sunflower, chia, and flax seeds. Avocados, almonds, pecans, and pumpkin seeds are good sources of MUFA (American Heart Association 2026). Healthy cooking oils rich in MUFA and PUFA include canola, corn, olive, peanut, safflower, soybean, sunflower, and vegetable (American Heart Association 2026). Higher amounts of SFA are commonly found in animal-sourced foods, such as various meats and dairy products, as well as coconut and palm oil (American Heart Association 2026).

Triacylglycerols (triglycerides), the basic building blocks of fats and oils, are made up of three fatty acids esterified to a glycerol molecule (Lichtenstein 2005). They are usually composed of 2–3 different kinds of fatty acids per molecule. During digestion, dietary triglycerides from animal and vegetable fats are hydrolyzed in the small intestine to release free fatty acids. These acids enter the intestinal cells and are used to resynthesize triglycerides, which become incorporated into large lipoprotein particles called chylomicrons; these in turn are released into the lymph prior to entering the plasma. Fatty acids with 10 or fewer carbon atoms can be absorbed from the gut directly into the bloodstream where they are bound to albumin in the plasma. At distal sites, triglycerides are again hydrolyzed by lipases before fatty acids can enter cells for further metabolism. Once inside peripheral cells, free fatty acids provide an immediate source of energy (fatty acids are a major fuel source for many tissues) or act as substrates for the biosynthesis of signaling molecules such as eicosanoids. Free fatty acids are also incorporated into other lipid classes, such as phospholipids, sphingolipids, and cholesteryl esters, or they may be resynthesized into triglycerides and stored for later use. Phospholipids, which are critical structural components of cellular membranes, tend to incorporate unsaturated fatty acids and so serve as a reservoir for MUFA and PUFA.

Humans are incapable of de novo synthesis of n-6 and n-3 PUFA because they lack the ability to insert a double bond any closer than 9 carbons from the methyl end. Thus, linoleic acid (18:2**n-6**) and alpha-linolenic acid (18:3**n-3**) are called "essential" PUFA in that they are required for good health but must be derived from food sources rather than through endogenous biosynthesis or metabolism. Both of these fatty acids are metabolized to longer-chain, more highly unsaturated forms. Note that SFA and MUFA in plasma are not expected to closely reflect dietary intake because these two classes of fatty acids can be endogenously synthesized from carbohydrates. The strongest correlations with dietary intake are provided by plasma concentrations of n-3 PUFA and *trans*-fatty acids (Sun 2007).

Health Effects

Foods and fats with SFA, MUFA, and PUFA influence various factors related to cardiovascular disease (CVD), including low-density lipoprotein (LDL) cholesterol-particle size and oxidation, remnant particles, lipoprotein (a), systemic inflammation, thrombosis and coagulation, endothelial function, insulin sensitivity, oxidative stress, and blood pressure, among other mechanisms that have established or potential causal effects on CVD (Astrup 2025).

SFA. Despite the established LDL-raising effect of SFA, debate persists regarding their impact on CVD (Siri-Tarino 2010; Muto 2018; Harrison 2020; Heileson 2020; King 2020; Valk 2022). Restricting fat intake and replacing SFA with other nutrients has revealed added complexity in the diet-heart issue. According to evidence from human studies, replacing SFA with unsaturated fatty acids lowers cardiovascular risk (Mozaffarian 2010; Nettleton 2017; Julibert 2019; Hooper 2020). Replacing SFA with carbohydrates may be harmful or beneficial depending upon the quality of the carbohydrate (Jakobsen 2009; Jakobsen 2010). Carbohydrates vary in the rate at which they increase blood glucose. Highly refined carbohydrates are amongst the former, and higher intake of these is associated with greater risk for the development of diabetes (Barclay 2008). Replacing SFA with highly refined carbohydrates and added sugars may increase heart disease risk through promotion of obesity and diabetes (Hu 2010). Fiber-rich whole grains, on the other hand, have shown beneficial effects on cardiovascular health (Briggs 2017). Emerging evidence highlights the importance of overall dietary patterns, rather than single nutrients like SFA, in improving cardiovascular health. Diets rich in minimally processed foods are more likely to improve cardiovascular health (Gershuni 2018; Wu 2019; Harrison 2020). The American Heart Association (AHA) presidential advisory panel on dietary fats and CVD concluded that "lowering intake of saturated fat and replacing it with unsaturated fats, especially polyunsaturated fats, will lower the incidence of CVD" (Sacks 2017). The AHA guideline for prevention of subsequent or secondary stroke recommends the adoption of a Mediterranean-style diet to lower stroke risk (Kleindorfer 2021).

