Chapter 2
Nutritional Aspects of Fats and Oils

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Abbreviations

AA	Arachidonic acid (20:4n-6)
AI	Adequate intake
ALA	Alpha-linolenic acid (18:3n-3)
CHD	Coronary heart disease
CVD	Cardiovascular disease
DHA	Docosahexaenoic acid (22:6n-3)
E%	Energy %
EPA	Eicosapentaenoic acid (20:5n-3)
HDL	High-density lipoprotein
LA	Linoleic acid (18:2n-6)
LDL	Low-density lipoprotein
MUFA	Monounsaturated fatty acids
NCDs	Noncommunicable diseases
PUFA	Polyunsaturated fatty acids
RCT	Randomized controlled trial
SFA	Saturated fatty acids
WHO	World Health Organization

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2.1 Introduction

Fats and oils are important for the quality of foods (taste, smell, texture, and nutrition). Dietary fat is a major nutrient, an excellent source of energy, and provides essential fatty acids. Moreover, fats and oils provide fat-soluble vitamins and other fat-soluble compounds such as carotenoids and sterols, and many flavor-active compounds. Dietary fat may play a significant role in the prevention and treatment of noncommunicable diseases (NCDs), also known as chronic diseases. According to the Global Burden of Disease study a diet high in saturated fatty acids (SFA) and *trans* fatty acids is a leading risk factor for several NCDs, in particular metabolic diseases such as cardiovascular disease (CVD) and type 2 diabetes [1, 2]. Thus, the World Health Organization (WHO) guidelines and National Dietary Guidelines recommend limiting the intake of saturated fat and replacing solid fat high in SFA and/or *trans* fatty acids with oils high in monounsaturated fatty acids (MUFA) and polyunsaturated fatty acids (PUFA), with the challenges this presents regarding taste and texture in food products. Oleogels are promising alternatives to solid fats for food applications since they successfully replace solid fat with oils in food products without compromising on food quality. They are reported to be an innovative structured fat system used for industrial applications due to their nutritional and environmental benefits [3]. The dietary guidelines for fat, the scientific support for these advices and the rationale for altering dietary fat intake, both quantitatively and qualitatively, will be discussed in the following sections. We will describe how all dietary fats and oils are made up of saturated fatty acids (SFA), monounsaturated fatty acids (MUFA), and polyunsaturated fatty acids (PUFA) in different proportions, affecting both melting point and nutritional value.

2.2 Fatty Acid Composition of Dietary Fats, Oils, and Food Products

Edible fats and oils may be of vegetable, animal, and marine origin. In food products, fats and oils come from raw materials or as added ingredients. They consist mainly of triacylglycerols and other acylglycerols, where fatty acids are esterified with glycerol. In commercial fats and oils, the content of other lipids is, in general, less than 2% [4]. The nomenclature for fatty acids indicates number of carbon atoms (chain-length), number of double bonds (degree of saturation), and location of the first double bond at carbon counted from the omega or n end; for example, the omega-3 fatty acid α-linolenic acid (ALA; 18:3n-3) has 18 carbons, 3 double bonds, where the first double bond is located at carbon number 3 from the n end.

The fatty acid composition has a major effect on the properties of dietary fats and oils. All dietary fats and oils are made up of SFA, MUFA, and PUFA in different

Table 2.1 Typical composition of major fatty acids (% of total fatty acids) in common solid dietary fats (milk fat, beef fat/tallow, and pork fat) and semisolid dietary fats (coconut oil and palm oil) [4]

	Milk fat	Beef fat / Tallow	Pork lard	Coconut oil	Palm oil
12:0 (lauric acid)	2.9	0.2	0.1	47.5	–
14:0 (myristic acid)	10.8	4.0	1.5	18.1	1.1
16:0 (palmitic acid)	26.9	24.3	26.0	8.8	44.0
18:0 (stearic acid)	12.1	21.4	13.5	2.6	4.5
18:1n-9 (oleic acid)	28.5	33.6	43.9	6.2	39.2
18:2n-6 (linoleic acid)	3.2	1.6	9.5	1.6	10.1
18:3n-3 (α-linolenic acid)	0.4	0.6	0.4	–	0.4
20:5n-3 (eicosapentaenoic acid)	–	–	–	–	–
22:6n-3 (docosahexaenoic acid)	–	–	–	–	–
SFA	65.0	53	41.7	92.1	49.6
MUFA	31.4	37.9	47.7	6.2	39.3
PUFA (n-6)	3.2	1.6	9.5	1.6	10.1
PUFA (n-3)	0.4	–	–	–	–
Melting range	28–36 °C	45–48 °C	32–33 °C	25–28 °C	36–45 °C

SFA saturated fatty acids, *MUFA* monounsaturated fatty acids, *PUFA* polyunsaturated fatty acids

proportions (Tables 2.1 and 2.2), affecting both melting point and nutritional value. The melting point for a fatty acid depends on both chain length and number of double bounds. In general, SFA have higher melting point than MUFA and especially higher than PUFA. Most double bonds in the dietary unsaturated fatty acids are in the *cis* configuration, while *trans* double bonds can be found in ruminant fats (low levels) and in partially hydrogenated oils (higher levels). The term fat often refers specifically to triacylglycerols that are solid or semisolid at room temperature, thus excluding liquid oils. Position of fatty acids in the glycerol backbone also influences fats' physical and nutritional properties [5, 6].

Animal fats such as beef fat, pork fat and butter contain high amounts of SFA (between 25% and 65%) with relative high melting points, and they are all solid at room temperature (Table 2.1).

They also contain a relatively high level of MUFA (30–55%), but a small amount of PUFA (2–20%), mainly linoleic acid (LA; 18:2n-6). Butter fat contains approximately 4–8% of *trans* fatty acids [6]. Vegetable fats include the semisolid fats like coconut oil and palm oil, with high amounts of SFA (55–95%) (Table 2.1). Among the SFA, coconut contains mainly lauric acid (12:0) and myristic acid (14:0). In palm oil, the major SFA are palmitic acid (16:0) and stearic acid (18:0) [5, 7, 8]. Fractionation of palm oil is used to produce materials with desired melting properties with varying fatty acid compositions.

