Nutritional Properties

7.1 Introduction

About 153 million tonnes of 17 commodity oils and fats were produced in 2006/07. It has generally been assumed that around 80% is used for human food (122 million tonnes) but the increasing demand for biodiesel has probably reduced this to a figure close to 115 million tonnes. However, this figure is too high as a measure of fat consumption by reason of loss and waste but also too low because of other fat sources not included in statistical tables for the commodity oils such as cheese and meat. It is not easy to derive a world figure for fat consumed by humans.

Many references in the media suggest that fats are dangerous compounds which should be removed from the diet altogether. *This is completely false.* As with other dietary components, there are appropriate levels of intake and other levels associated with ill health because intakes are too high or too low. Fats and oils are an essential part of the human diet for the following reasons:

- They are the most efficient source of energy.
- They are a source of many bio-active compounds and they contain important acids (essential fatty acids, EFA, see Section 7.2) which animals need but cannot bio-synthesise and must ingest from plant sources.
- They are carriers of important minor components such as fat-soluble vitamins and phytosterols.
- Fats add palatability to our food and contribute flavour and texture.

The body of a lean man of 70 kg is made up of water (~42 kg, 60%), protein and fat (each ~12 kg, 7%), with a balance (including minerals)

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of ~4 kg (6%). In an obese man of 100 kg, 35 kg will be fat. Put differently, of the extra 30 kg, 23 kg is additional fat. A woman's body has less water and more fat (~27%).

Optimum energy intakes have to be related to life style. Some among the very poor have too few calories while many in developed countries have too many for their present low-energy lifestyle, leading to overweight and obesity. When energy is needed it comes first from the limited supplies of glycogen in the body (~1.5 kg), then from stored fat, and ultimately, in cases of severe starvation, from protein. These three major nutrients have average energy levels as indicated: fat (38 kJ/g or 9 kcal/g), carbohydrate (17 kJ/g or 4 kcal/g), and protein (16 kJ/g or 3.8 kcal/g). Clearly, fat provides twice the energy of the other two major nutrients.

Dietary fat supplies come from obvious sources such as butter and spreads and salad oils and less obviously from other dairy products such as cream and cheese, meat and fish, baked goods that contain fat (cakes, biscuits, pastries, breads), chocolate and other confectionery items, and fried foods where frying oil has been added to the fat in the food itself. It is not easy to know how much fat is being consumed but weight and girth can be measured by reference to scales or to waistline measurement. In western countries it is recommended that fat consumption should not exceed 30% of energy requirements but for many persons intake is closer to 35% or even 40%. For a food intake corresponding to 2000 kcal/day fat intakes of 30%, 35%, and 40% require 67, 78, and 89 g/day of fat. In rice-consuming countries dietary customs are different and fat consumption is generally lower. Table 7.1 provides data on fatty acid composition of a small selection of foods. Further information can be gathered from the references provided with the Table.

Table 7.2 provides figures on 'disappearance' in units of kg/person/year over a 20-year period for the main consuming countries/regions. As explained in the footnote to this table disappearance is not the same as dietary consumption. For long, human consumption of oils and fats was believed to average 80% of total usage. With the growing industrial use of these commodities for biodiesel the global average is falling below 80% and is probably now just below 75%. Also this average varies between countries and depends mainly on the size of their oleochemical industry. The traditional oleochemical industry, producing mainly soap and other surface active compounds for personal care and for cleaning, is concentrated in USA, Western

Table 7.1 Levels of total fat and the major types of fatty acids (cited as g/100g of food) in selected foods taken from the publication Gunstone (2004) itself adapted from the original and larger listing of McCance and Widdowson (1998)

Food	Fat content	Saturated	*cis*-Monoun-saturated	Poly omega-6	Poly omega-3	*Trans* Total
White bread	1.9	0.40	0.25	0.62	0.04	0.00
Croissants	26.0	14.33	6.62	1.00	0.41	1.64
Danish pastry	14.1	8.57	1.54	1.65	0.24	0.84
Whole milk	4.0	2.48	0.93	0.10	0.02	0.14
Semi-skimmed	1.7	1.07	0.39	0.05	0.01	0.07
Double cream	53.7	33.39	12.33	1.34	0.48	1.83
Cheddar cheese	32.7	19.25	7.14	0.99	0.28	3.10
Cooking fat	99.5	24.52	31.56	27.06	1.49	10.37
Butter	82.2	52.09	18.48	1.41	0.68	2.87
Spread (70%)	70.0	9.44	31.63	11.73	3.79	10.45
Soybean oil	99.9	15.60	21.20	52.50	7.30	Trace
Olive oil	99.9	14.3	73.00	7.50	0.70	Trace
Lean beef cooked	8.2	3.26	3.41	0.36	0.09	0.28
Chicken roasted	3.7	1.02	1.58	0.60	0.13	0.05
Sausage grilled	19.5	7.69	8.35	1.26	0.15	0.39
Cod raw	0.7	0.13	0.08	0.02	0.26	0
Potato chips	11.0	5.96	2.69	0.16	0.01	0.43
Potato crisps	34.2	14.04	13.51	–[a]	–[a]	–[a]
Peanuts	46.0	8.66	22.03	12.75	0.35	0
Salad cream	31.0	3.29	11.44	13.55	1.00	0.10

[a]Present but not in known amount.

These figures originate in Supplement to McCance and Widdowson's 'The Composition of Foods', Ministry of Agriculture, Fisheries, and Food and The Royal Society of Chemistry, 1998, and are based on 550 results obtained between 1990 and 1997. The analyses are mean values measured on several samples. Full details of fatty acid composition are given in the book and some foods are detailed in the raw and cooked state. Some of the figures for prepared foods will be different from what they were in the 1990s.

Similar information is available from American sources (Hands, 1996 in *Bailey's* and USDA nutrient database <www.nal.usda.gov/fnic/foodcomp>).

