The Functions of Fats in the Body

Last Updated : 13 February 2015

This part of our *Facts on Fats* review explains in more detail the different functions that dietary fats have in the human body, it covers dietary recommendations on fats from (inter)national authoritative bodies, and discusses to what extent people comply with these recommendations by looking at the current consumption levels throughout Europe. A significant part of this review is dedicated to the current advances in nutrition science on the relation between dietary fat consumption and health outcomes, including obesity and cardiovascular disease. For easier understanding of the current document, written for a somewhat more advanced reader, it may be worthwhile to first read *Functions, Classification and Characteristics of Fats*.

1. Why are dietary fats important?

Functions, Classification and Characteristics of Fats describes the role of fats in taste perception and the importance of fats in a number of food technology applications. From a nutritional point of view, dietary fats are important for several health related aspects and for optimal functioning of the human body. Dietary fats are not just a source of energy; they function as structural building blocks of the body, carry fat-soluble vitamins, are involved in vital physiological processes in the body, and are indispensable for a number of important biological functions including growth and development. The importance of dietary fats is explained in more detail below.

Provision of energy

Fats are a source of energy in the human diet, together with carbohydrates and proteins, the other two main macronutrients. Fat is the most concentrated source providing 9 kcal per 1 gram consumed, which is more than double the energy content of protein or carbohydrate (4 kcal per gram) and more than quadruple the energy content of fibre (2 kcal per gram). Fat can be stored in the body's fat tissue, which releases fatty acids when energy is required (see box: Body fat).

Structural component

The membranes around the cells in our body physically separate the inside from the outside of the cell, and control the movement of substances in and out of the cells. They are mainly made of phospholipids, triglycerides and cholesterol (see *Functions, Classification and Characteristics of Fats*). Both length and saturation of the fatty acids from phospholipids and triglycerides affect the arrangement of the membrane and thereby its fluidity. Shorter chain fatty acids and unsaturated fatty acids are less stiff and less viscous, making the membranes more flexible. This influences a range of important biological functions such as the process of endocytosis in which a cell wraps itself around a particle to allow its uptake.[1]

The brain is very rich in fat (60%) and has a unique fatty acid composition; docosahexaenoic acid (DHA) is the major brain fatty acid. The lipids of the retina also contain very high concentrations of DHA.[2]

Carrier of vitamins

In the diet, fat is a carrier for the fat-soluble vitamins A, D, E and K, and supports their absorption in the intestine. Consuming sufficient amounts of fatty foods that contain these vitamins is thus essential for adequate intake of these micronutrients.

Other biological functions

Our bodies cannot produce the polyunsaturated fatty acids (PUFA) linoleic acid (LA) and alpha linolenic acid (ALA) as described in *Functions, Classification and Characteristics of Fats*. Without these essential fatty acids some vital functions would be compromised, thus they must be provided by the diet. LA and ALA can be converted to longer chain fatty acids and compounds with hormone-like or inflammatory properties (such as prostaglandins or leukotrienes, respectively). As such, essential fatty acids are involved in many physiological processes such as blood clotting, wound healing and inflammation. Although the body is able to convert LA and ALA into the long chain versions arachidonic acid (AA), eicosapentaenoic acid (EPA), and, to a lesser extent, to docosahexaenoic acid (DHA), this conversion seems limited.[3] The longer chain fatty acids EPA and DHA are said to be “conditionally essential” and it is recommended to consume direct sources of these particular long chain fatty acids. The richest source of EPA and DHA is oily fish, including anchovy, salmon, tuna and mackerel. See *Functions, Classification and Characteristics of Fats* for a more complete overview of the most common fatty acids and foods in which they can be found.