Common vegetable oils such as soybean oil, sunflower oil, olive oil, and rapeseed oil have less than 15% SFA and approximately 85% unsaturated fatty acids [7, 8]

Table 2.2 Typical composition of major fatty acids (% of total fatty acids) in common dietary oils

	Soybean oil[a]	Sunflower oil[a]	Rape seed oil[a]	Olive oil[a]	Cod liver oil[b]	Salmon oil[c]
12:0 (lauric acid)	–	–	–	–	–	–
14:0 (myristic acid)	0.1	0.1	0.1	–	3.6	3.2
16:0 (palmitic acid)	10.6	7.0	4.1	9.0	10.4	13.7
18:0 (stearic acid)	4.0	4.5	1.8	2.7	2.6	3.7
18:1n-9 (oleic acid)	23.3	18.7	60.9	80.3	16.2	29.6
18:2n-6 (linoleic acid)	53.7	67.5	21.0	6.3	1.5	16.7
18:3n-3 (α-linolenic acid)	7.6	0.8	8.8	0.7	–	3.3
20:5n-3 (eicosapentaenoic acid)	–	–	–	–	9.3	7.5
22:6n-3 (docosahexaenoic acid)	–	–	–	–	11.9	6.3
SFA	11.4	12.7	7.2	12.1	16.6	21.3
MUFA	23.4	18.9	62.9	80.9	46.4	40.5
PUFA (n-6)	53.7	67.5	21.0	6.3	1.5	17.8
PUFA (n-3)	7.6	0.8	8.8	0.7	23.6	20.5
Melting range	−20 to −23 °C	−18 to −20 °C	−9 °C	0 °C	−70 to 14 °C	−70 to 14 °C

SFA saturated fatty acids, *MUFA* monounsaturated fatty acids, *PUFA* polyunsaturated fatty acids
[a] [4]
[b] [9]
[c] [10]

(Table 2.2). In addition, olive oil and rapeseed oil are high in MUFA (60–70%) having oleic acid (18:1n-9) as the major fatty acid. The content of PUFA in rapeseed oil (ca. 30%) is higher than in olive oil (ca. 10%), and moreover rapeseed oil contains both LA (ca. 20%) and ALA (ca. 10%), which makes it a good source of both n-6 and n-3 fatty acids. Soybean oil, corn oil, and sunflower oil are high in n-6 PUFA (54–68%), mainly LA. Among them soybean oil is the only one to contain a significant amount of the n-3 PUFA ALA (8%). Different fish oils and fractions of fish oils are usually used as dietary supplements and dietary oils of marine origin are less usual. Refined cod liver oil and salmon oil are two dietary marine oils which are produced with food ingredient quality. Both are high in marine n-3 PUFA, the health-promoting eicosapentaenoic acid (EPA; 20:5n-3) and docosahexaenoic acid (DHA; 22:6n-3). The melting points of marine oils depend on refining grade since some of the refining procedure include precipitation of solid fat high in saturated fatty acid, mainly 18:0.

Typical composition of the main fatty acids in common dietary fats and oils is presented in Tables 2.1 and 2.2 and will be discussed in later sections concerning nutrition and health. Usually, fats contain more SFA than oils, which might lower their nutritional quality. However, solid fats or semisolid fats are often needed in food application to build desired textures and desired sensory properties as well as to improve storage stability. The physical state also influences the release of flavor-active compounds in the mouth. Nowadays, solid fats are obtained from natural fats, fully hydrogenated oils, and structured lipids, whereas the use of partially hydrogenated oils has diminished due to their content of *trans* fatty acids. Oleogels are a means to improve the nutritional quality of food products that need solid fats in the structure by replacing them with vegetable and marine oils.

2.3 Guidelines for Fat Intake

Dietary guidelines are statements that assist populations in choosing foods that deliver optimal nutrient intake and are associated with a reduced risk of NCDs, such as heart disease, cancer, chronic respiratory disease, and diabetes [11, 12]. The first Dietary Guidelines for Americans were released in 1980, where "avoiding too much saturated fat" was recommended [13]. In 2004, the WHO Global Strategy on Diet and Physical Activity recommended shifting consumption from saturated fat to unsaturated fats and limiting the level of saturated fat in the diet [14–16]. All Dietary Guidelines have recommended reductions in saturated fat, with the first numerical target of $<10\%$ of calories issued in 1990. Subsequent editions of the Dietary Guidelines for Americans (2010 and 2015) also introduced replacement of SFA with n-3 PUFA [17–20]. The message to decrease SFA has been supported by the American Heart Association/American College of Cardiology [21], the National Lipid Association [22], and the global recommendations issued by WHO [23].

The European Food Safety Authority (EFSA) (2010) has set dietary reference values only for a few fatty acids [24]. The content of SFA and *trans* fatty acids should be as low as possible. An adequate intake (AI) for LA was set to 4 energy% (E%) and for ALA 0.5 E%, and without reference values for upper intake. WHO recommends less than 10 E% from SFA and less than 1 E% from *trans* fatty acids [25]. There is no general official recommendation for daily intake of the marine n-3 PUFA (EPA and DHA). EFSA has suggested an AI of 250 mg of sum of EPA + DHA for adults [24], whereas WHO has recommended a daily dose of 300–500 mg EPA + DHA. However, the Global Recommendations for EPA and DHA are that healthy adults should consume a minimum of 500 mg of EPA + DHA daily to lower the risk of coronary heart disease (CHD), but higher doses of EPA + DHA (700–1000 mg/day) are often needed for individuals with metabolic risk factors, pregnant/lactating women, in infancy and during specific periods of development, and for secondary prevention of coronary heart disease (CHD) [26].