MUFA. The most common MUFA is oleic acid (C18:1n-9). Although humans can synthesize this fatty acid, it is obtained largely through a plant-based diet (e.g., vegetable oils, nuts, seeds, avocados). Evidence suggests that replacing dietary carbohydrates with MUFA decreases LDL-cholesterol concentration (Astrup 2011). Recent meta-analyses indicate that higher MUFA intake may reduce the risk of total and hemorrhagic strokes, but not ischemic stroke (Mehrabani 2025). The source of MUFA is critical. Plant-based MUFA is associated with lower mortality, while animal-based MUFA is linked to higher mortality (Guasch-Ferré 2019). Large U.S. cohort studies showed that replacing trans fats, SFA, or refined carbohydrates with plant-based MUFA, but not animal-based MUFA, lowered coronary heart disease risk (Zong 2018).

PUFA. The heart-healthy effects of PUFA are most often assessed based on their effects on the concentrations of total cholesterol, high-density lipoprotein (HDL)- and LDL-cholesterol, cholesterol ratios, and/or triglycerides. Intake of n-6 PUFA helps to lower total cholesterol and LDL-cholesterol, however, high intake may lower HDL-cholesterol levels. In contrast, n-3 fatty acid consumption has been shown to lower triglycerides, slightly increase HDL and even more LDL levels, but not the total cholesterol (Balk 2006). An NHANES analysis of dietary omega-3 PUFA intake from 1999 to 2018 showed that particularly alpha-linolenic acid, eicosapentaenoic acid (EPA), and docosapentaenoic acid (DPA) were associated with lower risk of all-cause and cardiovascular mortality among U.S. hypertensive adults (Chen 2023). In the United States, the FDA permits qualified health claims to be made about a diet-disease relationship for cardiovascular disease; currently, the majority of the claims for cardiovascular disease are related to the MUFA or PUFA content of nuts, oils and spreads, or fish oil supplements (U.S. Department of Health and Human Services and U.S. Food and Drug Administration 2024). In a randomized controlled trial of age-related macular degeneration, supplementation with docosahexaenoic acid (DHA)/EPA showed no significant difference in risk over a 10-year period (Chew 2022). While a recent meta-analysis of 51 randomized controlled trials including 40 crossover trials showed a small but significant improvement in blood lipid profiles (reduced total cholesterol ~5%, LDL ~3%, and triglycerides ~7%) with high PUFA-rich compared to high MUFA-rich diets (Prater 2026), the heterogeneity of diets and the risk of confounding by other nutrients (polyphenols and other bioactive components) in the diet led to uncertainty as to whether the differences in blood lipid effects between the PUFA-rich and MUFA-rich diets can be attributed exclusively to PUFA or MUFA (Astrup 2025).

Deficiency in the essential fatty acids is determined by the use of a plasma triene-to-tetraene ratio (eicosatrienoic [C20:3n-9]: arachidonic [C20:4n-6] acid); a ratio greater than 0.2 indicates deficiency (Institute of Medicine 2005). PUFA deficiency may manifest as peripheral neuropathy and skin problems, such as rough or scaly skin and dermatitis (International Life Sciences Institute 2001).

Intake Recommendations

Because the body makes more than enough SFA to meet metabolic needs, people have no requirement for these fatty acids. Evidence suggests that SFA are positively associated with total cholesterol and LDL-cholesterol concentrations and thus with CVD risk (Jakobsen 2009). The National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (ATP III) recommends lowering dietary intake of SFA to no more than 10% of caloric intake and replacing SFA with MUFA and PUFA to reduce the risk of CVD; moreover, lowering the percentage of calories derived from SFA to 7% of total calories, can further reduce risk of CVD (Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults 2001). The panel also recommends that MUFA not exceed 20% of calories. The AHA recommends aiming for a diet with less than 6% of total calories from saturated fat and the 2025-2030 Dietary Guidelines for Americans recommend less than 10% (American Heart

Association 2026; U.S. Department of Agriculture and U.S. Department of Health and Human Services 2026). Guidance about adequate intake (AI) is available for the essential fatty acids. The AI of linoleic acid ranges from 10–17 g/day for males and 10–12 g/day for females depending on age. For alpha-linolenic acid, the AI ranges from 0.9–1.6 g/day for males and 0.9–1.1 g/day for females depending on age. The 2025-2030 Dietary Guidelines for Americans recommend that when cooking with or adding fats to meals, one should prioritize oils with essential fatty acids, such as olive oil (U.S. Department of Agriculture and U.S. Department of Health and Human Services 2026). There are no upper tolerable limits for n-3 or n-6 PUFA (Institute of Medicine 2005).

