Dietary Fats

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Lipids comprise a group of polar and nonpolar compounds, including triglycerides (TGs), diglycerides, monoglycerides, fatty acids, phospholipids, and sterols. In the diet, lipids contribute to the taste, texture, and energy content of a food. In the body, lipids have many roles including a source of readily available and stored energy, a structural and functional component of all cell membranes, and precursors for eicosanoids and cell signaling molecules, in addition to helping with the absorption of fat-soluble vitamins and other food components.

TGs are composed of a glycerol backbone with 3 fatty acids of varying chain length and desaturation (i.e. number of double bonds). The description of fats on the Nutrient Facts label considers the number and position of double bonds in the carbon chain and/or the chain length of the fatty acids. SFAs have no double bonds, MUFAs have 1 double bond, and PUFAs have ≥2 double bonds. *Trans* fatty acids have ≥1 double bond in the *trans*, rather than *cis*, configuration, making them structurally more similar to SFAs. Fats can also be categorized by their chain length as short chain (3–6 carbons), medium chain (8–14 carbons), or long chain (16 or more carbons). Phospholipids contain 1 glycerol molecule that is esterified with 1–2 fatty acids and a polar head group. Phospholipids, cholesterol, and plant sterols comprise only a small portion of the diet. Thus, this review will focus on TGs which comprise 90–95% of the lipids in the diet and body.

Digestion of TGs is complex. Minimal digestion begins in the mouth with lingual lipases followed by emulsification and some digestion in the stomach through the action of gastric enzymes. The crude emulsion of lipids enters the duodenum and mixes with bile and pancreatic juice that contains enzymes (e.g. pancreatic lipase, colipase). TGs are digested to monoglyceride and 2 fatty acids and the subsequent absorption of these digestion products from the intestinal lumen into the enterocytes and secretion into circulation is a complex process. Although there is some further hydrolysis by pancreatic lipase that can result in the formation of glycerol and free fatty acids, monoglycerides with a fatty acid in the *sn-2* position and 2 fatty acids are the predominant forms that are available for absorption. The fatty acids are taken up by enterocytes via both protein-mediated (i.e. transporters) and protein-independent processes. Transport of absorbed lipids from the plasma membrane to the endoplasmic reticulum involves intracellular trafficking proteins. Here, the absorbed fats are reassembled into chylomicrons and to a lesser extent VLDLs and secreted from the enterocytes. In the liver, fats can be further metabolized and repackaged into lipoproteins (primarily VLDLs). Tissues (primarily adipose tissue) will remove the lipids from chylomicrons and lipoproteins, hydrolyze them with lipoprotein lipase, and use them for energy or storage. Across the life cycle, in the absence of intestinal or pancreatic diseases, fats from the diet are well-digested and absorbed before reaching the large intestine. However, if some fat does reach the large intestine, it will be metabolized by the microbiome. For a more detailed description of lipid digestion, absorption, and transport see (1).

Diet Recommendations

For infants, fat is the major source of energy and an adequate intake (AI) (31 g/d) from 0 to 6 months is based on the estimated fat intake of exclusively breast-fed infants (2). The AI (30 g/d) for older infants (7–12 mo) is based on the estimated intakes of human milk and complementary foods (2). For adults there is no established requirement for total fat and dietary fat recommendations are given as the acceptable macronutrient distribution range (AMDR) of 20–35% of energy (2). As they are not considered dietary essential, there are no AI for SFAs and MUFAs across the life cycle. There are no upper limits (UL) established for SFAs, MUFAs, or PUFAs (n–6 or n–3) at this time. As there are no benefits to health there is not a DRI for *trans* fatty acids, and the current recommendation is to consume as little as possible *trans* fatty acids in the diet (2).

Unlike SFAs or MUFAs, the body cannot synthesize n–3 and n–6 PUFA and a dietary source is essential. AI have been set for n–6 and n–3 fatty acids for the major form in

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the diet, linoleic and α-linolenic acid, respectively. The AI (19 to >70 y) for linoleic acid is 17 g/d and 12 g/d and for α-linolenic acid 1.6 g/d and 1.1 g/d for men and women, respectively (2). The AI for both of these fatty acids increases during pregnancy and lactation. Although there is some evidence for a functional role of linoleic and α-linolenic acid in the body, the metabolic essential forms of these PUFAs are generally accepted to be the 20 carbon and longer elongated and desaturated PUFAs. The AMDR for n–6 (linoleic acid) is 5–10% of energy and for n–3 is 0.6–1.2% with ≤10% of this consumed as eicosapentaenoic and/or docosahexaenoic acid (2). The 2015–2020 Dietary Guidelines for Americans offers the following recommendations: Avoid *trans* fat, limit SFAs to <10% of energy (calories) a day, replace SFAs with healthier MUFAs and PUFAs.