Europe, and Japan to which must now be added the oil palm-growing countries of South East Asia, particularly Malaysia. The proportion of available oils and fats used for human consumption will be greater than 75% in countries with little or no oleochemical industry. The table shows how *per capita* consumption has increased over time and how it varies between developed and developing regions of the world. Total demand for dietary fat (and for meat) increases with population and with income and is thus expected to increase

Table 7.2 Changing average 'disappearance' (kg/person/year) over 20 years in the four largest consuming countries/regions

Country	1986	1996	2006/07
World	14.4	16.9	23.0
USA	38.5	45.2	54.3
EU	36.4	43.3	56.7
China	6.3	11.2	21.7
India	7.4	9.3	11.9

Note: The term 'disappearance' relates to the total quantity of 17 commodity oils used for both food and non-food purposes.
Figures for EU relate to EU-15 in 1986 and 1996 and to EU-27 in 2006/07.
Source: *The Revised Oil World 2020 – Supply, Demand and Prices* (2002) and *Oil World Annual 2007* (2007) both published by ISTA Mielke GmbH, Hamburg, Germany.

for many years to come. It is of interest that developed countries like Australia and New Zealand with limited oleochemical industry had disappearance levels of 37.7 and 36.9 kg/person/year, respectively in 2006/07. These values must be close to annual dietary fat consumption in those countries though as already indicated they are too high as a measure of consumption by reason of loss and waste but also too low because of other fat sources not included in statistical tables.

It is not only total fat intake that needs to be controlled but also the nature of that fat, usually expressed in proportions of the type of fatty acids present. Concepts have moved over time partly as a consequence of changing availability of dietary fats such as the shift from animal to vegetable sources, partly as a result of a better understanding of the link between diet and health/disease, and particularly because of concern over cardiovascular disease (CVD). There is a range of risk factors for CVD that include, but is not confined to, dietary fat (Section 7.13).

7.2 EFA and fatty acid metabolism

The fatty acids present in our bodies as triacylglycerols and other lipid classes come from endogenous or exogenous sources. Either we make them in the body (endogenous) from precursors such as acetate (resulting from the catabolism of fats or carbohydrates) or we

get them directly from the fats in our diet (exogenous). In addition, the body is able to modify the structure of fatty acids mainly by chain elongation (adding two carbon atoms through a metabolic cycle) or by desaturation (inserting double bonds). However, there are some acids that humans, in common with other animals, cannot make and these must be dietary in origin and come from plant sources (or from eating the flesh of animals that have already consumed these acids). These are described as EFA. Once ingested, EFA may be metabolised to other physiologically important acids. Since these processes are not always efficient there is debate as to whether the important metabolites themselves should also be considered as EFA.

The major biosynthetic pathways to fatty acids involve three stages:

(1) *De novo* synthesis of palmitic (or other alkanoic) acid from acetate (C_2, a product of carbohydrate metabolism) by reaction with malonate (C_3), itself formed from acetate.
(2) Further chain elongation of saturated or unsaturated acids by one or more two-carbon units.
(3) Desaturation: particularly of stearic acid, first to oleic acid (18:1), and then to linoleic (18:2) and linolenic (18:3) acids.

Further sequences of elongation and desaturation producing discrete families of polyunsaturated fatty acids.

Whether in plants or animals these changes take place in different parts of the cell, under the influence of specific enzymes or enzyme complexes, and require the acids to be in appropriate substrate form.

While animals can produce their own supplies of fats they get these mainly as part of their dietary intake. Much of this exogenous fat will be metabolised through oxidation to produce energy, stored in part as ATP (adenosine triphosphate), but some will be stored as lipid, perhaps after modification. Phospholipid bilayers are fundamental components of all living matter and these must contain particular fatty acids.

7.3 *De novo* synthesis of saturated acids

In plant systems *de novo* synthesis occurs in the plastid and results mainly in the conversion of acetate to palmitate. All 16 carbon atoms in palmitic acid are derived from acetate. In this

pathway acetate (CH_3COOH) and malonate ($HOOCCH_2COOH$) react through a series of steps converting acetate first to butanoate (C_4), then to hexanoate (C_6), and sequentially thereafter, two carbon atoms at a time, to palmitate (C_{16}). At this stage a thioesterase liberates the acyl chain from ACP. The thioesterase is not completely chain-length specific and acids of other chain lengths may be produced. This is obviously true in the lauric oils where the major saturated acid (lauric, 12:0) is accompanied by lower levels of caprylic (8:0), capric (10:0), myristic (14:0), and palmitic acid (16:0).

7.4 Desaturation and elongation in plant systems

The first desaturation of a saturated acyl chain occurs in the plastid. The most common is the conversion of stearate (18:0) to oleate (18:1) and involves the removal of hydrogen atoms from C-9 and C-10 to give a *cis* olefinic bond under the influence of a Δ9 desaturase. The system is oxygen-dependent and involves the reduced form of ferredoxin.

Other saturated acids can be desaturated similarly so there is a group of Δ9 monoene acids such as myristoleic (9*c*-14:1), palmitoleic (9*c*-16:1), oleic (9*c*-18:1), and gadoleic (9*c*-20:1) with each unsaturated acid made by Δ9 desaturation of the corresponding saturated acid.

