# 17 Lipids


## Summary

Man consumes a wide variety of fats (lipids). Fatty acids are part of triglyceride, phospholipid and cholesterol ester molecules; they can be saturated, monounsaturated or polyunsaturated. Certain polyunsaturated fatty acids are essential in man's diet since he needs them and cannot make them. Essential fatty acids are of two types, the linoleic series, from principally vegetable sources, and the linolenic series, from principally marine sources.

Excessive consumption of fatty foods, along with other energy dense foods, has contributed to the prevalence of obesity in Australia. Dietary saturated fats and cholesterol raise and polyunsaturated fats lower the blood cholesterol concentration, which is an important risk factor for atherosclerotic vascular disease. There are some indications that control of this risk factor can reduce the risk of ischaemic heart disease in which there is a reduction of blood flow to the heart.


## Nutritional significance

Omnivorous: feeding on foods of animal and botanical origin.

Monogastric: having only one stomach, in contrast to a ruminant animal, which chews its cud back from the first stomach. Monogastric animals include man, pigs, chickens and rats.

Ruminant: a ruminant animal has a stomach consisting of four parts; the rumen, the reticulum, the psalterium and the abomasum. Ruminants include sheep and cattle.

Man is omnivorous* and, therefore, has a wide range of sources of fat, from both animal and plant sources. In historical terms, the availability of fat (lipids) from animal sources has progressively increased with domestication of animals. Wild animals were generally more lean and less readily available. For a monogastric animal*, the kind of fat in the carcass depends partly on the fat ingested, but for ruminants,* like sheep or cattle, fat is broken down into small units that are re-synthesized (rebuilt) to form saturated rather than polyunsaturated fat. Meats from ruminants contain a greater percentage of saturated than polyunsaturated fatty acids. Many of the pressures on animal production have meant that fattening is an end in itself so that, for example, chicken, once regarded as a lean meat, is now often 'fatty'.

Fat is the most concentrated dietary energy source, with 37 kilojoules per gram (9 kilocalories per gram), and, where energy intake has been the principal concern in man's food intake, fat has often allowed this need to be readily met. Perhaps this partly explains a preference for fat that has been observed in hunter-gatherer societies.

There is a great variation in the relative proportions of fat, carbohydrate, protein and alcohol as energy sources in the diets of people from one geographical locality to another. Fat does provide potential for flexibility in man's diet, but it is important to understand the consequences of a diet relatively high in fat and the extent to which fats might be essential in our diets.

To some extent, the tolerance we might have for a wide range of dietary intakes can reflect the level of physical activity we achieve. The greater the level of physical activity the greater the energy requirement. Fat, like carbohydrate and protein, can help us meet those energy needs. The Masai in Africa, for example, have a high fat intake that appears to have no ill effects by way of increasing the chances of heart disease; they also have a particularly high level of physical activity.

The relationships between appetite and fat intake are summarized in table 17.1. There are properties of fat that may increase appetite and others that may decrease it.

Table 17.1 Characteristics of lipids that affect appetite

| *Appetite increase* | *Appetite decrease* |
|---|---|
| *By good appearance and texture.* | *By a delay in gastric acid secretion and gastric emptying.* |
| *By good aroma and taste, because of lipid-soluble organoleptics.** | *By a greater propensity for ketones to form with relatively less carbohydrate.* |

Organoleptic properties of a food: those that affect the senses of taste, smell and sight.

## Chemistry of fats

Fats, or lipids, are characterized by their solubility in organic solvents such as alcohol, ether, chloroform and methanol and their negligible solubility in water. Fats (lipids) include the following:

1. free fatty acids;
2. triglycerides;
3. sterols: cholesterol, cholesterol ester; and
4. phospholipids.

Like other organic compounds, fats are made up of carbon, hydrogen, and oxygen atoms. In the case of phospholipid, the phosphorous atom is also found in the molecule.

### Free fatty acids

Free fatty acids (FFA) are so named because they are not associated or esterified* with other molecules, such as glycerol or cholesterol. When they are esterified they constitute part of the triglyceride molecule, the phospholipid molecule or the cholesterol ester molecule.

Esterified: the linkage between fatty acid and an hydroxyl group as in glycerol or cholesterol is an ester linkage.

Fatty acids are generally chains of carbon atoms joined together with three hydrogen atoms at one end (the methyl end) and two oxygens and a hydrogen at the other end (the carboxyl