Cholesterol
All animal cells contain cholesterol, a lipid that plays a role in the membrane's fluidity and permeability. Cholesterol is also a precursor of vitamin D, adrenal and sex steroid hormones, and bile salts that emulsify and enhance absorption of fats in the intestine.[4] The main dietary sources of cholesterol are cheese, eggs, beef, pork, poultry and (shell) fish. Dietary cholesterol helps to maintain a stable pool of cholesterol, but cholesterol is also synthesised by the liver. The human body regulates its cholesterol status. When the cholesterol intake is very low (as in vegans who consume no animal products), both gut absorption and synthesis increase. When cholesterol intake is high, the body's synthesis is suppressed and excretion via bile salts is increased. The amount of cholesterol, which passes daily through the small intestine, which is the sum of dietary cholesterol and produced cholesterol, is between 1 and 2 g.[2] The average cholesterol intake in Europe is 200-300 mg/day, meaning that the body's production is significantly higher. The blood cholesterol level is the net result of the absorption in the gut and the synthesis in the liver, minus the excretion via the faeces (as cholesterol, bile salts and products resulting from bacterial transformation) and the use of cholesterol by cells.[4] Importantly, for most people, eating foods that contain cholesterol has little effect on blood cholesterol levels (see also the recommendations in section 3). However, a small number of people (15-25% of the population) may be 'hyper-responders' to dietary cholesterol, and are advised to limit their cholesterol intake.[5] Cholesterol in the blood is carried by lipoproteins: LDL (low density lipoprotein) and HDL (high density lipoprotein). How the different levels of these lipoproteins in the blood relate to health will be further explained in section 5.

2. Dietary fat consumption, what are the recommendations?

This section covers the dietary recommendations for fats, issued by different international authorities including the World Health Organization (WHO) and the European Food Safety Authority (EFSA), and national governments and health authorities from a number of European countries. These are being reviewed every few years, and form the basis for the national dietary recommendations and for health related policy actions based on review of the scientific literature, and after consultation with panels of scientific experts.

The extrapolation from the scientific literature to actual dietary recommendations can differ between organisations and/or countries. The reason can be that the recommendations were issued at a later point in time, after newer research findings became available, or that study findings were interpreted slightly differently. One of the challenges is to translate research findings for different health related outcomes, e.g. heart disease, cancer, or death for which different consumption levels being beneficial/harmful, into population based recommendations. On top of that, outcomes from studies cannot always be easily extrapolated because of several reasons, including the selected study population (e.g. diabetic women over the age of 65), the study duration (shorter ones usually produces weaker evidence), or the dose and composition of the intervention (e.g. supplements versus whole foods).

Consequently, converting the outcomes from different studies into one general recommendation that targets the general population is a challenge. Moreover, there is no standardised methodology to define dietary recommendations, and background documentation does not always clearly specify the procedures that were used. More transparency in the evaluation of the scientific evidence used to set recommendations would therefore be desirable.[6] This is being tackled by initiatives for harmonisation, such as the EU funded EURRECA project.[7]

Dietary recommendations for fats

Historically, dietary recommendations focussed on the prevention of nutrient deficiencies. These guidelines are meant to advise people on a healthy diet that ensures adequate intakes of all nutrients. More recently, with higher prevalence of obesity and chronic diseases, nutrition recommendations have shifted to address food overconsumption and prevention of chronic (metabolic) diseases.

Generally, dietary advice for bodyweight management includes controlling total calorie intake, and recommends increasing consumption of lean meat, low-fat dairy, fruit and vegetables, whole grain cereals and fish.[6] For dietary fats, it has been suggested that changing the type of fats consumed (i.e. saturated fat replaced by unsaturated fat), or changing the type in combination with an overall reduction of fat are protective against cardiovascular events.

Tables 1 and 2 provide an overview of the recommendations for adults on the main fats (Table 1) and polyunsaturated fatty acids (Table 2) from a number of national and international authoritative bodies and professional organisations. For reasons described above, these recommendations somewhat differ per organisation/country. It is important to keep in mind that these dietary reference values are derived for population groups and not specifically for individuals. Personal needs may vary depending on a number of personal and lifestyle-related factors.

Energy percent (%E)
%E refers to the percentage of energy, based on the total daily energy recommendations, coming from a specific macronutrient (fat, carbohydrate or protein). For a normal-weight woman/man, with respective daily energy recommendations of 2,000/2,500 kcal, a recommendation of 35%E coming from total fat is equivalent to an intake of approximately 78 g/97 g of fat.

Table 1. Daily recommendations for fat and fatty acids intake for adults according to different bodies - Adapted from Aranceta et al. 2012[6]

Region/ Organisation/ Country	**Total Fat**	**SFA**	**TFA**	**Cholesterol**	**MUFA**	**PUFA**
Europe						
EURODIET, 2000[8]	20-30%E	<10%E	<2%E*			
EFSA, 2010[9]	20-30%E	As low as possible	As low as possible			
Fourth Joint Task Force of the European Society of Cardiology, 2011-2012[10,11]	25-35%E	<7%E	As low as possible from processed food, <1%E from natural sources			
The German Nutrition Society (DGE), 2006[12]	25-35%E	<10%E	<1%E	<300mg/day		7-10%E
UK Committee on Nutrition (COMA, 1991)[13]	35%E	10%E	2%E		12%E	6%E