Elongation by two carbon atoms occurs commonly in fatty acid biosynthesis. It is a variant of *de novo* chain lengthening and occurs with acetyl- or malonyl-CoA or their ACP derivatives. The substrate is any pre-formed saturated or unsaturated acid. For example, erucic (22:1) in high-erucic acid rapeseed oil and nervonic acid (24:1) in honesty seed oil are formed from oleic acid by two and three elongations, respectively. These belong to a family of omega-9 monoene acids.

$$\underset{\text{oleic}}{18{:}1\ (9)} \rightarrow \underset{\text{cetoleic}}{20{:}1\ (11)} \rightarrow \underset{\text{erucic}}{22{:}1\ (13)} \rightarrow \underset{\text{nervonic}}{24{:}1\ (15)}$$

Further desaturation in the cytoplasm converts oleate (18:1 in the form of a phosphatidylcholine) to linoleate (18:2) with a Δ12 desaturase and converts linoleate (as its monogalactosyldiacylglycerol derivative) to linolenate (18:3) with a Δ15 desaturase. *These desaturation steps are confined to plants and do not occur in animals.* They are very important changes as linoleate and linolenate are precursors

of important acids with significant physiological properties. The additional double bonds have *cis* configuration and are in a methylene-interrupted relation to each other. This 1,4 diene unit is characteristic of polyunsaturated fatty acids and is to be distinguished from the 1,3 (conjugated) systems in carotenoids and the 1,5 system in terpenes.

$$-CH=CH(CH_2)_nCH=CH-$$

n = 0 (conjugated 1,3), 1 (methylene-interrupted 1,4), or 2 (bismethylene-interruptrd 1,5)

Though common in animal systems Δ6 desaturase is less apparent in the plant world though not completely absent. It operates in the biosynthesis of γ-linolenic acid (6,9,12-18:3) from linoleate and of stearidonic acid (6,9,12,15-18:4) from α-linolenate. The C_{20} and C_{22} polyenes that characterise animal systems and particularly fish lipids either do not exist in plant systems or are exceedingly rare. The production of important acids such as arachidonic (5,8,11,14–20:4), eicosapentaenoic (5,8,11,14,17–20:5), and docosahexaenoic (4,7,10,13,16,19–22:6) in plant systems is a challenge for plant geneticists. Research has got as far as proof of concept but much remains to be done before an economically viable system is produced.

7.5 Desaturation and elongation in animal systems

Families of polyene acids are produced by a combination of elongation and desaturation processes starting with palmitoleic acid (omega-7 family), oleic (omega-9 family), linoleic (omega-6 family), and linolenic acid (omega-3 family). The acids in each family share a common structural feature viz. the position of the double bond closest to the methyl end of the molecule. These changes are particularly important in animal systems and lead to the long-chain polyunsaturated fatty acids that are of considerable nutritional significance. The changes occurring in mammalian systems are set out in Figure 7.1. The same enzymes are used in each family and there is competition for access to these. The ratio of omega-6 to omega-3 acids required in the diet for optimum health is a matter of present debate (Section 7.10).

Omega-6 family		Omega-3 family	
18:2 (9,12)	linoleic	18:3 (9,12,15)	α-linolenic
↓ Δ 6-desaturase		↓ Δ 6-desaturase	
18:3 (6,9,12)	γ-linolenic	18:4 (6,9,12,15)	stearidonic
↓ elongation		↓ elongation	
20:3 (8,11,14)		20:4 (8,11,14,17)	
↓ Δ 5-desaturase		↓ Δ 5-desaturase	
20:4 (5,8,11,14)	arachidonic	20:5 (5,8,11,14,17)	eicosapentaenoic
		↓ elongation	
		22:5 (7,10,13,16,19)	
		↓ elongation	
		24:5 (9,12,15,18,21)	
		↓ Δ 6-desaturase	
		24:6 (6,9,12,15,18,21)	
		↓ β-oxidation	
		22:6 (4,7,10,13,16,19)	docosahexaenoic

Figure 7.1 The omega-6 and omega-3 families of polyunsaturated fatty acids.

The most significant acids in these sequences are linoleic and arachidonic in the omega-6 family and α-linolenic, eicosapentaenoic (EPA), and docosahexaenoic acids (DHA) in the omega-3 family, The two C_{20} acids are precursors of an important group of eicosanoids including the prostaglandins and leukotrienes. The numbers in parenthesis indicate the positions of the double bonds all of which have the *cis* configuration.

7.6 Antioxidants (see also Section 6.2)

It is widely accepted that for the most part lipid oxidation occurring via reactive free radicals is an undesirable process. In fat-containing foods oxidation leads to unacceptable flavours and the foods are ultimately rejected. *In vivo*, radicals react with proteins and nucleic acids. The consequent changes are linked to several disease conditions and dietary antioxidants are considered to be important. It is for this reason that the presence of antioxidants of different kinds in several dietary components is loudly acclaimed.

It is generally assumed that the higher the concentration of antioxidant the greater the value of that product. But the link between particular antioxidants and good health or disease has not been clearly proved and we are some way from knowing the optimum levels required for human health. Results in the laboratory do not always transfer simply to the more complex reality of human life.

7.7 Cholesterol and phytosterols

Sterols are important minor components of most oils and fats (Section 1.6.). Sources of plant origin contain a range of phytosterols while those coming from animals are rich in cholesterol. Typical levels of the latter for lard (0.4%), beef fat (0.1%), mutton tallow (0.2–0.3%), and butter (0.2–0.4%) are indicated in parenthesis. Eggs contain ~300 mg of cholesterol per egg. The sterol may be present as free sterol or associated with a fatty acid as sterol ester.

The human body contains ~100 g of cholesterol and requires about 1 g of new cholesterol each day. Total cholesterol represents ~0.2% of body weight with one-third in the brain and nervous tissue, one-third in muscular tissue, and the remainder in cell membranes. The daily requirement will be mainly of endogenous origin (600–1000 mg) with the balance from dietary sources (250–500 mg) coming mainly from eggs (~300 mg per egg) and animal fats. Only about one half is absorbed. Phytosterols interfere with the absorption of cholesterol and are being added to spreads to reduce cholesterol uptake.

Cholesterol is an important and necessary compound used *in vivo* for the production of bile salts (emulsifiers) in the liver, steroid hormones (*e.g.* sex hormones) in the adrenal glands, and of vitamin D in the skin.