Food and Clinical Product Sources

Fat is abundant in the diet of North Americans and all food sources contain a variety of different fatty acids. The major source of SFAs are animal fats and deep-fried and bakery products. Oleic acid accounts for >90% of dietary MUFAs coming from olive oil, oils from the newer high MUFA cultivars of canola, soy, and sunflower, and animal fats (40% MUFAs). The major source of PUFAs are plant and seed oils, nuts, and food products made with these. Long-chain n–3 PUFAs in the diet are primarily from fish and other marine foods. The majority of *trans* fats in the diet have come from the partial hydrogenation of plant oils. A small proportion of *trans* fats come from ruminant animal products, such as milk and meat. However, the negative health implications have not been demonstrated for rumen-derived *trans* fatty acids and for this reason, the major ruminant-derived fatty acid, conjugated linoleic acid, is not included in the calculation of *trans* fat on the food label. Lipids, primarily TGs, comprise 20–40% of the energy in enteral products for individuals who are unable to consume sufficient amounts of food to meet their energy needs. Clinically, lipids constitute the major energy source for parenteral (intravenous) feeding to individuals who cannot consume food orally.

Deficiencies

Dietary fat is an important energy source and if insufficient intake (together with inadequate protein and carbohydrate) occurs, an individual will go into negative energy balance and lose weight and/or not grow. Classical deficiencies of n–3 and/or n–6 PUFAs include neurological abnormalities, a scaly rash, and poor growth. This level of deficiency only occurs in individuals with severe malnutrition/starvation and/or chronic fat malabsorption such as in cystic fibrosis, pancreatic insufficiencies, or damaged intestines.

Toxicity/Health Risks

Individuals have the capacity to adapt to a wide range of fat intakes. Higher fat intake, primarily SFA, has been associated with increased risk of obesity, type 2 diabetes, coronary heart disease, and cancer (3). However, recent research, coming from the popularity of low-carbohydrate high-fat diets, is challenging some of these concerns about high fat and high SFA intakes and disease risk (4). Importantly, low intake and or imbalanced proportions of n–6 and n–3 fatty acids have been associated with an increased risk of many chronic diseases, such as cancer and cardiovascular disease (CVD) (5).

Recent Research

Dietary fat and its relation to health is being heavily researched. Below are some trends in the current research.

- The association between fat consumption and the risk of chronic disease is among the most vexed issues in public health: Are dietary fats "villains," are they "benign", or are they even "heroes" that could help us consume better overall diets and promote health? This is the result of new research: high-fat diets promote weight loss better than high-carbohydrate diets; the biological effects and health risks of the different SFAs are not the same; the understanding that high LDL-cholesterol (increased by SFA intake) may not be the most important risk factor for CVD; and research demonstrates that dietary fats influence other biomarkers of CVD, including inflammation, thrombosis, ventricular arrhythmias, and blood pressure in positive ways (4).
- The emphasis on fat and types of fats is turning more towards a food/food pattern approach where the 'healthy diet pattern' contains a variety of different fats and may even be high in fat. In the era of genomics, it is becoming clear that dietary fats alter lipids and other biomarkers of health in not only racial and ethnic subgroups but also those with different genetic phenotypes and the future of fat recommendations may be moving further away from population recommendations towards individualized prescriptions.
- Certain fatty acids or groups of fatty acids previously considered benign, harmful, or nonessential, are being studied for their potential health benefits. This list includes MUFAs, medium chain fatty acids (coconut oil and role in inducing ketosis), long chain PUFAs (arachidonic, eicosapentaenoic, docosapentaenoic, and docosahexaenoic acids), and vaccenic acid (a *trans* fatty acid in dairy and meats from ruminant animals).
- As inflammation is a risk factor for almost every chronic disease, dietary fats are being studied as mediators/regulators of inflammatory mediators and processes in a variety of conditions, including obesity, type 2 diabetes, CVD, and cancer.

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