2.4 Polyunsaturated Fatty Acids and Health

2.4.1 Health Effects of n-6 and n-3 Polyunsaturated Fatty Acids

The n-6 PUFA LA and n-3 PUFA ALA are essential fatty acids that are vital to human health and must be provided in the diet. They have important physiological functions; for example, LA is essential for maintaining the water permeability barrier of the skin. Previous studies on intake of the LA and CHD risk have generated inconsistent results. In prospective observational studies, dietary LA intake is inversely associated with CHD risk in a dose-response manner [27], and these data provide support for current recommendations to replace SFA with LA to lower risk of CHD. On the other hand, Hoenselaar [28] raised comments regarding the review article by Farvid et al. [27], where the questions raised were: "Do different dietary sources of LA have the same influence on CHD?," and "Do different n-6 PUFA oils have various effects on CHD?." Lucas [29], also, commented on the review article by Farvid et al. [27], where Lucas hypothesized that an imbalance between n-6 and n-3 PUFA intakes may cause CVD, since LA is also the precursor of arachidonic acid (AA; 20:4n-6), from which pro-inflammatory eicosanoids and cytokines are derived. LA is present in variable quantities in many plant oils and human diets, and, therefore, specific LA deficiency does not seem to occur in the human body, and increased consumption of LA will not cause inflammation during normal metabolic conditions unless lipid peroxidation products are mixed in.

The n-3 PUFA ALA is found in certain plants, such as in dark green leafy vegetables, nuts, and oils from seeds (Table 2.2). Compared to the marine n-3 PUFA EPA and DHA, the health effects of ALA have been less studied [30]. It is well known that ALA is an essential fatty acid and a precursor of EPA and DHA in all mammals. On the other hand, the efficiency of this conversion in vivo is quite low in various species, and it is still debated whether dietary ALA can fulfil the needs of the human body or whether dietary intake of preformed DHA is necessary. The American Heart Association recommends that people without documented CHD eat a variety of fish (preferably oily) at least twice weekly (approx. 500 mg EPA + DHA) [31]. Epidemiological studies on the impact of fish consumption on CHD incidence have shown inconsistent results [32]. Previous meta-analyses showed that fish consumption reduces the risk of CHD; however, several incorporated studies show that fish consumption has no impact on CHD. In most of the published scientific papers the type of fish is not mentioned. Kris-Etherton and coworkers [33] noted that all fish are not equal in EPA and DHA content, and they raised the question "Does it matter whether the fish is fatty or lean?." The authors messages were "when studying fish intake in relation to risk of diseases it is important to have in mind that EPA and DHA content of fish varies between fish species, and that several factors such as sex, age, water temperature and season have an effect on the fatty acid composition of membrane lipids in all fish tissues." Findings strongly suggest that the data obtained from marine fatty fish-eating populations cannot be generalized to all fish-eating

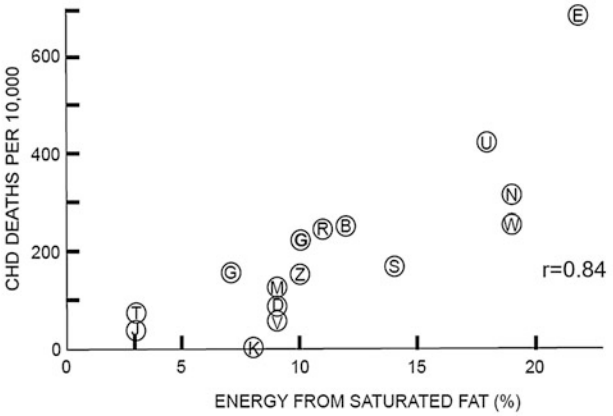
populations. No matter how many freshwater local catches are eaten in the communities studied, serum EPA and DHA concentrations are not affected [34].

The health benefits of marine n-3 PUFA EPA and DHA are well documented, indicating protective effects on CVD, autoimmune, and mental disorders [35]. A wide range of beneficial effects of EPA and DHA, including anti-atherothrombogenic effect, reduction in serum triglycerides, effects on arrhythmia, hypertension, and inflammation, have been suggested as possible explanations for the reduction in CVD [35, 36]. Recent research has demonstrated that EPA and DHA have distinct tissue distributions where they influence target organs in different ways [37]. EPA mainly provides the starting point for making hormones that regulate blood clotting, contraction, and relaxation of artery walls. Inflammation has been shown to improve atherosclerotic plaque stabilization in blood vessels, where it interferes with lipid oxidation and various signal transduction pathways linked to inflammation and endothelial dysfunction. These findings support a mechanistic basis for a potential benefit with dietary/supplemented EPA in reducing cardiovascular risk as is currently being tested in ongoing clinical trials [38], where health biomarkers will also be influenced by genetic variants [39] (see Sect. 2.5.3).

2.5 Saturated Fat and Cardiovascular Disease (CVD)

2.5.1 The Lipid Hypothesis in Atherogenesis

Saturated fat has been a topic of nutrition debate and dietary advice for more than a century. Consequently, the need to reduce levels of saturated fat in foods and the different ways of doing this have become one of the most important issues facing the food industry. The background is the supposed link between saturated fat intake and atherosclerosis, called the "lipid hypothesis." The lipid hypothesis postulates that (1) a high intake of saturated fat raises blood total cholesterol and (2) high total cholesterol leads to atherosclerosis and CVD, like CHD and stroke. This hypothesis was created more than 100 years ago when it was found that feeding high-fat diets and cholesterol to animals resulted in atherosclerosis [40]. Later it was observed that people with genetically high serum total cholesterol (familial hypercholesterolemia) died from myocardial infarction at young age. Since the 1950s systematic studies laid the basis for the understanding of how different fatty acids influence plasma total cholesterol [41, 42], and a strong positive association was found between intake of saturated fat and serum total cholesterol [43], as well as CHD mortality [44, 45] (Fig. 2.1). In recent years the lipid hypothesis has been debated [46], mainly due to meta-analyses of observational and randomized controlled trials (RCTs) showing no association between intake of saturated fat and CVD [47–51]. However, many of these meta-analyses (except the study of Harcombe et al. 2017 [48]), as well as other recent studies [27, 52–54] have concluded that replacing SFA with PUFA reduces risk of CVD, and hence such replacement still appears to be a useful strategy.