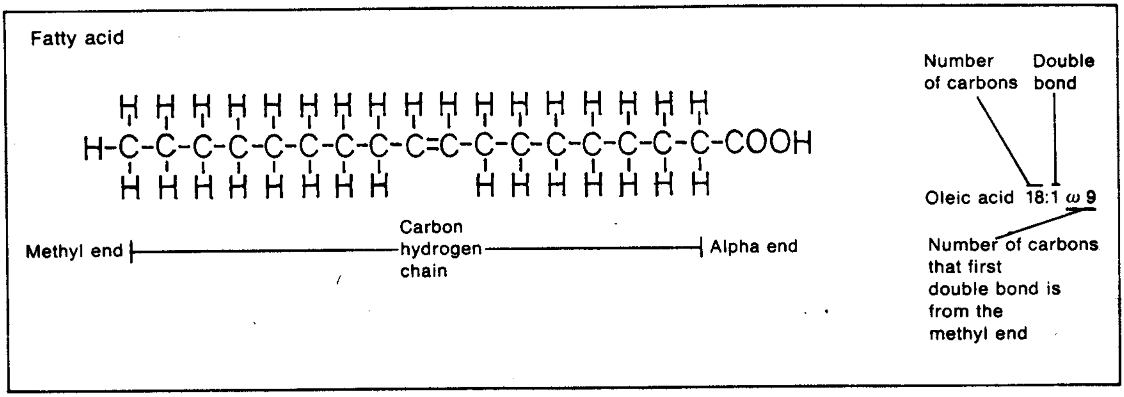



*Figure 17.1 The chemical structure of a fatty acid*

end). Fatty acids can be saturated, mono-unsaturated, or polyunsaturated. The term saturated means that there are no double bonds in the molecule, mono-unsaturated means that there is one double bond in the fatty acid molecule and polyunsaturated means that there are two or more double bonds in the molecule. A double bond differs from a single bond between two carbon atoms in that a bond that would have linked a hydrogen to each carbon atom forms an additional link between those carbon atoms.

Each fatty acid has its own name. For example, palmitic acid is a saturated fatty acid with sixteen carbon atoms; oleic acid is a mono-unsaturated fatty acid with eighteen carbon atoms; and linoleic acid is a polyunsaturated fatty acid also with eighteen carbon atoms.

## Triglycerides

Triglyceride is formed through the esterification or combination of three fatty acid molecules with glycerol. Most fatty acids in foodstuffs are bound to glycerol as triglycerides. Triglycerides are often spoken of as saturated or polyunsaturated. This means that the fatty acids from which triglyceride has been made are more of the saturated kind or more of the polyunsaturated kind.

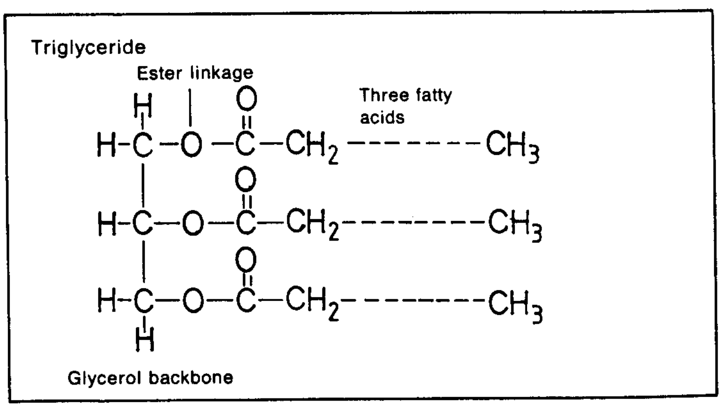



*Figure 17.2 The chemical structure of triglyceride*

## Sterols

The sterol of most interest to human nutritionists is cholesterol. It is not found in plant tissues but in animal tissues. It is a structure with four rings and a side chain.

At one part of the ring it is possible for an ester linkage to be created by the combination of cholesterol with fatty acid. So there are saturated, mono-unsaturated and polyunsaturated cholesterol esters because of the variety of fatty acids with which cholesterol may be complexed. When cholesterol is not esterified it is spoken of as 'free' or 'unesterified' cholesterol.

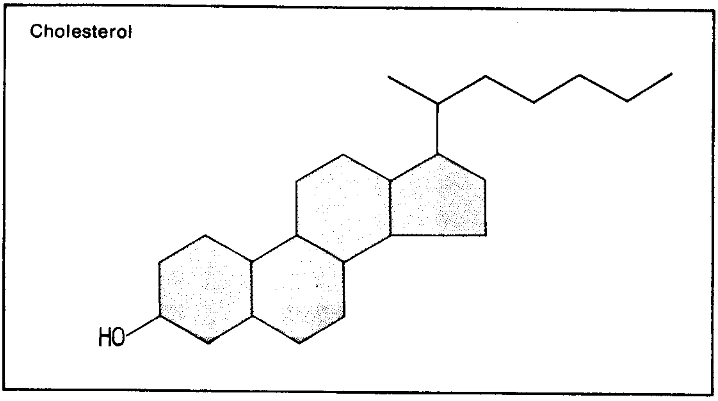



*Figure 17.3 The chemical structure of cholesterol*

## Phospholipids

Another class of lipids is the phospholipids. They are a heterogeneous group of fats including:

1. lecithin (phosphatidyl choline),
2. lysolecithin,
3. phosphatidyl ethanolamine,
4. phosphatidyl inositol, and
5. sphingomyelin.