Region/ Organisation/ Country	Total Fat	SFA	TFA	Cholesterol	MUFA	PUFA
Dutch Health Council, 2006-2011[14,15]	20-40%E	<10%E	<1%E	<300mg/day	(MUFA + PUFA: 8-35%E)	12%E
ANSES, 2011[16]	35-40%E	≤12%E	<2%E		15-20%E	
Conseil Supérieur de la Santé. Belgium, 2009[17]	30-35%E	<10%E	<1%E	<300 mg/day	>10%E	5,3-10%E
Nordic Nutrition Recommendations 2012[18]	25-40%E	<10%E	As low as possible		10-20%E	5-10%E
SENC. Spain, 2011[19]	≤35%E	≤10%E	<1%E	<350 mg/day; <110 mg/1000 kcal	20%E	5%E
North America						
USDA dietary guidelines for Americans, 2010[20]	20-35%E	<10%E (by replacing with MUFA and PUFA)	Avoid consumption of trans-fats from industrial sources	<300 mg/day; <200 mg/day for individuals with or at high risk for CVD and Type 2 diabetes		
Oceania						
NHMRC. Australia & New Zealand, 2013[21]	20-35%E	No more than 10%E from SFA + TFA combined				
International						
FAO/WHO, 2010[22]	20-35%E	10%E	<1%E*		Total fat [%E]–SFA [%E]–PUFA [%E]–TFA [%E];	6-11%E

SFA: Saturated Fatty Acids; TFA: Trans Fatty Acids; MUFA: Monounsaturated Fatty Acids; PUFA: Polyunsaturated Fatty Acids; %E: % Daily Energy Intake;

* TFA definition excludes conjugated linoleic acid

Table 2. Daily recommendations for polyunsaturated fatty acid (PUFA) intakes in adults according to different bodies - Adapted from Aranceta et al. 2012[6]

Region/Organisation/Country	n-6 (LA)	n-3	ALA	EPA	DHA	EPA + DHA
Europe						
EURODIET, 2000[8]	4-8 g/day	Marine	2 g/day			200 mg/day
EFSA, 2010[9]	4%E		0.5%E			250 mg/day
Fourth Joint Task Force of the European Society of Cardiology, 2011-12[10,11]						1 g/day (secondary prevention); 2 portion fatty fish/week
The German Nutrition Society (DGE), 2006[12]	n-6:n-3; ratio 5:1	n-6:n-3; ratio 5:1	1.8-2.0 g/day			250 mg/day
UK Committee on Nutrition (COMA, 1991)[13]	1%E		0.2%E			450 mg/day as two servings of fish/week
Dutch Health Council, 2006-2011[14,15]	2%E		1%E			450 mg/day as two servings of fish/week
ANSES, 2011[16]	4%E		1%E	250 mg/day	250 mg/day	
Conseil Supérieur de la Santé. Belgium, 2009[17]	2%E	1,3-2,0%E	>1%E			>0,3%E (approx. 667 mg/day)
Nordic Nutrition Recommendations 2012[18]	5-9%E	≥1%E				
SENC. Spain, 2011[19]	5%E	1-2%E	1-2%E		200 mg/day	500-1000 mg/day
North America						
USDA Dietary Guidelines for Americans, 2010[20]	5-10%E					Increase the amount and variety of seafood consumed by choosing seafood in place of some meat and poultry
Oceania						
NHMRC. Australia & New Zealand, 2013[21]	4-10%E		0.4-1%E			
International						
FAO/WHO, 2010[22]	2.5-9%E	0.5-2%E	2%E			0.250-2 g/day

LA: linoleic acid; EPA: eicosapentaenoic acid; DHA: docosahexaenoic acid; ALA: alpha-linolenic acid; %E: % Daily Energy Intake.

Total fat

Most authorities recommended intake for total dietary fat in adults ranges between 20-35%E (see box Energy percent). This means that it is advised that 20-35% of the total daily energy intake should come from dietary sources of fats. As described in section 2, fat has many essential biological functions, so the total consumption should not be lower than 15-20%. Moreover, diets that are low in fat (≤20%E) may adversely affect blood lipids by lowering HDL and increasing triglycerides, and may lead to an inadequate intake of essential fatty acids.[6] The upper limit for fat intake aims to ensure that people do not consume too many daily calories as fat, since it is the most energy-dense macronutrient.