Code	Cohort
A	U.S.Railroad, USA
B	Belgrade, Serbia
C	Crevalcore, Italy
D	Dalmatia, Croatia
E	East Finland
G	Corfu, Greece
K	Crete, Greece
M	Montegiorgio, Italy
N	Zutphen, Netherlands
R	Rome, Italy
S	Slavonia, Croatia
T	Tanushimaru, Japan
U	Ushibuka, Japan
V	Velika Krsna, Serbia
W	West Finland
Z	Zrenjamin, Serbia

Fig. 2.1 Correlation between average energy intake of saturated fat (E%) and number of coronary heart disease (CHD) deaths in the Seven Countries Study after 10 year's follow-ups. (Adapted from [44] with permission from Harvard University Press)

The lipid hypothesis has been modified as the complexity of the atherosclerotic process has become evident. However, although different scientific theories about the causation of CVD have been presented over the years, the lipid hypothesis has not yet been falsified officially, and altering dietary fat intake is still seen as one of the most efficient ways of preventing CVD [25].

2.5.2 The Role of Lipoproteins in Atherogenesis

Dietary lipids are not soluble in water and are transported in the blood as lipoprotein particles. The lipoproteins are commonly divided into four classes according to density: chylomicrons, very low-density lipoprotein (VLDL), low-density lipoprotein (LDL), and high-density lipoprotein (HDL). The chylomicrons have the lowest density and contain about 85% triglycerides and only 2% protein, whereas HDL has the highest density and contains more than 50% protein (Fig. 2.2a). The lipoproteins have different functional and pathological significance, playing different roles in the transport of lipids (Fig. 2.2b). Chylomicrons deliver triacylglycerol (TAG) from intestinal epithelial cells to cells in the body, and VLDL deliver TAG from the liver to non-liver cells in the body. LDL and HDL transport both dietary and endogenous cholesterol in the plasma. LDL is the main transporter of cholesterol and cholesteryl esters and makes up more than half of the total lipoprotein in plasma. LDL is the most cholesterol-rich lipoprotein transporting cholesterol from the liver to peripheral cells (Fig. 2.2a, b). The LDL particle, and in particular the small dense LDL particles, is considered an important pathogenic factor in atherogenesis [55]. At high concentrations LDL can penetrate the arterial wall, possibly after some injury to the endothelial cell layer lining the inner vessel wall [56, 57], where they are

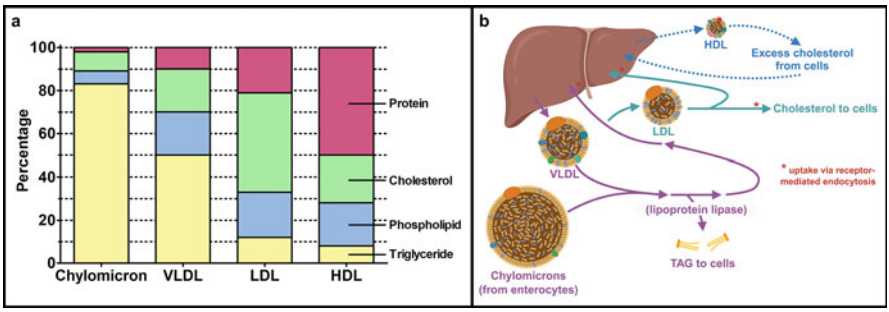


Fig. 2.2 (**a**) The cholesterol content of lipoproteins: 10% in chylomicron, 20% in very low-density lipoprotein (VLDL), 40% in low-density lipoprotein (LDL), 20% in high-density lipoprotein (HDL). Data were obtained from [151] (**b**) Transport of lipoproteins from intestinal epithelial cells to blood circulation and to cells in the body (created with BioRender.com)

modified, for example, by oxidation, and taken up in macrophages which become the so-called foam cells. The foam cells may eventually burst and leave their cholesterol to atheroma (plaque) formation, triggering the inflammatory process. The build-up of fibrous material and calcification inside the arterial walls cause the arteries to narrow and the blood flow may be inhibited. It may also end with rupture and thrombus formation. If this happens in a coronary artery, the result may be a myocardial infarction; if it happens in an artery to the brain, the result may be a stroke.

The HDL particles are supposed to transport cholesterol from peripheral cells and tissues to the liver for excretion [58] (Fig. 2.2b) and has been associated with decreased risk of CHD [59]. However, it has not been documented that altering HDL cholesterol levels by diet or drugs influences the risk of CHD [60, 61]. It appears that the association between HDL cholesterol and risk is complicated [62, 63] as there are multiple factors affecting plasma HDL cholesterol levels other than diet; for example, lack of physical activity and smoking have been associated with low HDL cholesterol. A potential role of the triglyceride-rich lipoprotein chylomicron and VLDL has been debated without reaching consensus [64, 65], although a high concentration of triglycerides is part of the metabolic risk cluster (see Sect. 2.4). Lipoprotein (a) is an LDL-like particle that is independently associated with CHD risk. It is strongly genetically determined and only to a minor degree influenced by diet. However, the primary target for risk reduction by dietary fat should still be LDL cholesterol. Total cholesterol is also considered an expedient target since it is strongly correlated to LDL cholesterol and a good marker of total amount of LDL particle mass.