Lecithin is found in plant and animal products. In general, when it is found in plant products, it is polyunsaturated, which means that the fatty acids that have contributed to the phospholipid molecule are polyunsaturated. Lecithin from animal tissues is

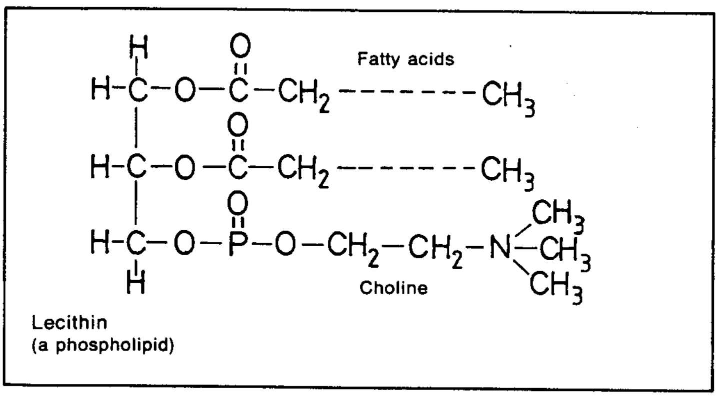



*Figure 17.4 The chemical structure of the phospholipid lecithin*

usually saturated. Thus, lecithin derived from soya is polyunsaturated, and lecithin derived from egg is saturated. Lecithins from different sources can have quite different nutritional implications. Lysolecithin has one less fatty acid than lecithin.

## Fats in food

Energy content of fat is 37 kJ/g
Energy content of carbohydrate is 16 kJ/g
Energy content of protein is 17 kJ/g
Energy content of alcohol is 29 kJ/g

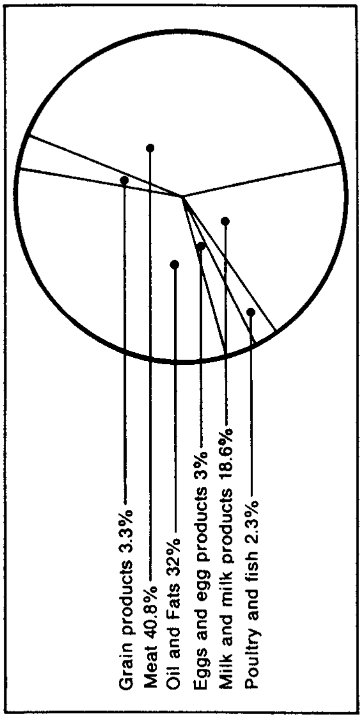



*Figure 17.5 Relative contribution of different foods to fat in the Australian diet*

Fat is the most energy-dense nutrient. Australians obtain most of their dietary fat from meat, 'oil and fats' and milk and milk products (figure 17.5). Of the meat sources of fat, beef and lamb contribute relatively the most.

It is important to be aware of the kind of fat that is found in food. In general, this means a knowledge of which foods are high in saturated fats, which are high in polyunsaturated fats, and which are high in cholesterol. Tables 17.2 to 17.4 show foods categorized according to whether they have high, moderate or low proportions of fats.

Table 17.2 Amounts of saturated fat in selected foods

| *Food* | *Saturated fat (g/100 g edible portion)* |
|---|---|
| HIGH CONTENT | |
| *Coconut: freshmeat* | 30 |
| *desiccated* | 54 |
| *Butter* | 45 |
| *Potato chips, cooked in* | |
| *palm oil* | 47 |
| *cottonseed oil* | 10 |
| *Red meat: lean* | 3 |
| *other* | 8–20 |
| *Cheese* | 16–23 |
| *Polyunsaturated margarine* | 11 |
| *Pastry* | 7–12 |
| *Ice cream* | 6 |
| MODERATE CONTENT | |
| *Eggs* | 4 |
| *Avocado* | 3 |
| *Chicken* | 3 |
| LOW CONTENT | |
| *Skimmed milk* | Trace |
| *Fruits* | Trace |
| *Vegetables* | 0–1 |
| *Cottage cheese* | Trace |
| *Cereals* | 0–2 |
| *Bread* | 1 |
| *Fish* | 0–2 |

Table 17.3 Polyunsaturated fat in foods

| *Food* | *Polyunsaturated fat (g/100 g edible portion)* |
|---|---|
| HIGH CONTENT | |
| *Certain vegetable oils (corn, cottonseed, maize, safflower, sunflower)* | *50–72* |
| *Soya oil* | *52* |
| *Walnuts* | *39* |
| *Polyunsaturated margarine* | *38* |
| MODERATE CONTENT | |
| *Pork from pigs (monogastric) raised on vegetable oil feeds* | *5* |
| *Beef products from animals (ruminants) fed vegetable oil feeds protected with casein* | *3* |
| LOW CONTENT | |
| *Most items high in saturated fat* | |
| *Foods with little or no fat* | |

Table 17.4 Cholesterol in foods

| *Food* | *Cholesterol (mg/100 g edible portion)* |
|---|---|
| *Brains* | *2000* |
| *Egg yolk* | *1500* |
| *Liver* | *300* |
| *Caviar* | *300* |
| *Butter* | *250* |
| *Shell-fish* | *100–200* |
| *Prawns* | *200* |
| *Cheese* | *100* |
| MODERATE CONTENT | |
| *Red meat (fatty)* | *70 (cooked 100)* |
| *Full cream milk* | *11* |
| LOW CONTENT | |
| *Fruits* | *0* |
| *Vegetables* | *0* |
| *Cereals* | *0* |
| *Bread* | *0* |
| *Polyunsaturated margarine* | *0* |