The recommendations for total fat intake are further subdivided in advised intakes for the specific fatty acids.[6] Read *Functions, Classification and Characteristics of Fats* for more information on the molecular structure and nomenclature of fatty acids.

Saturated fatty acids

The advice to keep saturated fatty acid (SFA) consumption below 10%E stems from its LDL cholesterol raising potential and effects on cardiovascular disease (CVD) risk. Some guidelines suggest keeping saturated fat intake as low as possible. There is a wide consensus that the most positive effects are seen when saturated fatty acids are replaced by PUFA.[6,23]

Trans fatty acids

The recommendations for trans fatty acids (TFA) are mainly to keep the intake either as low as possible, or below 1%E.[6] It has been convincingly shown that TFA adversely affect blood lipids and increase subsequent CVD risk.[24] In contrast to 10-15 years ago, the vast majority of the food products analysed recently for TFA content in Western Europe, do not contain high levels of TFA, and do not pose a major health risk. Although in some Eastern European countries TFA levels were found to be significantly higher.[25]

Monounsaturated fatty acids

The majority of dietary recommendations do not have specific advice for monounsaturated fatty acids (MUFA).[6] The Food and Agricultural Organization (FAO) indicated that the MUFA recommendation can be obtained with the following calculation: total fat [%E] – SFA [%E] – PUFA [%E] – TFA [%E], with 15 -20%E as a result.[26]

Polyunsaturated fatty acids

Not all (inter)national authorities have specific recommendations for total PUFA, but some do (Tables 1 and 2). Instead, they have set recommendations for the specific fatty acids, including the n-3 fatty acids ALA, EPA, DHA and EPA+DHA, and the n-6 fatty acids LA and in some cases also AA.[6] These recommendations vary considerably among the different countries, organisations, and consumer age groups, and are expressed either in ‘%E’ or in ‘g/day’ (Table 2). The reason for these differences may be because some organisations have focussed on avoiding deficiencies while others have established the recommendations in order to prevent chronic diseases.

Cholesterol

Most authoritative bodies do not provide a maximum amount for cholesterol consumption. When they do, the advice is to not exceed 300 mg/day.[6] The most recent scientific publications point out that in healthy individuals, dietary cholesterol has little impact on blood cholesterol levels (see box Cholesterol).

3. How much dietary fat do we consume?

Monitoring consumption levels of dietary fats in the population, and evaluating to what extent people adhere to the dietary guidelines is important to assess the effectiveness of recommendations.

Global fat consumption

Global food consumption data indicates that the level of total fat consumed is, on average, within the recommended range of 20-35%E. However, there are large country differences with levels ranging from 11.1%E in Bangladesh to significantly higher intakes in Europe, with 46.2%E in Greece.[27] In 2010, data representing 61.8% of the global adult population indicated the mean global SFA intake was less than the recommended maximum of 10%E (9.4%E), with highest intakes being noted in palm oil producing island nations in South-East Asia.[28] In terms of PUFA consumption, between 1990 and 2010 the worldwide intake levels of n-3 PUFA increased, but on average, are still lower than recommended.[28] Again, there are enormous differences between countries; one study, representing 52.4% of the global population, found that the intakes of both seafood and plant n-3 fatty acids ranged from <50 to >700 mg/day and <100 to >3000 mg/day, respectively.[28] Similarly, global intake levels of n-6 PUFA (2.5-8.5%E) are lower than recommended.[28]

European fat consumption

At a European level, food consumption data indicates that the level of total fat intake is generally higher than the recommended 20-35%E (Tables 3 and 4), with maximum intakes ranging from 37%E in the West to 46%E in the South.[26,27] Looking into the specific fatty acids, saturated fat consumption significantly exceeds the recommended maximum of 10%E in all of the regions. The highest consumption is found in the Central Eastern region, with over 25%E in Romania. However, methods for measuring consumption differ among countries, which may partly explain the observed differences. The current intakes of both total and saturated fats have slightly decreased as compared to the previous report in 2004. Intake of PUFA (5-8%E) and MUFA (11-14%E) is lower than recommended. Interestingly, in Mediterranean countries, the intake of MUFA, in accordance with the predominant use of olive oil, is the highest in Europe.[26] Recent action to reduce dietary TFA through means of food reformulation has resulted in a continual decrease in TFA intake, below the recommendation of less than 1%E, across Europe.[9]

