

# 18 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, mono-unsaturated 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 or omega-6 series, from principally vegetable sources, and the linolenic or omega-3 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 and mono-unsaturated fats lower, the blood cholesterol concentration, which is an important risk factor for atherosclerotic vascular disease. 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

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 resynthesised (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

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.

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 summarised in table 18.1. There are properties of fat that may increase appetite and others that may decrease it.

*Table 18.1* Characteristics of lipids that affect appetite

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

<i>Appetite increase</i>	<i>Appetite decrease</i>
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

## Chemistry of fats

Fats, or lipids, are characterised 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.

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

### Free fatty acids

Free fatty acids (FFA) are so named because they are not associated or esterified\* with other molecules, such as



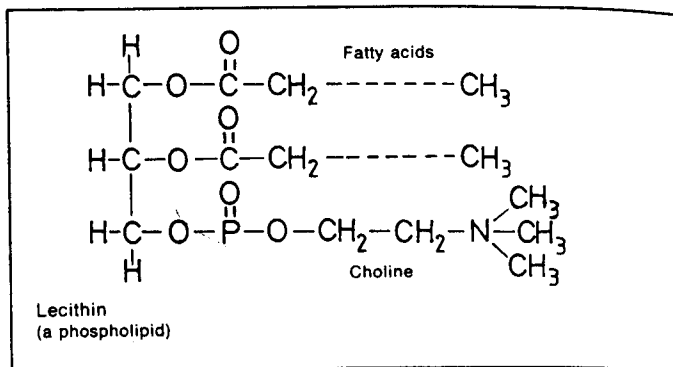


Figure 18.2 The chemical structure of triglyceride.

Figure 18.3 The chemical structure of cholesterol.

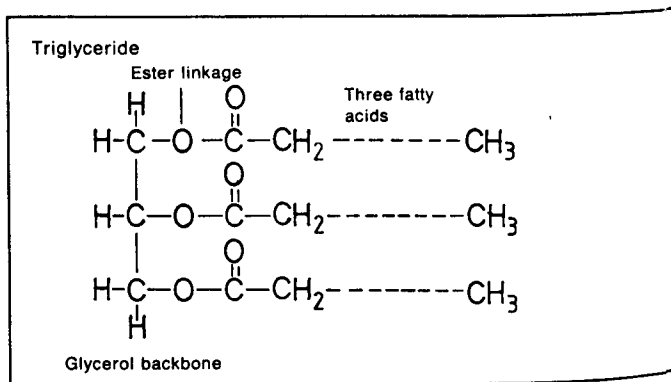
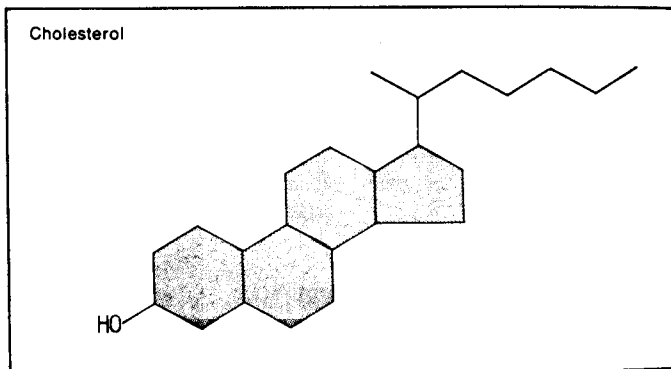


Figure 18.4 The chemical structure of the phospholipid lecithin.

which cholesterol may be complexed. When cholesterol is not esterified it is spoken of as 'free' or 'unesterified' 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 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

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 18.5). 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 18.2 to 18.4 show foods categorised according to whether they have high, moderate or low proportions of fats.

Table 18.2 Amounts of saturated fat in selected foods

Food	Saturated fat (g/100 g edible portion)
<i>HIGH CONTENT</i>	
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

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.

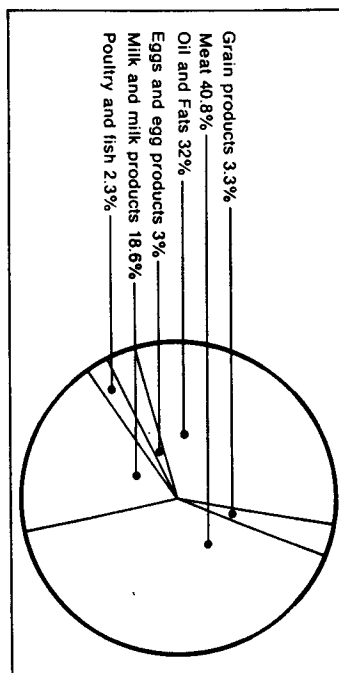


Figure 18.5 Relative contribution of different foods to fat in the Australian diet.