People with heart disease, or the risk of heart disease, are often asked to increase the ratio of polyunsaturated to saturated fat (p/s ratio) in their diet. The p/s ratio can be increased by decreasing the saturated fat or by increasing the polyunsaturated fat.

$$\text{p/s ratio} = \frac{\text{sum of all polyunsaturated fatty acids}}{\text{sum of all saturated fatty acids}}$$

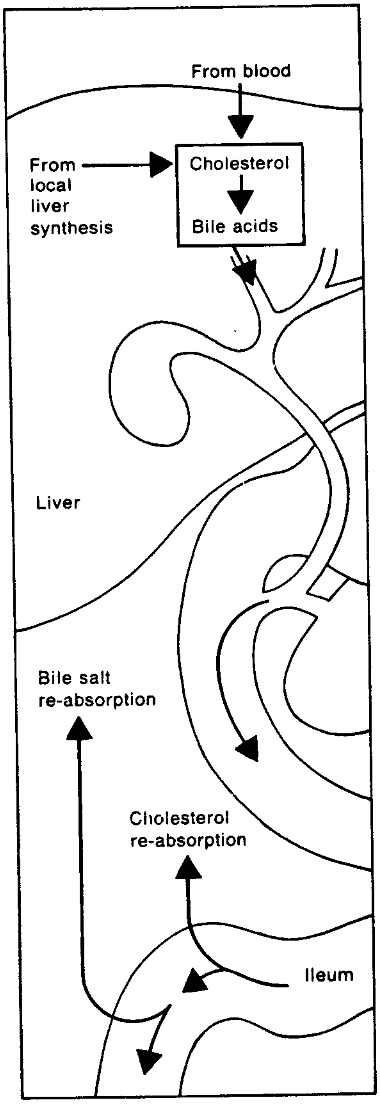



*Figure 17.6 In addition to cholesterol obtained from the diet, body cholesterol can be formed in the liver and other body cells, cholesterol can also be broken down to bile acids in the liver and excreted with them in the bile: it can then be re-absorbed from the small bowel*

Catabolized: broken down.

Lipoprotein: the combination of lipid with protein as in the plasma and in all membranes. Since lipid is itself water-insoluble, it can only be transported in blood in association with protein, which is water soluble.

## Fats made by body processes

Fats in the body can be obtained from dietary sources or can be made by the body's metabolic machinery.

Fatty acids are built up from the two carbon unit acetyl coenzyme A (CoA) (see chapter 13, Metabolism). Acetyl CoA can be derived from glucose (i.e. carbohydrate), amino acids (i.e. protein) and, indeed, fat itself. Cholesterol, too, is formed from the two carbon unit acetate. Imperfect control of cholesterol synthesis could lead to excessive cholesterol formation, and diet can influence cholesterol synthesis.

Cholesterol can be catabolized* in the liver to bile acids. Both cholesterol itself and bile acids are excreted in the bile. Thus the cholesterol in the small bowel could have been derived from the diet or from the bile. Cholesterol is re-absorbed, in part, further down the small bowel in the ileum. Bile salts are re-absorbed, in part, towards the terminal ileum. Bile acids can circulate from liver to small bowel back into the blood and to the liver six to ten times daily. With the diet that is commonly found in affluent societies, the synthesis of bile acids each day amounts to about 600 mg. This is a little less than the amount of cholesterol ingested in the diet each day. Bile acids and lecithin are the bile components that determine the solubility of cholesterol in the bile. If there is relatively too much cholesterol in the bile, then cholesterol gall stones develop.

## Blood fats

Lipids are only minimally soluble in water, so for them to be transported in blood they must be bound to protein. This transport lipid molecule is referred to as lipoprotein.*

The largest lipoproteins are the chylomicrons, which have the least protein and the most fat. Most of the fat is triglyceride, but phospholipid, cholesterol, and cholesterol-ester are also present. Chylomicrons are the form in which fat is transported from the gut through the lymph to the great veins of the neck and so into the blood circulation. They can serve as a source of fatty acids directly for energy utilization or for storage of energy. The classification of lipoproteins is shown in table 17.5.

The density of the lipoproteins can decrease in the blood (VLDL→IDL→LDL). This process delivers fatty acids to the tissues for use as a fuel or for storage in fatty tissue or deposition in arteries. In the same way chylomicrons can deliver fatty acids to the tissues. Cholesterol can also be delivered to the tissues, including the arteries, where it may be deposited. High density lipoprotein (HDL) seems to be a way in which cholesterol can be picked up from peripheral tissues and returned to the liver.