Table 3. Intake of energy and macronutrients (min.–max.) in adults in four European regions – Adapted from Elmadfa 1 2009[26]

Region/sex	Energy MJ	Protein %E	Carbohydrates %E	Dietary fibre g	Fat %E
North					
Male	9.2 - 11.1	13.7 - 16.8	42.4 - 51.0	18.0 - 25.0○	31.0 - 44.9

Region/sex	Energy MJ	Protein %E	Carbohydrates %E	Dietary fibre g	Fat %E
Female	6.8 - 8.2	13.7 - 17.2	42.9 - 51.0	15.6 - 21.0○	31.0 - 41.9
South					
Male	9.1 - 10.4	14.1 - 18.5	36.8 - 47.0●	19.3 - 23.5◊	28.4 - 45.0
Female	7.1 - 8.7	14.4 - 19.3	37.7 - 50.1●	16.9 - 23.7◊	29.9 - 47.2
Central / East					
Male	9.0 -13.9	13.5 -17.8	42.5 - 49.5	18.7-29.7^	31.3 - 38.9
Female	7.5 -11.4	13.1 - 17.1	43.6 - 53.9	19.7 - 24.7^	31.2 - 39.7
West					
Male	9.1 - 12.2	14.7 - 16.3	42.4 - 47.6□	12.8 - 24.4&	34.8 - 36.5
Female	6.6 - 8.4	15.6 - 17.0	44.4 - 48.0□	10.4 - 20.1&	35.1 - 36.9

North: SE (Sweden), NO (Norway), FI (Finland), EE (Estonia), LV (Latvia), LT (Lithuania), DK (Denmark); **South**: PT (Portugal), ES (Spain), IT (Italy), GR; **Central and East**: PL (Poland), CZ (Czech Republic), RO (Romania), HU (Hungary), AT (Austria), DE (Germany); **West**: UK (United Kingdom), BE (Belgium), NL (Netherlands), FR (France), IR (Ireland);

○ only SE, NO, FI, EE, LT and DK;

● only PT, ES and GR;

◊ only PT, ES and IT;

^ only PL, CZ, HU, AT and DE;

□ only UK, BE, NL and FR;

& only NL, FR, IR and UK.

Table 4. Intake of fat, fatty acids and cholesterol (min.-max.) in adults in four European regions – Adapted from Elmadfa I 2009[26]

Region/sex	Fat %E	SFA %E	MUFA %E	PUFA %E	Cholesterol mg
North					
Male	31.0 - 44.9	12.0 - 14.6○	11.0 - 16.9○	4.7 - 8.9○	256.0 - 477.9●
Female	31.0 - 41.9	12.0 - 14.4○	10.9 - 15.7○	4.7 - 8.7○	176.0 - 318.8●
South					
Male	28.4 - 45.0	8.8 - 12.7	12.3 - 21.9	4.8 - 6.4	282.9 - 378.4
Female	29.9 - 47.2	9.4 - 13.2	13.0 - 22.9	4.5 - 6.9	227.6 - 310.8
Central / East					
Male	31.3 - 38.9	11.7 - 26.3◊	12.5 - 16.2^	5.7 - 8.8	352.5 - 800.0
Female	31.2 - 39.7	11.7 - 24.8◊	14.0 - 15.0^	5.6 - 9.2	277.0 - 680.0
West					
Male	34.8 - 36.5	13.7 - 14.6	12.8 - 13.3□	6.7 - 7.0□	250.0 - 279.0&
Female	35.1 -36.9	13.7 - 14.7	12.8 - 13.1□	6.7□	201.0 - 215.2&

North: SE, NO, FI, EE, LV, LT, DK; South: PT, ES, IT, GR; Central and East: PL, CZ, RO, HU, AT, DE; West: UK, BE, NL, FR;

○ only SE, NO, FI, EE, LT and DK;

● only SE, NO, FI, EE and LT;

◊ only PL, RO, HU and AT;

^ only PL, HU and AT;

□ only BE and NL;

& only UK and NL.

4. How do dietary fats relate to our health?

This section explains in more detail the science underpinning the dietary recommendations. It provides an overview of the studies related to the consumption of dietary fat and its effect on a number of health related outcomes, but also describes findings from more recent work in the field of nutrition science that need further investigation. Only when a sufficient number of studies on humans consistently show a link between fat (or a specific fatty acid) and health, leading to a consensus between scientific experts, it may be incorporated in actual recommendations.