Table 18.2 cont'd

*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 18.3 Polyunsaturated fat in foods

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

Table 18.4 Cholesterol in foods

<i>Food</i>	<i>Cholesterol (mg/100 g edible portion)</i>
Brains	2000
Egg yolk	1500
Liver	300
Caviar	300
Butter	250
Shell-fish	100-200
Prawns	200
Cheese	100

Table 18.4 cont'd

## 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.

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

## 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 (figure 18.6).

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 catabolised\* 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.

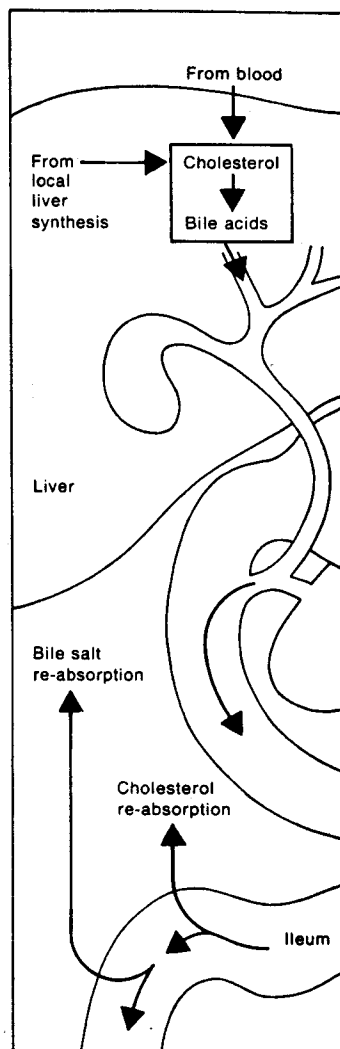


Figure 18.6 In addition to cholesterol obtained from the diet, body cholesterol can be formed in the liver and other body cells, cholesterol can also be broken down to bile acids in the liver and excreted with them in the bile: it can then be re-absorbed from the small bowel.

Catabolised: 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.

HDL: High-density lipoprotein.

## 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 utilisation or for storage of energy. The classification of lipoproteins is shown in table 18.5.

The density of the lipoproteins can increase 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 18.5 Classification of lipoproteins

<i>Lipoprotein</i>	<i>Dominant lipid contained</i>
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 synthesised 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.

## Diet and blood fats

Many people seem confused about whether it is possible to modify blood cholesterol and triglyceride concentrations by

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).

Three dietary fats affect blood cholesterol:

1. cholesterol;
2. saturated fat;
3. polyunsaturated fat.

diet. It is quite certain that dietary change can alter blood fats, and can be done in a variety of ways.

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 briefly, the effect does not usually 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. It may be that with very low fat intake (less than 20 per cent of energy), blood triglycerides rise.

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.

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

**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.

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

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\*.

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\*

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.\*

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 blood or from triglyceride fatty acids in the blood) or it may be synthesised 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.

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

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

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.

Atherogenic: giving rise to atherosclerosis.

Arterial synthesis: formation of substances within the arterial wall.

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

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

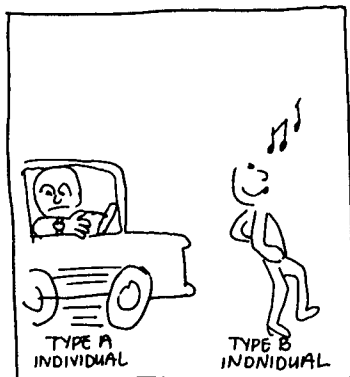
**Necrosis:** the death of tissue.

**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.

Alpha-linolenic and eicosapentaenoic acid belong to the 3 series of essential fatty acids which serve as precursors for those prostaglandins that reduce platelet aggregation.



and are called 'foam cells'. At this stage of development of the atherosclerotic lesion\*, 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 (figure 18.7).