Elevated cholesterol levels in blood are recognised as one of several risk factors in CVD. This level can be easily measured and is frequently used as an index of cardiovascular health. Levels vary between individuals. They are only slightly influenced by dietary intake of cholesterol and rather more by saturated fatty acids (SFA) and by non-dietary influences. Levels above ~230 mg/100 ml of blood (~6.0 mmol/l) are considered to be undesirable in terms of CVD (Section 7.13) but levels below 160–180 mg/100 ml may lead to risk of non-cardiovascular death.

The possible link between cholesterol oxidation products and coronary heart disease (CHD) and other disease states makes it appropriate to discuss the source and formation of such compounds. Cholesterol (Section 1.5) contains a cyclic double bond (Δ5) and two tertiary carbon atoms in its side chain (C-20 and C-25), all sites where oxidation may occur. Some cholesterol oxides are produced as part of the normal metabolism of cholesterol to bile acids but at higher levels they affect human health by contributing to the development of atherosclerosis. When cholesterol oxides replace cholesterol in the cell membrane they alter its fluidity, permeability, stability, and other properties. Oxidised animal-based foods represent a primary source of oxidised cholesterol. Such products are not present in fresh foods but are formed during handling prior to consumption, mainly through autoxidation (Section 6.2).

7.8 Conjugated linoleic acid

C_{18} acids with two double bonds conjugated with each other were recognised as trace components of milk fat over 50 years ago and have engendered renewed interest in recent years following the recognition of their potential value in the treatment of cancer, obesity, and diabetes.

Conjugated linoleic acid (CLA) has been identified at low levels in milk fat (3–6 mg/g of total fat), butterfat (12–14 mg/g), cheeses (2–20 mg/g), and in lamb and beef meat (4–5 mg/g). Several isomers may be present. The major component (the 9*c*11*t* isomer) is designated rumenic acid because of its formation in the rumen of the cow. It is believed to be a metabolic product resulting from linoleic acid by two linked pathways: isomerisation of linoleic acid (9*c*12*c*-18:2) and Δ9-desaturation of vaccenic acid (11*t*-18:1). The 7*t*9*c* and 10*t*12*c* dienes are also present at lower levels along with many other isomers.

$$\underset{\text{linoleic acid}}{9c\ 12c\text{-}18{:}2} \rightarrow \underset{\text{rumenic acid}}{9c11t\text{-}18{:}2} \rightarrow \underset{\text{vaccenic acid}}{11t\text{-}18{:}1} \rightarrow \underset{\text{rumenic acid}}{9c\ 11t\text{-}18{:}2}$$

CLA can be made in larger volumes and higher concentrations by alkali isomerisation of linoleic-rich vegetable oils such as safflower. The product contains two isomers (9*c*11*t* and 10*t*12*c*-18:2) as virtually the only CLA present along with unreacted palmitic, stearic, and

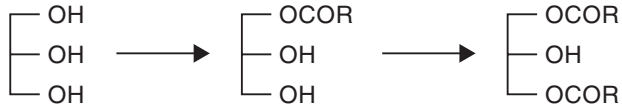
oleic acids from the starting material. These two CLA isomers show different physiological properties. The products of the isomerisation process are free acids and these are generally converted to triacylglycerols before being used in human or animal diets. This can be done enzymatically with lipases such as those from *Mucor miehei* or *Candida antarctica.* Esterification then proceeds under mild conditions without modification of the double-bond systems in the CLA.

Several potential benefits have been claimed for CLA. Dietary supplementation has been shown to reduce the number and size of mammary tumours with the 9*c*11*t* compound probably the more effective isomer. CLA has also been used to alter body composition since it is claimed to reduce body fat, increase lean mass, and improve feed efficiency. These effects are associated particularly with the 10*t*12*c* isomer. Positive results have been obtained with young animals and this is of interest to those concerned with meat production. Conclusions are less certain with humans.

It has also been claimed that in animals CLA affects the immune function and has an effect on bone remodelling. In animal husbandry attempts have been made to increase CLA levels and also to decrease fat production mainly by adjustment of dietary regimen. Increasing CLA levels in poultry meat and in eggs is seen as a potential method of increasing levels of CLA in the human diet.

7.9 Diacyglycerols

A cooking oil rich in diacylglycerols has been available in Japan since 1999. It is made by reaction of glycerol or 1-monoacylglycerol with fatty acids from natural edible oils using a 1,3-regiospecific lipase. The product is at least 80% diacylglycerol of which ~70% is the 1,3-isomer. This figure should be higher but some acyl migration occurs during the refining processes required to obtain the final product. Similar products are now available in the USA and elsewhere.



Acylation of glycerol to produce 1,3-diacylglycerols.

There is also evidence that phytosterols may be more effective in reducing blood cholesterol levels when taken in diacylglycerols rather than in triacylglycerols because of their greater solubility in the former. A diacylglycerol-rich oil with added phytosterols, is sold with permitted claims such as 'it is less likely to become body fat' and 'it lowers blood cholesterol levels'.

The differing physiological effects of di- and triacylglycerols, observed in both animals and humans, are not caused by differences in energy values (38.9 kJ/g and 39.6 kJ/g, respectively) or of absorption of the fates (both 96.3%) but by their different metabolic fats after absorption. 2-Monoacylglycerols, resulting from normal triacylglycerol digestion, are readily reconverted to triacylglycerols in epithelial cells but the 1-monoacylglycerol resulting from the diacylglycerol prepar-ations are only poorly re-esterified. In animal studies the structural differences between di- and tri-acylglycerols markedly affect their nutritional properties including body fat accumulation, serum lipid profile, and the development of hyperinsulinemia and hyperleptinemia.

Double-blind clinical studies in humans have shown that it is possible to reduce the magnitude of postprandial lipemia by consuming diacylglycerols in place of triacylglycerols and therefore the former may be less atherogenic than the latter. Dietary diacylglycerols may therefore reduce the risk of coronary arteriosclerotic diseases by lessening the postprandial increase in the concentration of remnant-like lipoprotein particles.