Table 17.5 Classification of lipoproteins

| *Lipoprotein* | *Dominant lipid contained* |
|---|---|
| *Chylomicrons (extremely low density lipoprotein)* | *Triglyceride* |
| *Very low density lipoprotein (VLDL)* | *Triglyceride* |
| *Intermediate density lipoprotein (IDL)* | *Triglyceride and cholesterol* |
| *Low density lipoprotein (LDL)* | *Cholesterol* |
| *High density lipoprotein (HDL)* | *Phospholipid* |
| *Albumin-free fatty acid (extremely high density lipoprotein)* | *Free fatty acid (FFA)* |

## Function of lipids

The nutritionally related functions of fats in food and in the body are:
1. to increase palatability of food;
2. to act as a vehicle for fat-soluble vitamins in food;
3. to contribute to membrane structure: both phospholipid† and cholesterol are an integral part of the cell membrane;
4. to transport energy
(a) as VLDL triglyceride fatty acid,
(b) as free fatty acid bound to albumin;
5. to store energy: as triglyceride;
6. to control metabolism
(a) by free fatty acids (modulate glucose uptake by tissues)
(b) by prostaglandins†
(c) by steroid hormones (e.g. cortisol and sex hormones);
7. to transport cholesterol
(a) by LDL (arises from VLDL from liver and carries cholesterol to peripheral tissues)
(b) by HDL* (from tissues to liver).
†Normality dependent on availability of essential fatty acids.

Even if certain fats were not essential for man's metabolism, they add to the quality of food by making it more attractive to eat and by serving as a vehicle for fat-soluble vitamins. They are also part of the structure of every cell, and they serve as fuels for the body, and they assist in regulation of the body's metabolism.

## Essential fatty acids

For each of the functions of lipids, fats can be synthesized by the body, with the exception of those situations where essential fatty acids are required. Essential fatty acids are fatty acids that the human body cannot manufacture. Essential fatty acids are polyunsaturated, but not all polyunsaturated fatty acids are essential fatty acids.

Functions of essential fatty acids are listed below.

1. One of the best documented roles for essential fatty acids is in the formation of prostaglandins.* Prostaglandins appear to be principally intracellular regulators* of metabolism. They influence a number of physiological events including temperature regulation, blood vessel control, platelet aggregation, contraction of the uterus, and the flux of free fatty acids from fatty tissue. Many drugs act by modifying prostaglandin formation; for example, aspirin reduces fever through inhibition of prostaglandin formation.
2. Essential fatty acids modify the removal of VLDL triglyceride fatty acid from the circulation.
3. The extent to which cholesterol can be removed from the body is influenced by the availability of essential fatty acids.
4. The structure of cell membranes is influenced by the availability of essential fatty acids for phospholipids, as they form an integral part of cell membranes.

Prostaglandins: intracellular regulators of metabolism formed from essential fatty acids.

Intracellular regulators: for the cell's activities to be appropriate to needs, regulation is required within the cell (intracellular) as well as between cells (achieved by chemical changes in the cell's environment, hormones, and nervous impulses).

## Diet and blood fats

Many people seem confused about whether it is possible to modify blood cholesterol and triglyceride concentrations by diet. It is quite certain that dietary change can alter blood fats, and can be done in a variety of ways.

Three dietary fats affect blood cholesterol:
1. cholesterol
2. saturated fat
3. polyunsaturated fat

The confusion probably arises firstly because the effect of a given dietary change varies from individual to individual. To change from a typical Australian diet (chapter 4, Australian eating patterns) towards one that is most likely to lower the blood cholesterol level, the average reduction in plasma cholesterol concentration would be between 10 and 15 per cent, but the range of change might be from 3 or 4 per cent to 30 per cent. This is about all that can be achieved by change in dietary fat alone. Dietary changes that lead to a decrease in body weight have a favourable effect in lowering blood levels. So weight reduction is often the first advice given to people with high blood fat.

Dietary modification of blood lipids aims to decrease the tissue levels of cholesterol, especially the levels in arterial walls. Tissue levels might fall without a fall in the blood level. In some individuals it may be months before the blood cholesterol itself starts to fall. Usually, with dietary change, the blood fats start to change within days or weeks.

The dietary lipid change that is most beneficial in reducing cholesterol concentration in the blood is a reduction in saturated fat intake. But a reduction in cholesterol intake and an increase in polyunsaturated fat intake also help. A change in the p/s ratio not only lowers cholesterol but also lowers triglyceride. A reduction in cholesterol intake reduces only blood cholesterol levels.

It is often said that a high carbohydrate diet induces high blood triglycerides (hypertriglyceridaemia).* Although it may do so

Hypertriglyceridaemia: an increase in the concentration of triglyceride in blood plasma above the normal level, which is associated with health problems. Triglyceride is found as a lipoprotein, mainly chylomicrons, for 3 or 4 hours after a meal.

briefly, the effect does not last, so there is no good reason to recommend low carbohydrate diets for the management of high blood fats. In fact, a high carbohydrate diet enables a reduction in the relative contribution of fat to energy intake, and, therefore, to the control of blood fats.

Non-absorbable carbohydrate (dietary fibre) also contributes to the blood-lipid lowering potential of a dietary regime. It is possible that different dietary fibres act in different ways to achieve this.

It is possible also that factors accompanying dietary fibre are contributory, such as saponins.* The preferred diet for lipid lowering is now believed to be one that is high in unrefined carbohydrate and low in saturated fat and cholesterol.