2.5.3 *Effects of Individual Fatty Acids on Plasma Lipoproteins*

Intervention studies and meta-analyses have provided solid knowledge on the effects of individual dietary fatty acids on serum cholesterol [41, 42, 67–72] (Fig. 2.3). Short and medium-chain SFA (4:0 to 10:0) are considered to have no effect on serum cholesterol [72]. These fatty acids are partly soluble in water and not dependent on incorporation into micelles for uptake in the intestine and follow a different path of metabolism. Also, stearic acid (18:0) is considered to have no effect on serum cholesterol. In most studies lauric acid (12:0) has been found to moderately increase serum cholesterol, but most of this increase may be due to an increase in HDL cholesterol [68]. Myristic acid (14:0) is the most cholesterol-increasing fatty acid and increases both LDL and HDL cholesterol. Palmitic acid (16:0) also increases LDL cholesterol and to a minor extent HDL cholesterol. Trans fatty acids present in partially hydrogenated vegetable and hydrogenated fish oils increase total cholesterol and LDL cholesterol to about the same extent as 16:0 (Fig. 2.3), but in contrast to SFA they decrease HDL cholesterol [68, 73, 74].

A large body of evidence supports the cardioprotective effects of unsaturated fatty acids. The plant derived n-6 and n-3 PUFA, LA and ALA, have been found to reduce serum cholesterol, whereas EPA and DHA of marine origin seem to have minor effect or no effect at all [76–78]. However, EPA and DHA may exert their cardioprotective effects by reducing other risk factors than LDL cholesterol, such as serum triglycerides, blood pressure, platelet aggregation, endothelial function,

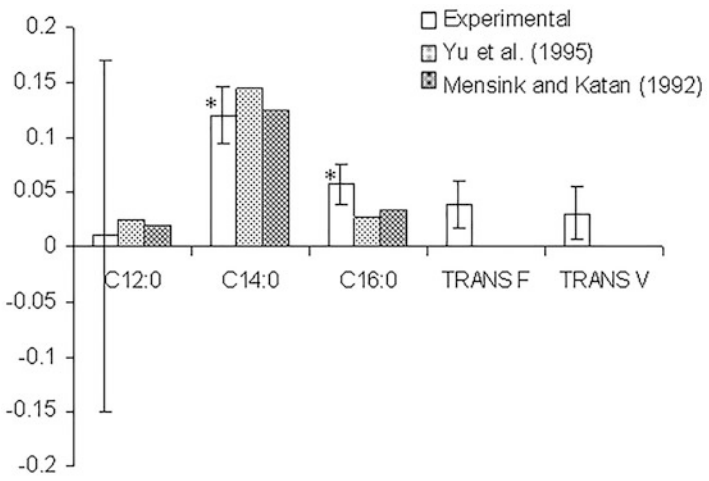


Fig. 2.3 Effect of individual dietary fatty acids on serum cholesterol. Regression coefficients of individual saturated fatty acids (12:0, 14:0, 16:0) and trans fatty acids from partially hydrogenated fish oil (TRANSF) and vegetable oil (TRANSV) expressed as mmol/L serum total cholesterol per E% change in fatty acid intake. (Adapted from [69] with permission from John Wiley and Sons)

and inflammation [35, 36] (see Sect. 2.6.1). Hence, potential detrimental effects of SFA may be counteracted by PUFA, and as observed in many studies replacing SFA with PUFA has beneficial effect on serum cholesterol as well as CVD risk.

2.5.4 *Evidence Linking LDL Cholesterol to Development of Atherosclerosis and CHD*

Animal Experiments
Hypercholesterolemia and atherosclerosis have been induced by feeding cholesterol and fat in virtually every species of laboratory animals [40]. Of special interest are experiments in primates where it has been possible to induce serious atherosclerosis and myocardial infarction after a relatively short time feeding with an ordinary high-fat Western diet [78].

Clinical Observations
The most well-known example of clinical observations is that of familial hypercholesterolemia, a genetic disorder affecting the LDL receptor [79, 80]. The reduced uptake of cholesterol in the liver results in high serum cholesterol levels and development of CHD. Those that are homozygote for the defect may die from myocardial infarction in early childhood [79].

Intervention Studies
Since the 1960s several secondary and primary prevention studies have demonstrated that reducing SFA and increasing PUFA in the diet may decrease serum cholesterol as well as reduce atherosclerotic events [81, 82]. In the Oslo Diet and Smoking Study, a 5-year randomized intervention study including men with elevated serum cholesterol levels, a diet low in total and saturated fat resulted in a 47% reduction in CHD events [83]. It was estimated that most of the effect could be accounted for by reduction in serum cholesterol. A recent follow-up study showed that the difference in mortality between the intervention group and controls was still significant after 40 years [84]. Interventions at national level, often promoted by national nutrition policy programs, provide further evidence of a potential link between LDL cholesterol and development of atherosclerosis. In most Western countries CHD mortality has declined during the last 50 years, and studies suggest that reduction in serum cholesterol may have contributed to about 35% of this decline [85, 86]. In Finland where CHD mortality shifted from being the highest in the world to being among the lowest, reduction in serum cholesterol due to reduced intake of SFA (mainly dairy) and increased intake of PUFA can explain most of the decline [87]. Similarly, in Norway reduction in cholesterol level due to decrease in dietary SFA and trans fatty acids and increase in PUFA can explain a major part of the decline in CHD mortality [88]. Also, interventions with cholesterol-reducing drugs (e.g., statins, bile acid sequestrants) support the hypothesis of serum cholesterol being a causative factor in CVD [89, 90]. A reduction in CHD mortality has been observed for different statins in long-term clinical intervention studies both in individuals with previous CHD and in individuals free of disease at entry [92–94].

Observational Studies (Cohort Studies and Case Control Studies)
Many prospective cohort studies in different parts of the world have identified CVD risk factors such as serum cholesterol, smoking, and high blood pressure. Meta-analyses of such studies have provided evidence that serum LDL cholesterol concentration is strongly and log-linearly associated with a dose-dependent increase in CVD risk [89]. However, although the relative risk associated with serum cholesterol (after correcting for smoking and blood pressure) is the same in different populations, the absolute risk may be very different. As an example, in the 1990s the risk of dying from a myocardial infarction was five times higher in northern Europe than in the Mediterranean region or in Japan at the same serum cholesterol level [45, 94]. This shows that there must be other factors in addition to serum cholesterol that influence CVD risk. Variations in habitual lifestyle could explain some of the discrepancies observed between populations, for example, variations in dietary intake of fruit and vegetables (antioxidants), fish (marine n-3 PUFA EPA and DHA), and physical activity. Dietary antioxidants are believed to have a significant impact, as oxidation of LDL has been related to increased CVD risk in many studies [95, 96]. Other factors that may influence the pathogenesis of CVD are arrhythmia and thrombotic processes [97, 98], stress [99], and inflammation [56, 57, 100]. The role of dietary fat in these processes is, however, not well documented and awaits further studies.