In contrast to triacylglycerols, diacylglycerols suppress body weight and regional fat deposition, both visceral and hepatic. In a study of obese Americans, decreases in body weight were significantly higher in patients consuming diacylglycerols compared to those on triacylglycerols. It has also been suggested that the consumption of diacylglycerol-rich oil might be useful for maintaining the quality of life of patients with diabetes.

7.10 Recommended intake of fats and of fatty acids

Dietary requirements apply to healthy adults. Different recommendations may be appropriate for those who are not in good health, for babies and children under 5 years, for pregnant and lactating females, and for older people. Dietary advice to the general

population should always be part of a package that includes advice to stop smoking, to take more exercise, to maintain a healthy weight, and to relieve stress. Frequently there is undue emphasis on fat consumption and the other risk factors are overlooked.

Dietary recommendations have been made in most developed countries. These tend to be fairly similar. This is not surprising for it takes a measure of courage (or stupidity) to differ from everyone else. It seems likely that few of those giving the advice have read the original research reports on which the advice is based. Many of these recommendations are based on the need to lower plasma cholesterol levels to reduce CHD. The following statements seek to present an overview of the general recommendations relating to fat. In summary they represent a call to limit the consumption of total fat and of saturated fat and to improve the omega-6/omega-3 ratio (when we can agree on what that should be).

Although humans have the ability to synthesise most (but not all) fatty acids, most of our fat is dietary in origin. Some of this is metabolised to produce energy, some is laid down as adipose tissue, and some finds its way into cell membranes as phospholipids. The fatty acids may be modified before being laid down.

Total fat intake ranges from 15% to 40% energy across most of the world's population. Some have argued that the maximum should be 35% as a practicable first step for those consuming in excess of this figure. The prevalent view is that the maximum should be 30 energy% and that it may be desirable to be below this level.

Prior to World War II most people in developed countries depended mainly on animal fats such as butter, lard, and tallow but since around 1950 this has changed so that present day diets are dominated by fats of plant origin. Nevertheless many still consume a significant portion of their dietary fat from dairy products (milk, cream, butter, cheese) and from meat.

Concern about the growing incidence of CHD in developed countries led to changes as to what was considered to be a healthy fat intake. The first fatty acids to be targeted were the saturated acids. It had been known since the mid-1950s that there is a link between saturated acids, blood cholesterol levels, and CHD and that replacement of dietary saturated acids by mono- or poly-unsaturated acids led to a reduction of cholesterol level and of the incidence of CHD. This accelerated the changeover from animal to vegetable fats and the development of soft spreads containing less saturated fat and more polyunsaturated fat as an alternative to butter.

It is now generally agreed that saturated acids should not exceed one-third of total fat intake giving a figure of 10% in a total of 30% though there are those who argue for lower levels (around 8%). However, this is too simple as all saturated acids do not have the same effect on cholesterol level. Acids with up to 10 carbon atoms in their molecules (present in dairy fats and in lauric oils) are metabolised in a different way from other saturated acids and are not cholesterol-raising. Also, stearic acid is considered to have only a small effect, possibly because it is so easily metabolised to oleic acid through the activity of the ubiquitous Δ9-desaturase. This leaves lauric (12:0), myristic (14:0), and palmitic (16:0) acids. Myristic acid is considered by some to have the greatest cholesterol-raising influence but this is generally a minor dietary component compared with the more common palmitic acid. One factor that has not yet been clearly defined is how far it matters whether these acids are present in the *sn*-2 or *sn*-1/3 positions of triacylglycerol molecules. This factor is not likely to be taken seriously so long as saturated acids are listed as a single class on food labels.

Monounsaturated acids with *cis* configuration are believed to be the safest of all fatty acids. This refers to oleic acid and to the smaller amounts of dietary palmitoleic acid (16:1). Longer-chain acids such as erucic acid (22:1) are considered to be less desirable. This acid was the major fatty acid in traditional rapeseed oil (now called high-erucic rapeseed oil, HEAR) but this has been bred out of canola and other rapeseed oils used for food purposes. HEAR is still produced but mainly for industrial purposes.

In recent years it has been accepted that monounsaturated acids with *trans* unsaturation are undesirable dietary components because of their effect on cholesterol levels. They raise LDL (low-density lipoprotein) levels and lower HDL (high-density lipoprotein) levels and so change the important ratio of these two in an undesirable direction. There are two sources of dietary *trans* acids: dairy (ruminant) fats and partially hydrogenated vegetable oils. Both contain a range of *trans* 18:1 acids but differ in detailed composition. The presence of vaccenic acid (11*t*-18:1) in dairy fats may be of less concern because it is readily converted to 9*c*11*t*-18:2 (rumenic acid, a CLA) which has favourable attributes. The concern about *trans* acids led to labelling requirements in the USA in 2006 with the consequential reformulation of many fat-containing foods to minimise levels of *trans* acids or to have them at a level that allows a claim for zero *trans* ($<$0.5 g per serving of 14 g).

Sometimes this lowering of *trans* is accompanied by a small rise in the level of saturated acids in order to achieve the necessary melting behaviour.

For polyunsaturated fatty acids early recommendations for all acids of this class taken together have been replaced by separate recommendations for omega-6 (linoleic acid and its long-chain metabolites) and omega-3 acids (linolenic acid and its long-chain metabolites). It is now being asked whether there should even be separate targets for C_{18} members and for C_{20} and C_{22} members because, particularly in the omega-3 series, there is concern over the limited ability of animals to convert ALA (itself frequently consumed at less than optimum levels) to the very important EPA and DHA. This is more apparent in males than in females. Particularly because of the high availability of linoleic-rich soybean oil and sunflower oil and the competition that exists between metabolism of linoleic and linolenic acids, there is concern that present omega-6/omega-3 ratios are too high. This is a problem in those countries such as the USA where there is a high consumption of linoleic acid (via soybean oil) and a low consumption of fish (see Chapters in Breivik, 2007).