Saponins: compounds known as triterpenes and found in high concentrations in vegetables such as chick peas.

The sterols in plants also appear to have properties that allow them to add to the cholesterol-lowering effect of a more vegetarian-type diet. This may in part be because the sterols compete with cholesterol for absorption.

In Australia, one of the more important dietary factors leading to high blood fats is alcohol abuse. This primarily increases the blood triglyceride level and with it, to some extent, the blood cholesterol level. Some people are particularly sensitive to alcohol, and blood fats rise markedly with even small amounts. However, alcohol may increase the high density lipoprotein (HDL) concentrations in blood; the higher these are the greater the protection against ischaemic heart disease.*

Ischaemic heart disease: ischaemia refers to the shortage of blood supply to an organ or part of it, in this case the heart.

Exercise also seems to be associated with higher concentrations of HDL in blood. This may be one of the means through which regular exercise protects against coronary disease. It also appears that during prolonged exercise triglyceride levels fall. The effects of exercise on total cholesterol levels are less consistent, with either no change or a small fall in concentrations.

The vitamin, nicotinic acid (niacin), when administered in high dosage (about 3 g per day instead of the recommended daily allowance of 15 mg per day) has the effect of lowering blood cholesterol and triglyceride concentrations. This can be called a megavitamin effect. It is a pharmacological effect (drug-like effect) of nicotinic acid rather than a physiological effect (normal function). One cannot extrapolate from this difference in vitamin action at low intakes as opposed to high intakes to what might happen with other vitamins. Niacin is used to manage blood-fat concentration that cannot be adequately controlled by diet alone.

## Blood fats and atherosclerosis*

Atherosclerosis: derived from Greek words 'atheros' meaning gruel or porridge and 'scleros' referring to hardening; hardening of the arteries due to the deposition of gruel-like lipid.

The various lipoprotein sources of cholesterol, then, are regarded as atherogenic* particles. Most of the cholesterol that deposits in the artery is derived from the blood rather than from local arterial synthesis.*

Atherogenic: giving rise to atherosclerosis.

Arterial synthesis: formation of substances within the arterial wall.

The form in which cholesterol is deposited is principally cholesterol ester, although there is some free cholesterol* (unesterified cholesterol). The fatty acid with which the cholesterol is esterified may come from the blood (from free fatty acids in the

Free cholesterol: the cholesterol molecule without an attached fatty acid molecule.