Although the major non-communicable diseases (NCDs) seem to be interrelated (e.g. CVD and cancer are often attributed to overweight and obesity, and type 2 diabetes affects blood lipids independently of body weight), the following overview of scientific studies is subdivided by disease/health condition.

Obesity

People who are affected by obesity or overweight have an increased risk for developing chronic diseases, such as CVD, metabolic syndrome, type 2 diabetes mellitus and certain types of cancer.[29] Visceral fat that accumulates around the organs in the abdomen is particularly associated with higher risk of developing these diseases. Maintaining a normal body mass index (BMI) and waist circumference, as an indication of a healthy ratio between fat and lean body mass, is therefore important for staying healthy. WHO data from 2014 show that the prevalence of obesity [defined by a BMI over 30 (kg/m2)] worldwide has nearly doubled since 1980, and point to energy imbalance as the fundamental cause. Both physical inactivity and the increased intake of energy-dense foods are explicitly mentioned as an explanation for the global increase of obesity.[29] Since having too much body fat seems harmful, it is reasonable to think that an increased dietary fat consumption is associated with higher body fat levels and a subsequent increased disease risk. But what is the scientific evidence behind this?

When more calories are consumed than used, an imbalance of energy occurs.[30] With time, a sustained imbalance results in an increase of body weight and body fat. While fat contains the most calories per gram, compared to carbohydrates and proteins, there is no scientific evidence that shows an independent role of dietary fat in the development of overweight and obesity. Also, a low-fat diet without total calorie reduction will not lead to weight loss. In other words, a person is unlikely to gain weight on a high fat diet, if the total amount of recommended daily calories is not exceeded and energy expenditure is normal. Furthermore, fat and calorie restriction alone are not sufficient for long-term weight reduction, increased physical activity is also required.[30]

Body fat
There are two types of body fat (or adipose tissue): white (WAT) and brown adipose tissue (BAT).[28] *Adipose tissue* In humans, fat tissue is located under the skin (subcutaneous fat), around the organs (visceral fat), in bone marrow (yellow bone marrow) and in breast tissue. These fat deposits are used to meet energy demands when the body needs it, for normal daily activities, but also when energy requirements are higher such as during high levels of physical activity, pregnancy, lactation, infancy and child growth and in the case of starvation. Although its main function is energy storage, fat tissue is more metabolically active than previously thought. It contains many small blood vessels and fat cells – adipocytes. Adipocytes produce and secrete a broad array of proteins and other molecules such as leptin, adiponectin, tumor necrosis factor-α (TNF-α), and interleukins 6 and 1β (IL-6, IL-1β) that are important for immune responses in host defence and play roles in reproduction (estradiol) and energy/lipid metabolism.[31] Fat deposits also help to insulate the body and cushion and protect vital organs. But, excess body fat, especially visceral fat is associated with insulin resistance, impaired fatty acid metabolism and increased cardiovascular risk. A high accumulation of visceral fat around the organs may lead to the typical 'apple shape' figure. However, it is important to recognise that a person can appear lean and still have a relatively high percentage of body fat.[32] *Brown fat* Whereas WAT is mainly used for energy storage, BAT contains more mitochondria (energy producing cell components) and has the capacity to generate heat by burning triglycerides.[31] Hybernating animals are known to use BAT to keep the adequate body temperature while in resting state. In humans, this specific type of tissue has previously only been known in babies. There are now indications that similar heat-producing cells are also present in human adults, which may be activated through a reduction in body temperature.[33,34] Surrounding temperature therefore influences the energy balance by increasing the energy expenditure. Potential long-term implications for weight management have yet to be investigated.[35]

Blood lipid profile & cardiovascular disease

According to the WHO, CVD is the number one cause of death globally, accounting for 30% of total mortality.[36] In the 1970s, a link between total/saturated fat consumption and the risk for heart disease mortality was established in the Seven Countries' study, which led to nutrition recommendations by several authoritative bodies to reduce saturated and total fat from the diet, to prevent CVD.[6] However, more recent studies in nutrition science point out that an independent relation between fat intake, especially saturated fat, and cardiovascular conditions, has not been consistently shown. In fact, it has become more evident that a replacement of SFA by PUFA reduces the risk for CVD.[6,23]

Research in this area consists mainly of 1) intervention studies in which the effect of a certain diet, e.g. high in saturated fat, on blood lipid levels is examined, and 2) observational studies that investigated the association (not cause-effect relation) between the consumption of dietary fat and the incidence cardiovascular events, e.g. heart attacks or strokes, over a long period of time. An overview of the available scientific studies is described below.