Dietary intake of omega-6 PUFA will be mainly as linoleic acid and only to a small extent as arachidonic acid (AA) (20:4). It has been suggested that the omega-6 acids should be around 6 energy% with 3–10% representing a safe range but there is a growing opinion that even these levels might be too high. Arguments have been put forward for a more limited intake between 2% and 3%. It has been calculated that with about 20–40% of human body mass being fat and 15–20% of body fat being linoleic acid an adult American will have more than 3 kg of linoleic acid in his tissues. This large reservoir of omega-6 acid may need 3 years to equilibrate with a change in dietary lipids.

For omega-3 acids separate recommendations are now available for linolenic acid and for EPA/DHA. One authority suggests a total intake of 1 energy% (within a range of 0.5–2.5%) for linolenic acid and 0.5 % (within a range of 0–2.0%) for EPA and DHA. Another group have expressed this slightly differently at 1.0% for linolenic acid and 0.3% for the higher acids with a minimum of 0.1% for each of EPA and DHA. A further recommendation is for a minimum daily intake of 450 mg of EPA and DHA. Many food labels indicate levels of omega-3 acids but do not distinguish between 18 and 20/22 acids. This generally means that linolenic acid is the only omega-3 acid present and may confuse the consumer.

The use of the omega-6/omega-3 ratio has been questioned. It is now considered that with the present ratio of between 5 and 10 it is better to be closer to the lower end of this range. Some have argued for ratio levels below 5.

The balance of dietary fat should consist of *cis*-monounsaturated acids (almost entirely oleic acid) and this becomes particularly important when total fat intake exceeds 30%. It is considered that oleic acid should represent 11–16% of dietary intake but should not exceed this upper limit.

Because DHA is such an important component of the brain appropriate supplies of this acid are required by a developing foetus and are supplied by the mother via the placental cord. This need continues after birth and should be met through human breast milk that, in a well-nourished mother, will contain adequate levels of DHA. Problems arise when the child is not breast-fed and, more significantly, in pre-term infants since brain development is particularly marked in the final trimester before birth. It is now recommended that during pregnancy and lactation dietary fat intake should be as for other adults except that the intake of DHA should be at a minimum level of 300 mg/day. Infant formula for Western countries, expressed as percentage of fatty acids, should contain linoleic (10.0%), α-linolenic (1.50%), arachidonic (0.50%), DHA (0.35%), and EPA ($<$0.10%). Slightly different levels are used in Japan with linoleic acid at 6–10% and DHA at 0.6%. The American Heart Association now recommends that patients with known CHD consume $\sim$1 g/day of EPA and DHA. For those without known CHD the recommendation is for at least two servings of fish (preferably oily fish) each week – equivalent to 500 mg/day. Other recommendations range from 200 to 650 mg/day for the general population, with higher levels up to 1000 mg/day for those at risk of CVD. A recent UK proposal is for 450–1800 mg/day.

7.11 Role of fats in health and disease

Modern medicine and modern standards of hygiene have freed the world, or at least many parts of it, from the killer diseases of previous centuries such as tuberculosis, smallpox, and diphtheria and we inhabit a world where increasing numbers live healthier and longer lives. This has highlighted the diseases that remain and are

killers, not only of the old, but of men and women in their prime, particularly CHD and cancer. We are increasingly aware that many diseases that remain, whether killers or not, are related in some part to lifestyle of which diet, pollution of the environment, and level of physical activity are all important factors.

The following sections are related to the role of fat in some disease conditions. However, it is important to realise that fat is only part of our diet and that diet is only part of the problem and of the solution. It is inadvisable to focus on a single issue and ignore others. Fat has a very negative image at the present time and we need to correct that. Fat is an essential part of the diet and is linked to good health as well as to disease. It is important to optimise the quality and the quantity of fat consumed in relation to other aspects of lifestyle. This implies: that we know what fats we should consume and in what quantity, that agriculture and the food industry can supply these, and finally that we can persuade human populations to choose healthy and affordable diets. We must recognise that we are a long way from discovering the final truth in respect of dietary fat and that what is written in the following sections simply represents present views that may have to be modified in the light of new and further research. This is one of those areas of life in which 'time makes ancient good uncouth'.

7.12 Obesity

Body mass index (BMI) is used as a measure of weight to height ratio and allows us to recognise four categories of body sizes. The BMI is defined as weight (expressed in kilogram) divided by height squared (expressed in centimetre) and one classification is:

- Underweight <18.4
- Normal 18.5–24.9
- Overweight 25.0–29.9
- Obese 30.0–39.9
- Severely obese >40.0

A growing number of persons fall into the last three categories, probably as a consequence of imbalance over many years between increased caloric intake and decreased energy requirement resulting

from more sedentary and less active lifestyles. An alternative measure considers the distribution of adipose tissue expressed in a waist to hip ratio. In tackling this problem attention is focussed on fat because it is the most energy dense of our nutrients. It is worth noting that the human race has developed over evolutionary time in a situation where lack of food was a common occurrence and surplus food, over anything but short periods of time, was virtually unknown except for a few rich people. The human system did not need to be designed to deal with the problem of over-consumption.

The problem is not new, even if it is becoming more widespread. Shakespeare has Henry tell Falstaff 'Leave gourmandising. Know that the grave doth gape thrice wider for thee than for other men'.

The problem of obesity is partly genetic and partly environmental (food intake and physical inactivity). Attention is often focussed on long hours spent TV watching where inactivity is often accompanied by poor eating habits. In the USA nearly two-thirds of the population is overweight or obese and almost 40% are clinically obese. Concern is growing about the increase of obesity in children and adolescents.