## Risk factors for atherosclerosis

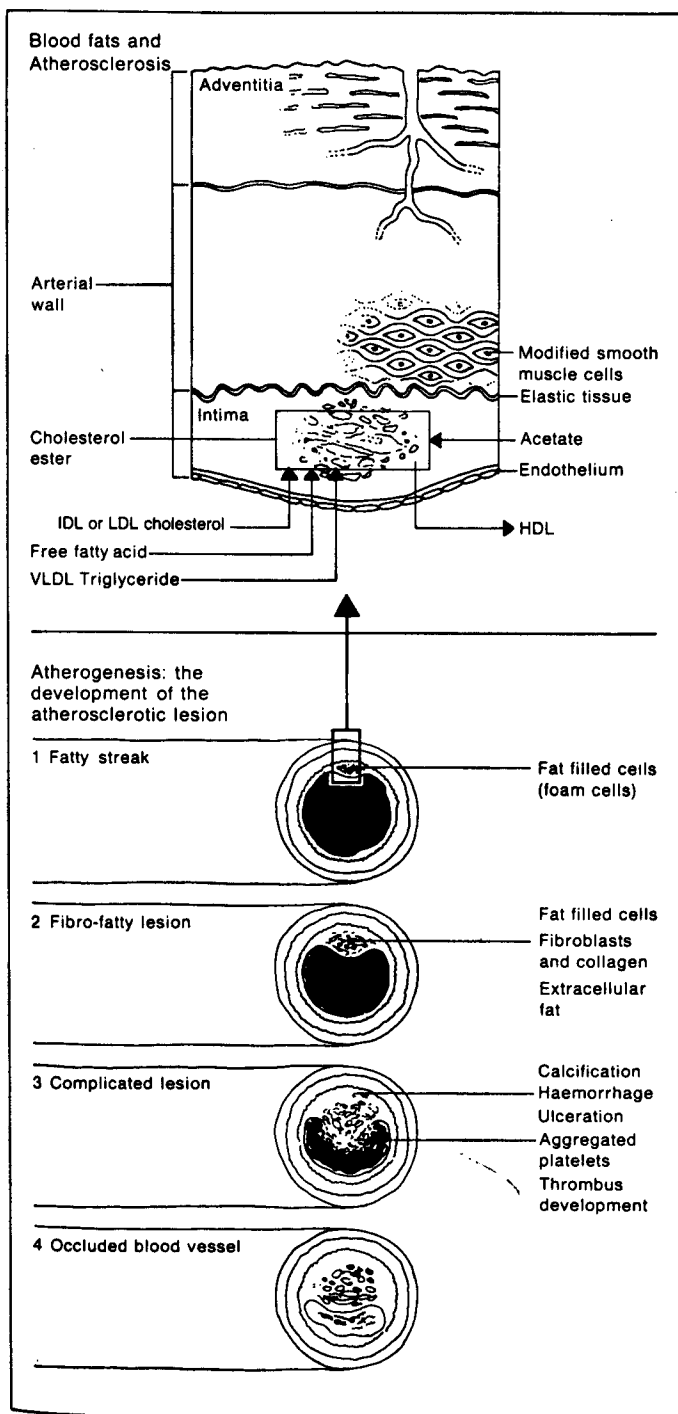
Nutritionists are interested in the ways in which diet adds to the risk of atherosclerotic vascular disease. The most well defined nutritionally related risks are those of elevated fats, cholesterol and triglycerides. Elevated cholesterol concentration seems to be a major risk. The risk constituted by 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 alpha-linolenic 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 (figures 18.8, 18.9).

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. Alcohol also raises blood pressure.

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 gender, 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 suggested that a personality type A may be particularly prone to coronary



*Figure 18.7* 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.