Considering the well-documented effect of SFA on serum cholesterol level in short-term randomized controlled trials (RCTs), it is reasonable to assume that there would be a strong association between intake of SFA and risk of CHD. In the Seven Countries Study [43, 45] as well as in international comparisons [101], this correlation has been very high. However, some recent meta-analyses of prospective cohort studies have failed to find significant associations between intake of SFA and CHD risk [48–51]. A challenge with these studies is the adjustment for confounders [102], as well as difficulties in obtaining reliable data for food intake [103, 104]. The importance of small dense LDL particles that are particularly pathogenic [55] has been put forward as a possible explanation for the lack of association between SFA intake and CHD risk, as many studies claim that saturated fat increases the level of large LDL particles that are much less strongly related to CVD risk. Moreover, the food matrix in which the fat is embedded may have significant impact but is usually not taken into consideration [18]. The lack of association between dietary SFA and serum cholesterol in many of these studies also needs an explanation. Clearly, it can be hard to demonstrate an association between fat intake and serum cholesterol since the biological variability in serum cholesterol is much higher than the changes that can possibly be induced by changes in dietary fat [105]. Furthermore, many of the studies that show no association between total intake of SFA and CHD risk very often show reduced risk when replacing SFA with PUFA, which is in line with published predictive equations and studies showing that the dietary SFA/PUFA ratio is a better predictor of serum cholesterol levels than total intake of SFA [106].

2.6 Saturated Fat and Other Noncommunicable Diseases

2.6.1 Metabolic Syndrome and Type 2 Diabetes

The metabolic syndrome is defined as a cluster of risk factors that predisposes for type 2 diabetes and CVD. These include insulin resistance, impaired blood lipids (e.g., increased triglycerides, decreased HDL), abdominal obesity, inflammation, and hypertension [107]. Some studies suggest that high intakes of total fat and SFA increase the risk of metabolic syndrome and that high intakes of MUFA and PUFA may reduce the risk [108, 109]. Others conclude that there is not sufficient evidence to establish an association between metabolic syndrome and intake of any fat, and that prevention should primarily focus on correcting overweight by reducing the total intake of fat.

When it comes to type 2 diabetes it is well documented that both the quantity and quality of dietary fat influence insulin resistance. Intervention studies have shown that reducing dietary SFA and replacing them with MUFA and PUFA improve insulin sensitivity [110, 111], and it has been discussed whether specific fatty acids, in particular palmitic acid (16:0), may promote insulin resistance more efficiently than other fatty acids [112]. Epidemiological evidence also suggests that replacing SFA with PUFA and MUFA has beneficial effects on insulin sensitivity and is likely to reduce risk of type 2 diabetes [109, 110, 113].

2.6.2 Cancer

The high incidence of certain cancers like colorectal cancer, prostate cancer, and breast cancer in Western countries suggests that lifestyle factors may play an important role. For most cancer types, existing data are not sufficient to conclude on any significant effect of dietary fat [114, 115], although there is some evidence that SFA moderately increase breast cancer risk and that progression of prostate cancer is more rapid on high SFA intake. It was also recently shown that 16:0, but not 18:1 or 18:2, promotes metastasis in oral carcinomas and melanoma in mice [116]. A recent review and meta-analysis on olive oil (high in 18:1) and cancer risk showed that increasing intake of olive oil lowered the risk of overall cancer, including breast cancer and colorectal cancer [117]. It is, however, not known to what extent this was due to changes in dietary fatty acid composition, for example, intake of SFA, or other beneficial components in olive oil.

The World Cancer Research Fund [118, 119] has stated that there is limited, but suggestive evidence that foods containing animal fat high in SFA, such as processed meat, increase colorectal cancer risk. It has been suggested that the association between high dietary fat and colorectal cancer may be due to increased bile acid secretion into the gastrointestinal tract with increased microbial formation of secondary bile acids that are carcinogenic [120]. Human data are scarce, but animal

studies have shown that a diet high in saturated fat increases the gut microbial conversion of primary to secondary bile acids that causes inflammation and may play a role in colorectal cancer. Furthermore, high dietary fat, in particular saturated fat, has been shown to increase the synthesis of taurine conjugated bile acids that promote the growth of the gut bacteria Bilophila, generating H_2S gas which is genotoxic [121].

2.7 Benefits of Replacing Saturated with Unsaturated Fat in Foods

2.7.1 Beneficial Effects on Serum Cholesterol Levels

Healthier food products can be obtained by removing saturated fat and/or adding polyunsaturated liquid oils (vegetable oils, fish oils) to processed products using emulsion technology and oleogels that mimic the function of solid fats in food products. Replacing saturated with unsaturated fat in food products has the potential to lower serum cholesterol. In a recent study, replacing dietary SFA with PUFA showed a significant reduction in serum cholesterol of 8% already after 3 days [122]. It has been shown that if a group of individuals are given the same diet over a certain time, cholesterol levels will be almost normally distributed. The serum cholesterol level, as well as the response to changes in diet, is dependent on our genes. Hence, when we are talking about the effects of fatty acids on serum cholesterol, we are talking about average changes in the cholesterol observed in a group of individuals.