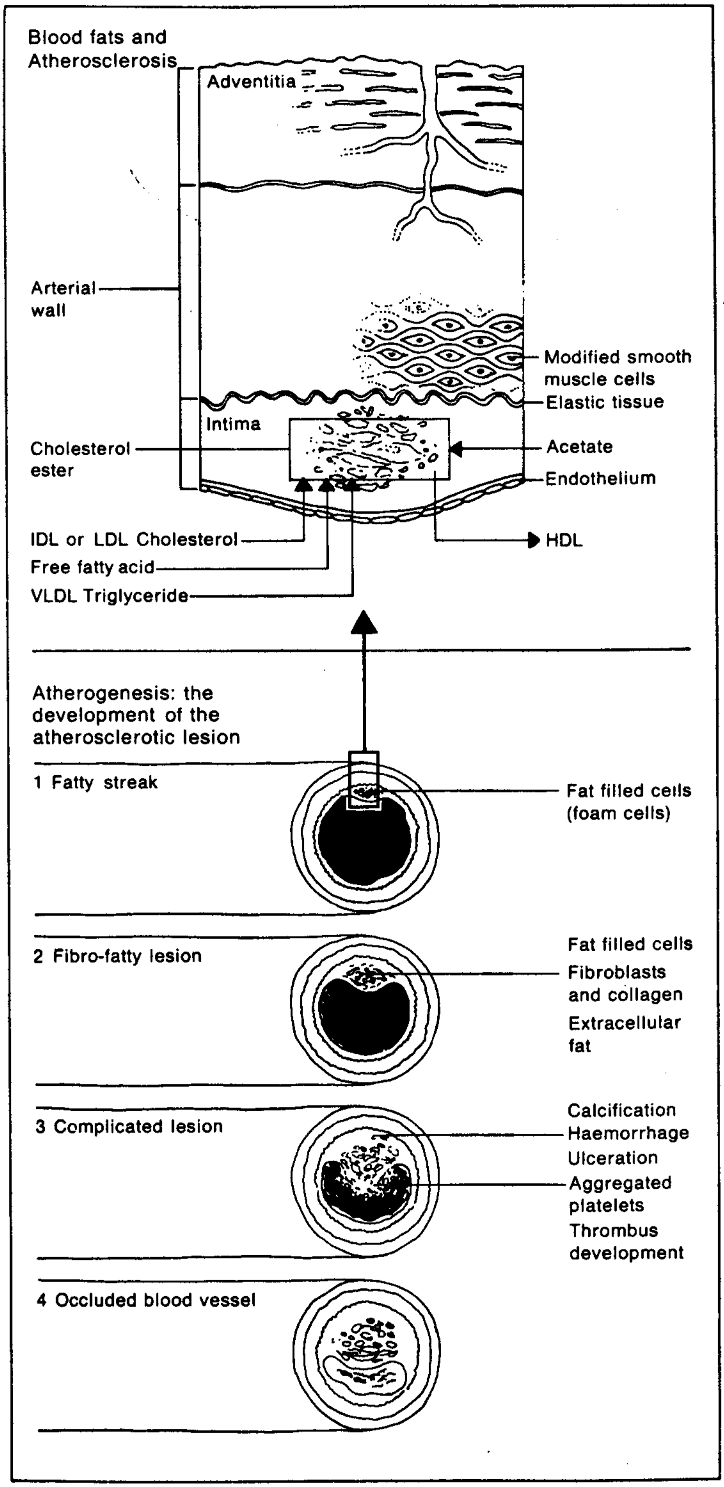



*Figure 17.7 Atherosclerosis is the disease commonly known as 'hardening of the arteries'. It affects the inner aspect of the artery, the intima and media. Cholesterol accumulates at first in cells which later break down. The lumen of the artery becomes progressively narrowed by the developing lesion or by thrombus, which is like a clot.*

blood or from triglyceride fatty acids in the blood) or it may be synthesized in the arterial wall from acetate. The other lipid contributing to atherosclerosis is phospholipid.

Atherosclerosis is the process that is generally referred to when 'hardening of the arteries' is discussed. It occurs at particular places, such as where arteries branch or at points of mechanical stress, rather than uniformly throughout the body. It affects large and medium-sized arteries. It affects, for example, the aorta (the main artery leading away from the heart), all of the first branches of the aorta, and some of the further branches. It affects arteries supplying the heart itself, the head and neck, the gut, the kidneys and the lower limbs.

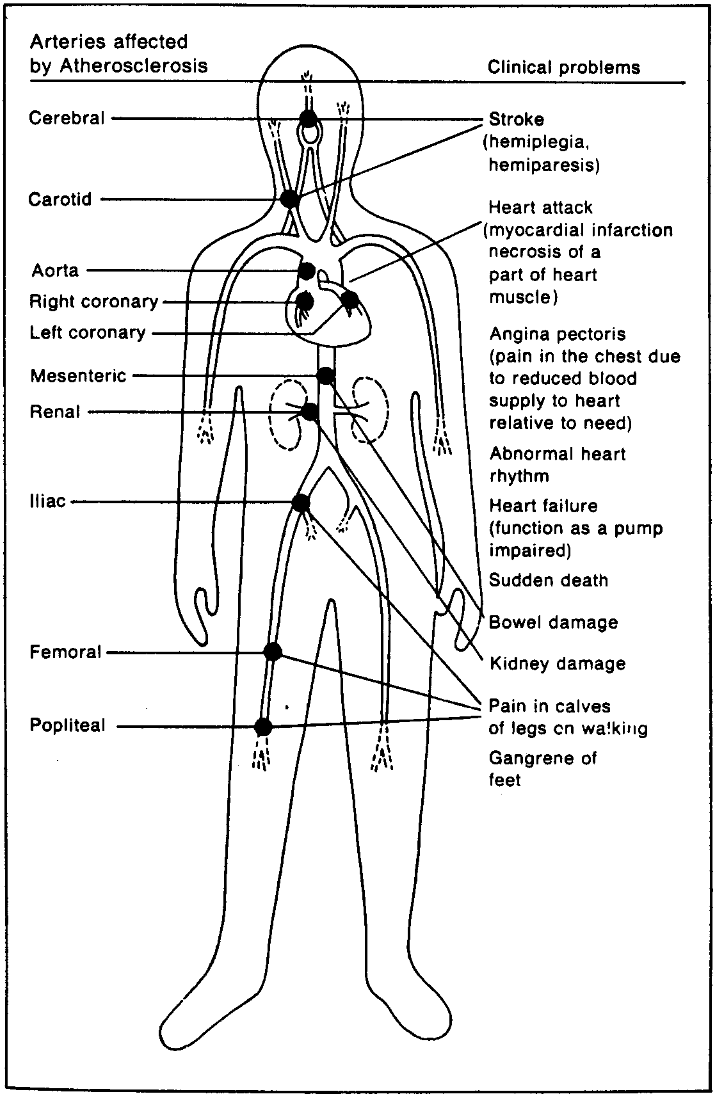



*Figure 17.8 Any of the large or medium sized arteries of the body can be affected by atherosclerosis and so the tissues they supply can be deprived of blood. The most important tissues at risk are the brain, the heart, the kidneys, the gut and the legs.*

It is a disorder that principally affects the inner arterial wall. Initially most of the lipid that is deposited is found within cells, principally modified smooth-muscle cells. The cells that accumulate a lot of lipid take on a foamy appearance and are called 'foam cells'. At this stage of development of the atherosclerotic process, yellow raised areas can be seen on the inner surface of the artery and these are called 'fatty streaks'. As lipid is released from these cells as they undergo breakdown or necrosis*, and as fibrosis* (the laying down of connective tissue as in repair) takes place, the lesion becomes tougher and is described as 'fibro-fatty'. With time, this lesion can ulcerate, calcify, or haemorrhage may occur into it. In addition, a thrombus (a kind of clot or the process of thrombosis) may be superimposed and the vessel may rapidly occlude.* The principal component of a thrombus is aggregated platelets.* The atherosclerotic lesion is metabolically active, so perhaps lipid removal as well as deposition occurs. In the lesion itself the kind of fatty acid available, polyunsaturated or saturated, can affect the extent to which cholesterol is deposited.