According to the AHA, patients with coronary heart disease should be encouraged to increase their consumption of EPA and DHA to about 1 gram per day, preferably from oily fish. The AHA dietary guidelines recommend at least two servings of fish per week (particularly fatty fish) for patients without documented coronary heart disease (Kris-Etherton 2002). In addition, they recommend including vegetable oils (e.g., soybean, canola, walnut, flaxseed) and food sources (e.g., walnuts, flaxseeds) high in alpha-linolenic acid in a healthy diet for the general population. The Dietary Reference Intake Report for Energy and Micronutrients supports 5–10% of dietary energy from n-6 fatty acids (Institute of Medicine 2005), while the Adult Treatment Panel III recommends that up to 10% of total calories may be consumed from polyunsaturated fats (Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults 2001).

Biochemical Indicators

The long-term fatty acid content of the diet is best represented by the adipose tissue triglyceride content. This is because of the two-year half-life of adipose tissue fatty acids. Erythrocytes, due to their 120-day half-life, reflect intermediate-term (weeks-to-months) habitual dietary intake and are a strong indicator of long-term n-3 status (Dicklin 2024). However, it has also been shown that large changes in the fatty acid composition of erythrocytes can be observed within days of altering dietary fat intake (Hodson 2008). Serum or plasma concentrations represent more recent intake (days-to-weeks).

Few studies have compared the fatty acid composition of plasma with red blood cells (RBC) to assess which substrate best reflects dietary intake. In one small study of a few hundred U.S. women, fatty acid correlations with food frequency questionnaire data were only slightly stronger for erythrocytes compared with plasma (Sun 2007). The triglyceride fraction of plasma appears to demonstrate the greatest day-to-day variation of any circulating lipid fraction. Thus, whenever serum or plasma is collected for fatty acid analysis, fasting is preferred to minimize the within- and between-person variability at the time of specimen collection. Generally, fatty acid data are expressed as percentage by weight of total fatty acids, although percentage by mole would be more meaningful. The possible advantages of using absolute concentrations of individual fatty acids have not been well investigated (Hodson 2008).

The omega-3 index, representing the sum of EPA and DHA concentrations in RBCs, is strongly correlated with the risk of sudden cardiac death and has therefore been proposed as a marker of cardiovascular health (Harris 2004). Omega-3 index values <4%, 4%–8%, and >8% are considered undesirable, intermediate, and optimal, respectively, and correspond to high, intermediate, and low coronary heart disease risk, respectively (Harris 2004; Harris 2025).

Analytical Methods

Capillary gas chromatography (GC) is the technique most frequently used to separate fatty acids for quantitative analysis. Detection methods include flame ionization or electron capture negative chemical ionization mass spectrometry. Internal standards are used to correct for losses during sample preparation and improve the accuracy and precision of measurements.

Findings from NHANES

The National Health and Nutrition Examination Survey (NHANES) is the only source for nationally representative data on fatty acids for the U.S. population (Pfeiffer 2026). A multiple regression analysis of NHANES 2003–2004 showed that after controlling for demographic variables (age, sex, and race and Hispanic origin), smoking, supplement use, fasting, inflammation, and renal function, inflammation was associated with significantly higher SFA (10.7%), MUFA (11.3%), PUFA (4.0%), and total fatty acid (7.3%) concentrations. Impaired renal function was associated with significantly lower SFA (-4.8%) and PUFA (-7.5%) concentrations (Haynes 2013). Pregnancy (in women 20–49 years of age) was associated with significantly higher SFA (28%), MUFA (29%), PUFA (18.6%), and total fatty acid (22.9%) concentrations (Haynes 2013).

Data from NHANES 2003–2004 showed that 80.6% of U.S. adults (≥20 y) had plasma long-chain omega-3 concentrations below the AHA recommended level of 2.5% (Murphy 2015). Using serum fatty acid data from NHANES 2011–2012, investigators calculated the percent of total fatty acids for two long-chain omega-3 fatty acids (EPA and DHA), as well as for the sum of EPA and DHA representing the omega-3 index, and studied the relationship between dietary intake of total fat and different fatty acid classes with the omega-3 index (Jin 2025). They found that intake of total fat, SFA, and MUFA was inversely correlated with omega-3 index levels (when EPA+DHA intake was constant).

Using the first nationally representative data for RBC fatty acids from NHANES August 2021–August 2023, investigators calculated the omega-3 index from the sum of EPA and DHA (Powers 2026). They found that over half of the U.S. population had an undesirably low omega-3 index (<4%) and 98% had values less than optimal (<8%), which was similar to the finding from the Canadian Health Measures Survey (Demonty 2021).