In 1991 deaths associated with obesity in the USA (300 thousand) were second only to deaths associated with smoking (400 thousand) and it is likely that in the intervening years these numbers have become closer. Obesity is a potent risk factor for type-2 diabetes, hypertension, and dyslipidemia. The average US person seeking treatment for obesity weighs around 100 kg. However, it is worth noting that while fat intake in the USA fell on the basis of percentage of energy from ~42% in the 1960s to ~35% in 1990 fat intake in terms of gram per day fell between the 1960s and the 1980s then stabilised and began to increase in the mid-1990s.

In Europe also, obesity figures are on the increase and it is reported that 20% of men and 25% of women in the UK are obese, that these levels have tripled in the last 20 years, and that 9000 deaths a year are associated with this condition at a cost of £2.5 billion. Obesity is a major public health problem throughout Europe, especially among women in Southern and Eastern European countries. These countries are also among the highest for CVD. In Europe the treatment of obesity-related diseases accounts for 8% of all medical costs.

There are factors other than dietary fat that are important in obesity and attention has been drawn to the beneficial role of dietary calcium in the partitioning of dietary energy, resulting in reduction in body fat and acceleration of weight loss and fat loss during periods

of energy restriction. Dairy sources of calcium exert substantially greater effects than supplemental or fortified sources of calcium. This is considered to have important implications in the prevention of paediatric and adult obesity particularly in the light of the marginal calcium intakes exhibited by the majority of the population.

In discussing diet, obesity, and cardiovascular risk Bonow and Eckel (2003) have written: 'The recipe for effective weight loss is a combination of motivation, physical activity, and caloric restriction; maintenance of weight loss is a balance between caloric intake and physical activity with lifelong adherence. For society as a whole prevention of weight gain is the first step in curbing the increasing epidemic of overweight and obesity … physicians should recommend a healthy lifestyle that includes regular physical activity and a balanced diet'. For dietary fat they recommend: total fat 33 energy%, saturated acids 10%, polyunsaturated fatty acids 6% (and not exceeding 10%), *cis*- monounsaturated acids 12%, and *trans* unsaturated acids $<2\%$.

7.13 Coronary heart disease

It has long been known that the blood system is important as a means of moving oxygen and nutrients from cell to cell and from organ to organ and as part of the system by which animals dispose of waste products. This system depends on a complex series of channels of wide-ranging diameter through which blood flows and through which gases and liquids pass from cell to blood stream and vice versa, and of a pump to force the necessary movement. All these have to last for life and any fault with the pump (heart) or the channels (veins and capillaries) will cause difficulty.

CVD is a broad-term embracing diseases of the blood vessels of the heart (CHD), brain (cerebrovascular disease, stroke), and the limbs (peripheral vascular disease). CVD is usually a culmination of atherosclerosis (accumulation of material – plaque – in the walls of arteries of cells comprising connective tissue, lipids, calcium, and debris resulting from cellular breakdown) and thrombosis.

CHD is a major cause of death in the developed world with a peak age of death of 70–74 for men and 75–79 for women but its too-common occurrence at an earlier stage in life is of greater concern. There are three stages in the development of CHD. Initial arterial injury leads to deposition of lipid and cell material (atherosclerosis)

and to small blood clots (thrombi) which contribute to the build up of fibrous plaque. Finally, instability of the plaque triggers formation of a major blood clot (thrombus) in the already-narrowed artery. This gives the potential for the blood (and oxygen) supply to the heart muscle to be blocked completely leading to myocardial infarction (heart attack). More simply: the three stages are injury of coronary arteries, fibrous plaque formation, and thrombosis leading to heart attack or stroke. The following have been recognised as risk factors: high blood pressure, high levels of plasma LDL cholesterol, low levels of plasma HDL cholesterol, high levels of plasma fibrinogen, and low levels of plasma antioxidants. These risk factors are linked to a range of controllable and uncontrollable factors. The uncontrollable factors are family history, being male, advancing age, racial origin (Asians show higher rates of incidence than white Caucasians), and possibly low birth weight. Controllable factors include smoking, lack of exercise, stress, and diet. Serum cholesterol level should be below 230 mg/100 ml but very low cholesterol levels (below ~160/180 mg/ml) are also undesirable.

The lipid hypothesis in respect of CHD is concerned with the relationship of blood cholesterol and SFA with CHD mortality. Diets with a high content of fat/SFA/cholesterol lead to high concentrations of total cholesterol in the blood and especially of LDL-cholesterol which results in a high morbidity and mortality from CHD. However, reducing the amount of fat/SAF/cholesterol in the diet reduces the concentration of cholesterol in the blood and especially in the LDL and this results in a lower risk of CHD and eventually a fall in morbidity and mortality. There remains, however, the question as to how far reduced CHD mortality is linked to dietary changes and how far it is related to improved methods of medical treatment.

This hypothesis is the basis of much dietary advice relating to fat consumption though it should be noted that there are those who dispute this proposal and have mounted strong arguments against it (Gurr, 1999). Once the link between cholesterol blood levels and fatty acid intake was accepted it became apparent that blood cholesterol levels could be raised or lowered by dietary changes in fatty acid composition and attempts were made to express these changes in the form of mathematical equations. These predictive equations are related to changes in cholesterol levels and not to absolute values. The earliest of these equations (Keys, 1957) covered only saturated and polyunsaturated acids (expressed as percentage of energy) but later versions include *cis*-monounsaturated

acids, *trans* monounsaturated acids, and some even distinguished between individual fatty acids.