Blood lipid profile

Intervention studies on the effects of fat intake on CVD have mainly studied the effects of a reduction in total/saturated dietary fat (replaced by other nutrients) on the levels of blood lipids. Abnormal blood lipid levels are a risk factor for developing CVD.[37] A higher risk is indicated mainly by high levels of LDL ("bad") cholesterol, but also an increased ratio between LDL (or total) cholesterol and HDL ("good")

cholesterol. This ratio is suggested to be a better marker for CVD risk than LDL alone.[38] In addition, recent research indicates that Apo A1 and Apo B (proteins that are involved in lipid transport in the body) blood levels, and the size of the LDL particle, may be good risk markers for CVD.[39] A smaller LDL particle size is more likely to induce atherosclerosis - the formation of plaques on the inside of blood vessels that increases the risk of blockage and subsequent (cardio) vascular events. An elevated level of blood triglycerides is also linked to a higher risk of CVD.[37]

There is evidence that lowering saturated fat intake has a positive effect on LDL cholesterol and the total/HDL cholesterol ratio, and subsequently on the CVD risk, but only if SFA are replaced by PUFA (both the n-3 and n-6), and not by digestible carbohydrates such as starch or glucose.[22,23,38] In some intervention studies, replacement of saturated fat by digestible carbohydrates has been linked to a more atherogenic blood profile and dyslipidaemia (elevated triglyceride levels, decreased HDL cholesterol and smaller sized LDL particles).[23] Also, replacement of SFA by MUFA shows positive effects on blood pressure and blood lipid profile; but these effects are not as strong as those of PUFA.[23,40] Intervention studies also show that replacing digestible carbohydrates with MUFA has positive effects on raising HDL cholesterol, lowering LDL cholesterol and the total/HDL cholesterol ratio, and may improve insulin sensitivity.[23,41]

The effects of SFA on the blood lipid profile may be further broken down into the effect of individual SFA, as it may vary for fatty acids with different chain lengths. However, there is currently insufficient evidence to link any specific saturated fatty acid to a strong adverse effect on blood lipids or a disease endpoint.[42] SFA with a shorter carbon chain (e.g. lauric acid) do increase LDL cholesterol stronger than the ones with a longer chain, e.g. stearic acid, but at the same time the former also have a higher HDL-raising potential.[38,42] The short-chain fatty acids may show more positive effects on the total/HDL cholesterol ratio.[38] A recent review shows that palmitic acid, the most abundant saturated fatty acid in the diet, seemed to increase LDL cholesterol, but also HDL cholesterol, and has not been shown to increase the risk for CVD.[42] With data on the relation between specific SFA and CVD endpoints lacking, the total body of evidence is insufficient to favour one SFA over another regarding CVD benefits and further research is needed to confirm any differences in health effects between these fatty acids.[42]

The adverse health effects of TFA, have been consistently shown, not only in comparison with PUFA, but also compared to saturated fat, and the effects are not limited to blood lipid levels and CVD.[24,43,44] TFA also induce low-grade inflammation and may, particularly in individuals predisposed to insulin resistance, decrease insulin sensitivity which is related to the development of type 2 diabetes.[45] Limited data show that both industrial and ruminant TFA seem to exert similar effects when consumed in the same amounts, but very rarely people consume high enough amounts of ruminant TFA to be comparable to that from industrial sources.[24]

Cardiovascular disease

Meta-analyses of observational studies, which look at the long-term effects of consumption on the actual disease outcome, indicate that: 1) there is no independent association between the consumption of saturated fat and the risk for CVD, and 2) replacement of saturated fat by PUFA, rather than digestible carbohydrate or MUFA, lowers the risk for CHD.[23,46] A Cochrane review found a reduction of CVD risk in studies of fat modification (i.e. SFA replaced by MUFA/PUFA) when studies lasted at least two years; the reduction was found in men, but not in women.[46] Replacing 5% of energy intake of saturated fat by PUFA, would result in a 10% CVD risk reduction.[41] Similarly, it was estimated that, in populations