For more information on polyunsaturated fatty acids, see the Dietary Reference Intakes report (Institute of Medicine 2005) and fact sheets from the National Institutes of Health, Office of Dietary Supplements (http://ods.od.nih.gov/FactSheets/Omega3FattyAcidsandHealth.asp).

Data in the 2026 tables

Data presented are from univariate analysis that was not adjusted for demographic variables (e.g., age, sex, race and Hispanic origin) or other blood concentration determinants (e.g., dietary intake, supplement use, smoking, BMI). Data for plasma or serum concentrations of fatty acids were available for three survey cycles. Data from NHANES 2003–2004 were acquired using surplus plasma that was stored at -70°C until 2010–2011 when fatty acids were measured. Based on limited information in the literature about absolute concentrations, these 24 fatty acids are estimated to comprise at least 90% of the total fatty acids circulating in plasma in all lipid classes (fatty acyls, glycerolipids, glycerophospholipids, sphingolipids, and sterols). The six additional fatty acids measured in serum for NHANES 2011–2012 and 2013–2014 were minor contributors to the overall fatty acid profile. While eicosatrienoic acid needed to assess deficiency in essential fatty acids was not part of the plasma fatty acid profile measured for NHANES 2003–2004, it was included in the serum fatty acid profile measured for NHANES 2011–2014.

Data for RBC fatty acids were available for one survey cycle, NHANES August 2021–August 2023. Data were acquired from saline-diluted RBCs using a profile of 21 fatty acids. RBC fatty acid data allow for the calculation of the omega-3 index, which is an indicator of overall cardiovascular health.

All unsaturated fatty acids are assumed to be in the *cis* configuration; *trans*-fatty acids were not measured as part of these four NHANES survey cycles. The same analytical method has been used across all three survey cycles: gas chromatography-mass spectrometry (GC-MS) based on a modification of a published method (Lagerstedt 2001). Plasma and serum fatty acid concentrations are reported in µmol/L units. RBC fatty acid data are reported in weight percentages. For each analytical run, all measured RBC fatty acid concentrations (mg/L), including those below the limit of detection (LOD), were used to calculate the total fatty acid concentration so that reported weight percentages reflected the complete run-specific fatty acid profile. There were 15 cases where a value <LOD was used in the calculation (2 results for eicosapentaenoic acid and 13 results for myristic acid). Individual fatty acids whose mass concentrations were <LOD or whose weight percentage values failed QC were reported as missing. All reported weight percentages were calculated using the total fatty acid concentration from the same analytical run as the denominator.

	NHANES survey cycle		
	2003–2004	**2011–2014**	**2021–2023**
	Plasma	**Serum**	**RBC**
	Fasting adults ≥20 y	**Children 3–11 y and fasting persons ≥12 y**	**Persons ≥6 y**
Omega-3 index (sum EPA + DHA)			X
Saturated fatty acids			
Capric (10:0)		X	
Lauric (12:0)		X	
Myristic (14:0)	X	X	X
Pentadecanoic (15:0)		X	
Palmitic (16:0)	X	X	X
Margaric (17:0)		X	
Stearic (18:0)	X	X	X
Arachidic (20:0)	X	X	X
Docosanoic (22:0)	X	X	X
Tricosanoic (23:0)		X	
Lignoceric (24:0)	X	X	X
Monounsaturated fatty acids			
Myristoleic (14:1n-5)	X	X	
Palmitoleic (16:1n-7)	X	X	X
cis-Vaccenic (18:1n-7)	X	X	
Oleic (18:1n-9)	X	X	X
Eicosenoic (20:1n-9)	X	X	X
Docosenoic (22:1n-9)	X		
Nervonic (24:1n-9)	X	X	X
Polyunsaturated fatty acids			
Linoleic (18:2n-6)	X	X	X
alpha-Linolenic (18:3n-3)	X	X	X
gamma-Linolenic (18:3n-6)	X	X	X
Stearidonic (18:4n-3)		X	
Eicosadienoic (20:2n-6)	X	X	X
homo-gamma-Linolenic (20:3n-6)	X	X	X
Eicosatrienoic (20:3n-9)		X	
Arachidonic (20:4n-6)	X	X	X
Eicosapentaenoic (20:5n-3)	X	X	X
Docosatetraenoic (22:4n-6)	X	X	X
Docosapentaenoic (22:5n-3)	X	X	X
Docosapentaenoic (22:5n-6)	X	X	X
Docosahexaenoic (22:6n-3)	X	X	X

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