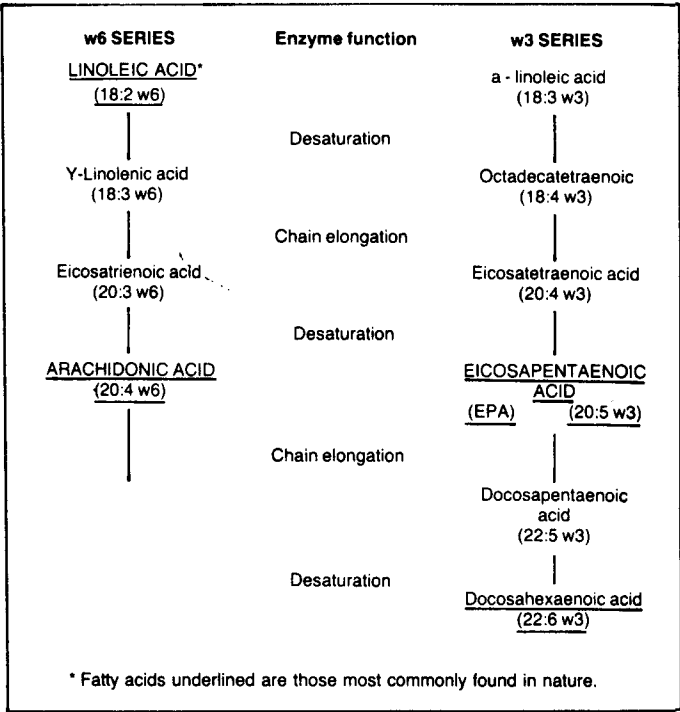
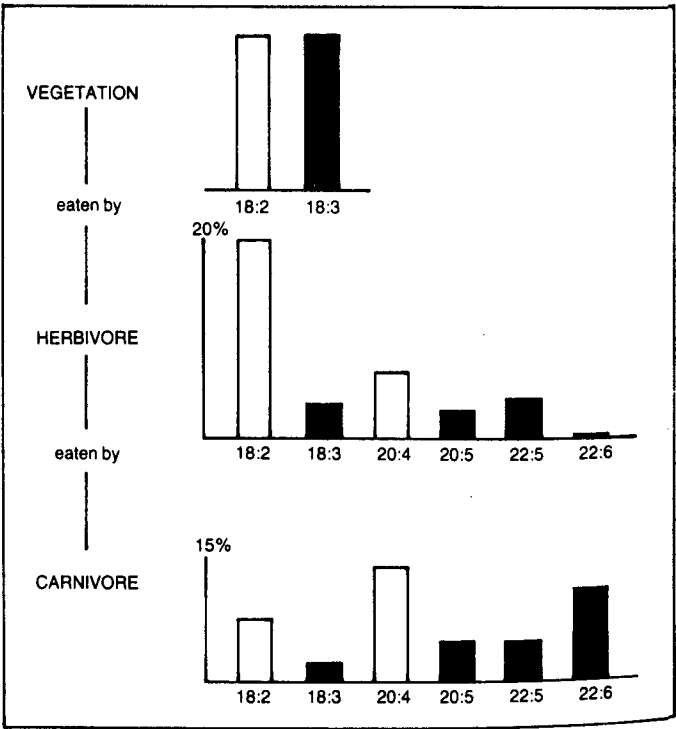


Figure 18.8 Summary of fatty acid metabolism in man. After O'Dea and Sinclair, 1983.

Figure 18.9 Fatty acids in the food chain. This illustrates the progression of fatty acids from vegetation through the herbivore to the carnivore. The height of the bars represents relative amounts of the different fatty acids at each point in the chain.



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. Controversy remains about these findings.

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, hypertension and diabetes. Reduction in body weight does reduce these risks.

Some years ago, it was reported by Dr Morris that, in a group of men studied over a period of sixteen years, those who had 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. Similar findings, indicating a beneficial effect of higher energy intake in decreasing both coronary and total mortality, have now also been reported from the Netherlands and Sweden. Apart from indicating that in the long term it is better to be physically active, these studies also suggest that the extra intake of energy should come from foods of vegetable and marine origin.

## 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.

### Further reading

CRAWFORD, M. A. SINCLAIR, A. J. 'Nutritional influences in the evolution of mammalian brain'. In *Lipids, malnutrition and the developing brain*. Ciba Foundation Symposium, 1971, pp. 267-292.

LEWIS, E. and LEWIS, B. *The Heart Book*. Barrie and Jenkins, London, 1980.

NATIONAL HEART FOUNDATION OF AUSTRALIA. *Planning fat controlled meals*. N.H.F. of Aust., P.O. Box 2, Woden, ACT 2606.

O'DEA, K. SINCLAIR, A. J. 'The Modern Western Diet — the exception in man's evolution'. In *Agriculture and Human Nutrition: how close are the links?*. Edited by K. A. Boundy and G. H. Smith, Australian Institute of Agricultural Science, Department of Agriculture, Victoria 1983, pp. 56-61.

SEBRELL, W. H. JR and HAGGERTY, J. J. *Food and Nutrition*. Life Science Library. Time-Life International, Nederland, 1973.

SPAIN, D. M. 'Atherosclerosis' in *Human Nutrition. Readings from Scientific American*. Edited by N. Kretchmer and W. van B. Robertson. W. H. Freeman & Co., San Francisco, 1978.