Atherosclerotic lesion: the area of the artery affected by atherosclerosis.

Necrosis: the death of tissue.

Fibrosis: replacement of body tissues with fibrous or scar tissue.

Occlude: to block off.

Platelets: those fragments of cells without nuclei in blood which are responsible for repairing damaged endothelium and helping to form blood clots.

## Risk factors for atherosclerosis

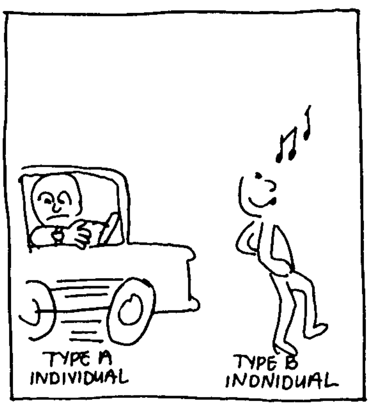
Nutritionists are interested in the ways in which diet adds to the risk of atherosclerotic vascular disease. The most well defined nutritionally related risks are those of elevated fats, cholesterol and triglycerides. Elevated cholesterol concentration seems to be a major risk. The risk constituted by triglycerides is uncertain. Decreased high-density lipoproteins also increase risk for atherosclerotic vascular disease.

Diet also seems to influence platelet aggregation. The more platelets aggregate the more likely are atherogenesis and thrombosis to occur. It appears that the higher the intake of alphalinolenic acid* and of eicosapentaenoic acid* the less the platelets are likely to aggregate. Eicosapentaenoic acid is found in marine fats and may be important in the protection of Eskimos from atherosclerotic heart disease.

Alphalinolenic and eicosapentaenoic acid belong to the $\omega$ 3 series of essential fatty acid which serve as precursors for those prostaglandins that reduce platelet aggregation.

Another major risk factor is high blood pressure (hypertension). There is probably no one factor that accounts for the development of hypertension in most individuals. However, dietary sodium intake seems to be important. Essential fatty acid intake and the formation of prostaglandins from these fatty acids contribute to the development of hypertension.

Smoking is the other avoidable principal risk factor in the development of heart disease. It seems likely that certain components of cigarette smoke damage the arterial wall. The other principal risk factors, age and sex, are unavoidable! Women do not develop heart disease as early in life as men.




If we were able to define more clearly *personality*, *significant life events*, and *social mobility* there could be another useful addition to the risk profile. The studies of Drs Rosenman and Friedman in the United States suggest that a personality type A may be particularly prone to coronary heart disease. This kind of personality is one in which there is a sense of the pressure of time, even though the actual work load might not differ from that of another person. At the other end of the personality spectrum is the type B individual, largely relaxed and easy-going, even though the person's work load could be considerable.

Studies generally have not indicated *obesity* to be a major risk factor. This at first seems curious, since we know obesity reduces life expectancy and that much of this excess risk is by way of cardiovascular (heart and blood vessel) disease. This paradox might be explained because studies have not considered that obesity might increase the risk by way of high blood fats and hypertension. Reduction in body weight does reduce these two risks.

The level of physical activity and its relationship to heart disease has also been difficult to define. There might be differences in leisure time as opposed to on-the-job activity. A recent study reported by Dr Morris from London, in which he studied a group of men over a period of sixteen years, indicated that those who have the highest energy intake and who maintained body weight (which is to say they had the highest energy output and therefore level of physical activity) had the least heart disease. In the same study, those who had the highest intake of dietary fibre from bread also had the least heart disease.

## Consequences of atherosclerosis

One ordinarily thinks of atherosclerosis principally in terms of heart disease or coronary disease. But any artery affected by atherosclerosis is liable to blockage, which can lead to death of the tissue or organ that it supplies. Narrowing of the vessel by even 70 per cent leads to a substantial reduction in blood flow to the tissue beyond the point of narrowing; this is termed 'ischaemia'.

Blockage of the coronary arteries can lead to sudden death or myocardial infarction ('a heart attack'). A reduction in blood flow can lead to chest pain or 'angina pectoris'. Abnormal heart rhythms or impairment of the heart's function as a pump can also occur. When the arteries supplying the brain are blocked a stroke occurs. The vessels to the legs can be blocked resulting in pain in the calves of the legs on walking, or gangrene (death of tissue).

## Can atherosclerosis regress?

Ideally, we would like to prevent the development of atherosclerosis. However, almost everybody in an affluent society has evidence of atherosclerosis to some extent, even in their teens. To reduce atherosclerosis, we need to know if regression of atherosclerosis can occur. There is now encouraging evidence that it can.

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# FOOD & NUTRITION IN AUSTRALIA

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