Predictive equations based on how individual fatty acids affect serum cholesterol can be a valuable tool in product development, making it possible to optimize the fatty acid composition of the products [69, 123]. Several predictive equations have been published during the years. They are all based on regression analyses of intervention studies and show similar trends; SFA (12:0, 14:0, 16:0) and trans fatty acids increase serum cholesterol, whereas PUFA (LA, ALA) and MUFA (18:1n-9) are cholesterol-reducing (see Table 2.3).

In the Western diet, myristic acid (14:0) makes up about 10% of the SFA, while palmitic acid (16:0) contributes to more than 50%. Even if 14:0 is more cholesterol increasing (Fig. 2.3), the higher amount of 16:0 makes it the most important cholesterol-increasing fatty acids in the Western diet. Animal fat accounts for the majority of 16:0 today, but palm oil is also an important source. Palm oil is semi-solid at room temperature and fractionated and/or interesterified palm oil is commonly used in industrially processed foods, for example, as a replacement of trans fat, in order to achieve desired sensory quality and product consistency. However, fully hydrogenated and interesterified vegetable oils to increase the content of the "cholesterol neutral" stearic acid (18:0) at the expense of 16:0 may be a better alternative [124].

Table 2.3 Published predictive equations for estimating changes (Δ) in serum cholesterol (Chol) (mmol/L) in response to changes in dietary fatty acids (in E%)

Predictive equations	References
$\Delta Chol = 0.062 \times \Delta SFA - 0.03 \times \Delta PUFA$	Keys 1965 [42]
$\Delta Chol = 0.054 \times \Delta SFA - 0.032 \times \Delta PUFA - 0.003 \times \Delta MUFA$	Hegsted 1965 [41]
$\Delta Chol = 0.056 \times \Delta SFA - 0.016 \times \Delta PUFA - 0.003 \times \Delta MUFA$	Mensink 1992 [67]
$\Delta Chol = 0.052 \times \Delta SFA - 0.001 \times 18:0 - 0.025 \times \Delta PUFA - 0.012 \times \Delta MUFA$	Yu 1995 [71]
$\Delta Chol = 0.052 \times \Delta SFA - 0.026 \times \Delta PUFA - 0.005 \times \Delta MUFA$	Clarke 1997 [66]
$\Delta Chol = 0.01 \times \Delta 12:0 + 0.12 \times \Delta 14:0 + 0.06 \times \Delta 16:0 + 0.03 \times \Delta TFA - 0.017 \times \Delta PUFA - 0.004 \times \Delta MUFA$	Müller 2001 [69]
$\Delta Chol = 0.07 \times \Delta 12:0 + 0.06 \times \Delta 14:0 + 0.04 \times \Delta 16:0 + 0.03 \times \Delta TFA - 0.021 \times \Delta PUFA - 0.006 \times \Delta MUFA$	Sanders 2009 [70]

SFA saturated fatty acids (12:0+14:0+16:0), MUFA monounsaturated fatty acids (18:1), PUFA polyunsaturated fatty acids (18:2+18:3), TFA trans fatty acids ($18:1_{trans}$)

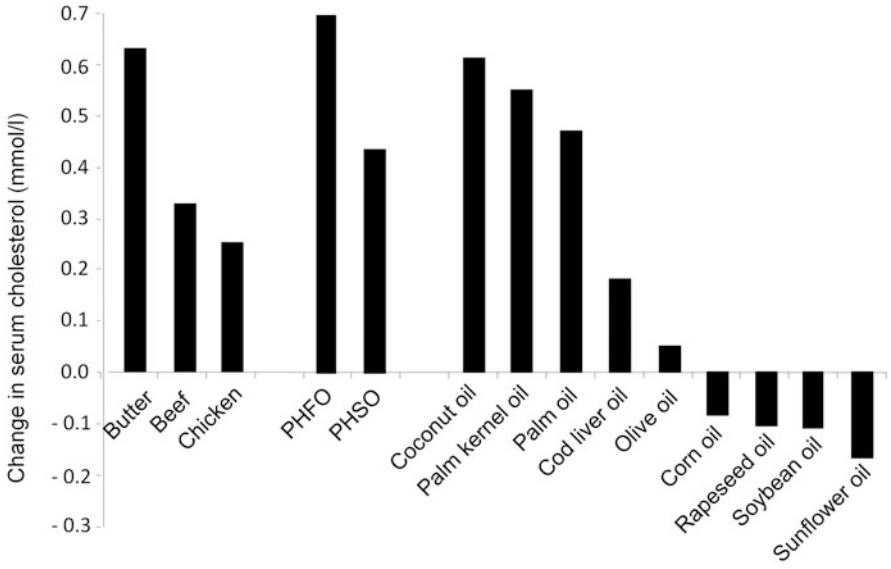


Fig. 2.4 Predicted changes in serum cholesterol (mmol/L) when 20E% carbohydrates (or "neutral" fat) is replaced by the fat in various products, using the predictive equation of [69]. PHFO = partially hydrogenated fish oil, PHSO = partially hydrogenated soybean oil

Figure 2.4 gives an indication of how different fat-containing products and oils affect the level of serum cholesterol using the predictive equation of Müller et al. [69]. Butter, partially hydrogenated oils, refined coconut oil, and palm oil are the most

hypercholesterolemic, whereas oils from soybean, rapeseed, and sunflower are the most cholesterol-reducing. The estimated cholesterol effects are in good agreement with recent meta-analyses that compare effects of butter [125, 126], palm oil [127], coconut oil [128, 129], olive oil [130], and rapeseed oil [131], which taken together show that oils rich in unsaturated fatty acids (e.g., olive oil, rapeseed oil) are preferable to oils and fats rich in saturated fatty acids (e.g., butter, palm oil, coconut oil).

2.7.2 *Benefits of Bioactive Compounds Naturally Occurring in Liquid Oils*

Although the degree of saturation is assumed to be the primary mediator in terms of health effects of dietary fats and oils, it is not the only factor. Liquid oils may also contain health beneficial n-3 PUFA (see Sect. 2.4.1), polar lipids, and micronutrients such as vitamins, phytosterols, polyphenols, and carotenoids that may contribute to an even more favorable nutritional profile.