# FOOD & NUTRITION IN AUSTRALIA

Edited by Mark L. Wahlqvist

in conjunction with the Department of Human Nutrition, Deakin University

Contributors: David R. Briggs, Jill B. Carey,  
Patricia A. Crotty, Delia M. Flint, Gwyn P. Jones,  
Richard S. D. Read, Ingrid H. E. Rutishauser,  
Boyd J. G. Strauss

Illustrations by Neville Todd



Nelson

First published 1981  
Second edition 1982  
Reprinted 1983  
Reprinted 1984 twice  
Reprinted 1985  
Reprinted 1986  
Third edition 1988  
Reprinted 1989  
Reprinted 1992

Thomas Nelson Australia  
102 Dodds Street South Melbourne 3205

© Cassell Australia Ltd 1981  
© Methuen Australia Ltd 1982  
© Thomas Nelson Australia 1988

Cover design by Green Poles Design  
Illustrated by Neville Todd  
Photographs on pages 20, 27, 28, 37, 46 and 66 by Peter Wilson  
Set in 10/11 Garamond by SRM Productions, Malaysia  
Printed in Singapore by  
Kyodo Printing Co. Pte Ltd

All rights reserved. No part of this publication may be reproduced or transmitted in any form or by any means, electronic or mechanical, including photocopying, recording or by any information storage and retrieval system, without permission in writing from Thomas Nelson Australia.

National Library of Australia  
Cataloguing-in-Publication Data

Food and nutrition in Australia.

3rd rev. ed.  
ISBN 0 17 007343 2.

1. Food. 2. Nutrition. 3. Food — Social aspects.  
— Australia. I. Wahlqvist, Mark L. II. Briggs,  
David R., 1944 — . III. Todd, Neville.

641'.0994

'I'm an Aussie' reproduced by permission of P. Best and  
Monahan Dayman and Adams  
'The Schoolboy's Lament' reproduced by permission of Brenda Ryan  
'A Dip into the Past' reproduced by permission of Phillip Adams

# Contents

## Section One The sociology of food 1

- 1 Nutrition: does it matter? Mark L. Wahlqvist 2
- 2 History of nutrition in Australia Mark L. Wahlqvist 12
- 3 Culture and food choice Patricia A. Crotty 22
- 4 Australian eating patterns Ingrid H. E. Rutishauser and Delia M. Flint 30
- 5 Food and the law David R. Briggs 49
- 6 Food Faddism Delia M. Flint and Mark L. Wahlqvist 64

## Section Two The science of food 69

- 7 Food production Richard S. D. Read 70
- 8 Food processing Gwyn P. Jones 88
- 9 Food microbiology David R. Briggs and Gwyn P. Jones 99
- 10 Food preparation Jill B. Carey and Richard S. D. Read 115
- 11 Food additives David R. Briggs 138

## Section Three Physiology and metabolism 152

- 12 Digestion and absorption Boyd J. G. Strauss 153
- 13 Metabolism Mark L. Wahlqvist 167
- 14 Exercise and nutrition Richard S. D. Read 176

## Section Four Nutrients and their significance 189

- 15 Energy Jill B. Carey and Richard S. D. Read 190
- 16 Carbohydrates Mark L. Wahlqvist 215
- 17 Dietary fibre Gwyn P. Jones 228
- 18 Lipids Mark L. Wahlqvist 243
- 19 Protein Richard S. D. Read 259
- 20 Water Boyd J. G. Strauss and Mark L. Wahlqvist 273
- 21 Vitamins Mark L. Wahlqvist 281
- 22 Major elements Boyd J. G. Strauss 309
- 23 Minor elements Boyd J. G. Strauss 322
- 24 Alcohol Boyd J. G. Strauss 329
- 25 Natural toxicants in food David R. Briggs 340
- 26 Food composition tables and dietary allowances  
Ingrid H. E. Rutishauser and Delia M. Flint 352

## Section Five Nutritional status 365

- 27 The individual Boyd J. G. Strauss and Delia M. Flint 366

28 The community Ingrid H. E. Rutishauser 373

## Section Six Nutrition and the ages of man 383

29 Pregnancy and lactation Ingrid H. E. Rutishauser 384

30 Growing up: infant to adolescent Ingrid H. E. Rutishauser 401

31 The adult and the family unit Mark L. Wahlqvist 430

32 The elderly Delia M. Flint and Mark L. Wahlqvist 433

## Section Seven Some issues in nutrition 439

33 Survival nutrition Richard S. D. Read and Gwyn P. Jones 440

34 Nutrition and cancer Mark L. Wahlqvist 449

35 Nutrition and the brain Mark L. Wahlqvist 453

36 Food sensitivities David R. Briggs 457

37 Our neighbours Delia M. Flint and Mark L. Wahlqvist 466

38 Future food supply Richard S. D. Read 472

39 Nutrition education Patricia A. Crotty 485

40 National nutrition policy Mark L. Wahlqvist 498

## Section Eight Nutrition resources 507

Index 512