The Keys equation of 1957 showed that a rise in saturated acids led to a rise in cholesterol levels, that polyunsaturated acids had the opposite effect, and that monounsaturated acids were considered to be neutral. This conclusion led to the development of spreads with reduced content of saturated acids and increased levels of linoleic acid, often based on sunflower oil. The coefficients in the equations indicate that the beneficial effect of polyunsaturated acid is only one half the undesirable effect produced by raising the level of saturated acids. More recent equations distinguish three saturated acids and ignore those of shorter- or longer-chain length. Lauric acid has only a minor effect and myristic is greater than palmitic acid. *trans*-Acids also raise cholesterol levels with a slightly greater effect for those from partially hydrogenated fish oils than for those from partially hydrogenated vegetable oils. In a survey of five recent equations the regression coefficients for total cholesterol levels are saturated acids (+0.56 to +0.50), *trans* acids (+0.31 to +0.39), and for polyunsaturated acids (−0.15 to −0.31). The cholesterol-raising effects of the saturated acids are greater than the cholesterol-lowering effects of the polyunsaturated fatty acids.

These equations relate to fatty acids present in the diet as glycerol esters and this requires another factor to be taken into account since there is good evidence that SFA are more atherogenic in the 2-position. For example, tallow and lard both contain about 24% of palmitic acid but differ in that in lard almost all the palmitic acid is in the 2-position. Lard is much more atherogenic than tallow but after interesterification (randomisation) both fats with about 8% of palmitic in the 2-position are equally atherogenic. This effect may be related to the fact that palmitic acid in the 2-position is absorbed more efficiently. Similar effects have been observed with synthetic triacylglycerols and with appropriate vegetable oils.

One study of CHD concludes that fats in the diet should not exceed 33 energy% with saturated acids (10%), polyunsaturated fatty acids (6% and not exceeding 10%), monounsaturated fatty acids (12%), and *trans* acids (<2%) at the levels indicated. This is accompanied by advice to eat more fruit and vegetables, more starch foods, more oily fish, and less full-fat dairy products, fatty meat and meat products, spreadable fats, and high-fat bakery products, to choose low-fat options where possible, and to use less salt. Dietary advice to the general population should always be part of

a package that includes advice to stop smoking, to take more exercise, to maintain a healthy weight, and to relieve stress.

The evidence for a beneficial role for long-chain omega-3 polyunsaturated fatty acids is becoming stronger, especially for secondary prevention. Intakes of 800–1000 mg/day are considered to be prudent for those at risk of a secondary attack. At the same time, a high intake of linoleic acid should be discouraged because of its antagonistic effect on the incorporation of omega-3 acids into membranes. The most that can be claimed is that 'consumption of long-chain omega-3 acids may reduce the risk of CHD'. EPA-derived leukotrienes have less potent leukocyte activating effects than AA-derived leukotrienes and at least part of the anti-atherogenic mechanism of the omega-3 acids is likely to be due to their effect on eicosanoid metabolism.

7.14 Diabetes

Diabetes mellitus is a chronic disease in which the metabolism of sugars (and of fats and proteins) is disturbed by a lack of the hormone insulin produced by the endocrine part of the pancreas or by its decreased activity. The main symptom is an increase in the level of blood sugar provoking acute symptoms such as thirst, frequent voiding, and weight loss. The incidence of this disease is increasing all over the world and it is predicted that it will affect 210 million people by 2010. Diabetes is an independent risk factor for CVD.

Type-1 diabetes, representing only about 15% of cases, is found particularly in children, adolescents, and young adults. It results from auto-immune destruction of the insulin-secreting cells of the pancreas. Production of insulin declines and eventually ceases. However, most diabetic individuals (85%) have type-2 diabetes. Two dysfunctions are involved: decreased insulin secretion after a glucose challenge and a decrease in its activity on target organs (liver and muscles). This is called insulin resistance. Obesity is a major pre-disposing factor of this type of diabetes which is largely determined by genetic factors. The metabolic consequences of this defect may not be apparent until the appearance of chronic complications.

One discussion on the nutritional management of this disease suggests that individuals with normal body weight and normal lipid levels should limit fat intake to less than 30% total energy with

SFA restricted to 10%, polyunsaturated acids to less than 10%, and monounsaturated acids at 10–15%. Those with elevated LDL levels should reduce saturated acids to 7% and cholesterol intake to less than 200 mg/day. However, omega-3 polyunsaturated fatty acids should not be restricted. Other aspects of nutritional management of this disease are not included here.

It is known that the various desaturases involved in the conversion of C_{18} polyunsaturated fatty acids to the important acids of longer-chain length such as arachidonic acid, EPA, and DHA are decreased in diabetic patients. As a consequence the phospholipids in tissue lipids contain more saturated and monounsaturated acids and less LCPUFA, especially AA. This, in turn, affects membrane fluidity and eicosanoid production.

7.15 Inflammatory diseases

Inflammation is characterised by swelling, redness, pain, and heat in localised areas of the body. These symptoms result from a series of interactions between cells of the target tissue, cells of the immune system, their products such as eicosanoids, cytokines, and immunoglobulins, and blood components.

Polyunsaturated fatty acids of the omega-3 series act directly by inhibiting AA metabolism and indirectly by altering the expression of inflammatory genes. They are considered to be of therapeutic value for a variety of acute and chronic inflammatory conditions. But what is the appropriate balance between omega-6 and omega-3 acids? This may differ at different parts of the life cycle such as early development and ageing and has yet to be determined.

7.16 Psychiatric disorders

In view of the importance of brain phospholipids and their component acids it is not surprising that the relation between dietary lipids and psychiatric disorders such as schizophrenia and depression have been investigated. Schizophrenic patients are known to have lower levels of polyunsaturated fatty acids (especially linoleic and arachidonic) in their brain phospholipids. This may be the consequence

of increased phospholipid hydrolysis and/or decreased incorporation. The role of omega-3 acids and especially EPA is under active consideration.

7.17 Cancer

The possibility of a link between cancer and dietary fat has received intensive study but no consensus has emerged. Such studies are complicated by the fact that cancers in different organs may react differently to dietary fats. Much attention has been given to the possible beneficial treatment of breast cancer with CLA (Section 7.8) but there is still no final conclusion.