Vitamins
Vitamin E (α-tocopherol) is commonly found in all vegetable oils. Tocopherols are known to be efficient antioxidants. Sunflower oil has significant higher level of α-tocopherol compared to other commonly used vegetable oils [132]. Soybean oil is particularly high in vitamin K1, whereas fish oils are high in the fat-soluble vitamins A and D.

Phytosterols
It is well documented that phytosterols lower serum cholesterol [133, 134]. The underlying mechanisms for the cholesterol reduction are sterols replacing cholesterol in the micelles, thereby inhibiting cholesterol absorption in the small intestine. Phytosterols are naturally occurring in the cell membranes of plants and may be found in varying amounts (approximately 1%) in vegetable oils depending on the processing conditions used during refining and frying [135, 136]. In recent years advances in food technology have made it possible to add sterols to a variety of food products including margarines, yogurts, fruit juices, and cereal bars. Phytosterols may also be introduced in edible oleogels as building blocks (oleogelators) [137].

Polar Lipids (Phospholipids and Galactolipids)
Polar lipids (phospholipids and galactolipids) are major lipid constituents of plant cell membranes and may be present in small amounts in vegetable oils. Beneficial effects of dietary phospholipids have been proposed since the 1990s in relation to, for example, coronary heart disease, inflammation, or cancer; however, more research is needed to understand the impact of phospholipids supplementation in humans [138]. Oat oil is particularly high in polar lipids (about 15%) and commercial oat oil fractions containing even higher amounts (40%) can be obtained [139]. The main component of polar lipids in oats is the galactolipid digalactosyldiacylglycerol (DGDG) containing one or two fatty acids linked to a

glycerol moiety. Galactolipids have been shown to possess anti-inflammatory and anti-tumor promoting activities [140], and influence fat digestion [141], and when used in food emulsions galactolipids prolong fat digestion and increase satiety [142, 143].

Polyphenols
Polyphenols are natural antioxidants found in fruits, vegetables, and cereals. The biological activity of polyphenols is strongly related to their antioxidant properties. Polyphenols found in virgin olive oil (hydroxytyrosol and oleuropein complex) are well documented to protect against harmful oxidation of LDL [144] and have been associated with lower risk of CVD in some studies [145].

Carotenoids
Edible oils may contain carotenoids with antioxidant capacity. Red palm oil contains high concentrations of beta- and alpha-carotene (approximately 500–800 μg/g) [135] that also have provitamin A activity. Astaxanthin is a powerful antioxidant found in krill oil, but not in most fish oils.

Processing, like heating and refining, have no or minor effect on fatty acid composition, but it may impact the preservation of bioactive compounds in vegetable oils [132, 146]. Cold-pressed oils (virgin oils) are believed to ensure better preservation of bioactive compounds. Hence, depending on the source, raw material quality and processing conditions, edible liquid oils may contain varying levels of compounds that are beneficial to health, and replacing SFA in foods with oleogels containing liquid oils may improve the nutritional profile beyond the expected cholesterol-lowering potential.

2.8 The Impact of the Food Matrix on Nutritional Value

Dietary patterns are composed of foods and nutrients, and this inter-relationship must be acknowledged in dietary research [147]. Based on several recent studies, mass media reports are suggesting that full fat dairy is better for consumers [148]. Rice [149] reviewed 18 epidemiological studies that showed that total dairy intake did not contribute to higher CVD risk, and that consuming milk or fermented dairy products such as yogurt and cheese may reduce CVD risk. The impact of full fat dairy products is difficult to determine, while dairy products contain other compounds that may reduce CVD risk [148]. Dietary studies [18, 150] have shown that despite high content of SFA, dairy fat does not promote atherogenesis in the order of magnitude that could be expected. However, while milk and cheese are associated with a slightly lower CVD risk compared to meat, dairy fat results in a significantly greater CVD risk relative to vegetable sources of fats and unsaturated fatty acids. Whether the food matrix may modify the effect of dairy fat on health outcomes warrants further investigation.

Nutrition research must consider dietary patterns and the complex inter-relationship between dietary components to ensure translation into meaningful and

health-promoting dietary recommendations. Each type of fat has its own unique effects on the body. While most nutrition studies look at effects of individual nutrients, even the same specific type of fat may have different effects and varying nutritional value depending on its origin and the food matrix in which it is embedded.

2.9 Conclusions

Dietary fat is an excellent source of energy and provides essential fatty acids. Moreover, fats and oils provide fat-soluble vitamins and compounds such as carotenoids and sterols. The lipid hypothesis postulates that a high intake of saturated fat raises blood total cholesterol, and that high total cholesterol leads to atherosclerosis, CHD, and stroke. Several predictive equations based on regression analyses of intervention studies have shown that SFA (12:0, 14:0, 16:0) and *trans* fatty acids increase serum cholesterol, whereas LA, ALA, and MUFA (18:1n-9) are cholesterol-reducing. These data provide support for current recommendations to replace SFA with MUFA and PUFA to lower risk of CHD. LA and ALA are essential fatty acids and must be provided in diet. ALA is found in different amounts in plant oils and is a precursor of n-3 PUFA EPA and DHA in mammals. The health benefits of EPA and DHA, provided by marine oils, are well documented, indicating protective effects on CVD, autoimmune, reduction in serum triglycerides, arrhythmia, hypertension, and inflammation. The efficiency of the conversion of ALA to EPA and DHA in humans is low, and it is still debated whether dietary ALA can fulfil the needs of the human body or whether dietary intake of preformed EPA and DHA is necessary.

According to dietary guidelines, healthier food products can be obtained by removing saturated fat and/or adding polyunsaturated liquid oils (vegetable oils, fish oils) to processed foods using emulsion technology and oleogels that mimic the function of solid fats in food products. Fat contributes highly to the food quality (taste, smell, texture, and nutrition) and the choice of oils in oleogels is of high importance for the nutrition quality and health.

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