consuming a Western-type diet (a diet high in refined grains, fat and sugar, and low in wholegrain)[47], the replacement of 1% of energy from saturated fat with PUFA lowers LDL cholesterol, and is likely to produce a reduction in CHD incidence of 2 to 3%.[40] The European Food Safety Authority (EFSA) Panel on Dietetic Products, Nutrition and Allergies (NDA) has concluded that a cause and effect relationship has been established between the consumption of mixtures of dietary SFA and an increase in blood LDL-cholesterol concentrations, and that replacement of a mixture of SFA with cis-MUFA and/or cis-PUFA in foods or diets on a gram per gram basis reduces LDL cholesterol concentrations.[48] This scientific opinion relates specifically to low fat spreadable fats (margarine). It has been suggested that this may be partly related to the anti-inflammatory properties of n-3 fatty acids.[49] Furthermore, a relationship was found between n-3 fatty acids and a lower total mortality risk, largely attributable to fewer cardiovascular deaths. Individuals with the highest n-3 fatty acid levels lived on average 2.22 years longer, after the age of 65 years.[50] Two recent large studies investigated the effects of n-6 fatty acids on the risks of death[51] and coronary heart disease[52], respectively. They concluded that linoleic acid, the main n-6 fatty acid, lowered the risk for both these endpoints.

There is currently no scientific evidence for a link between individual SFA (e.g. lauric acid, stearic acid or palmitic acid) and CVD risk.[37] For TFA on the other hand, there is scientific consensus about the link between consumption and an increased risk of developing CVD.[24,43,44,53] TFA consumption from ruminant sources, such as dairy and meat, has not been related to disease endpoints, probably because the intake levels from ruminant derived products were significantly lower than from industrial sources when these studies were performed. However, evidence is insufficient to establish whether there is a difference between ruminant and industrial TFA consumed in equivalent amounts on the risk of coronary heart disease.[24,43,44,53]

Type 2 diabetes

The effect of fat consumption per se on the development of type 2 diabetes is not clear, since much of the risk seems to be related to overweight. However, there are some indications that the type of dietary fat can influence where fat accumulates in the body, with SFA leading to more fat around the organs, including liver, which is linked to type 2 diabetes.[54]

Changing the types of fat (PUFA instead of SFA), rather than reducing the total amount of fat in the diet, may also have a positive effect on glucose metabolism.[55] Animal studies have shown improvement of several metabolic factors, including insulin sensitivity, underlying the development of type 2 diabetes when SFA are replaced with PUFA. Insulin sensitivity refers to the capacity of body cells to respond to the hormone insulin, which supports the uptake of glucose, amino acids and fatty acids. A 12-week n-3 PUFA supplementation in people with obesity, insulin resistant children and adolescents, showed positive effects on blood lipids and insulin sensitivity.[56] However, two recent meta-analyses did not find evidence for fish and n-3 fatty acid consumption to lower the risk for type 2 diabetes in humans.[57,58]

There seems to be a relation between insulin resistance and the way the body responds to fat intake.[59] People with insulin resistance respond less favourably to a diet lower in total and saturated fat (aiming to lower CVD risk) than people who respond normally to insulin. Moreover, being insulin resistant is associated with an increased risk for CVD, even at moderate LDL-cholesterol concentrations in the blood.[58]

Inflammation

Chronic low-grade inflammation in fat tissue of individuals affected by obesity has been associated with the pathogenesis of insulin resistance and the development of the so-called metabolic syndrome.[54] What actually causes the inflammation is unknown, but several factors may be involved, including the activation of innate immune processes by SFA. The n-3 fatty acids, EPA and DHA, on the other hand, may have anti-inflammatory properties that modulate adipose tissue inflammation.

A low n-6/n-3 or LA/ALA intake ratio has been proposed to have an anti-inflammatory effect, and therefore to be beneficial for cardiovascular health.[41] However, there is no consensus about this marker, based on the current available evidence and conceptual limitations of the use of this ratio.[60] Losing weight and thereby reducing adiposity seems an efficient strategy to lower inflammation, and improve fatty acid metabolism and insulin sensitivity.[56]

Cancer

Similar to the risk of diabetes, excessive body weight increases the risk of developing different types of cancer, which may explain why in some countries the prevalence for this disease is higher. The current scientific evidence is limited and does not confirm a strong association between total and specific fatty acids intake and development of cancer. The joint initiative of World Cancer Research Fund and American Institute for Cancer Research reported that there is little evidence to suggest a link between total fat intake and breast, lung or colorectal cancers.[61] Whereas emerging evidence suggests that a higher level of n-3 fatty acid consumption may be associated with reduced risk of certain cancers, for n-6, this does not seem to be the case.[61]

Neurological health, cognitive functioning & dementia .

The n-3 fatty acid DHA is an import

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