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ILSI EUROPE CONCISE MONOGRAPH SERIES

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**HEALTHY  
LIFESTYLES  
*NUTRITION  
AND PHYSICAL  
ACTIVITY***

ILSI



International  
Life Sciences  
INSTITUTE

**HEALTHY LIFESTYLES**  
*NUTRITION AND  
PHYSICAL ACTIVITY*



**ILSI Europe**

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Printed in Belgium

ISBN 1-57881-003-5

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## FOREWORD

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In response to the European Community Action on Health Promotion, Information, Education and Training of the European Commission Directorate-General V, ILSI Europe set up a Healthy Lifestyles Task Force. This task force decided to review the existing science that underlies the promotion of healthy lifestyles. This concise monograph provides an overview of the scientific underpinning of those aspects of healthy lifestyles in which ILSI has expertise. To ensure that this is an authoritative document, it has been deliberate policy to omit topics on which ILSI has not developed expertise. Whereas the topics included here are clearly of major importance to an overall healthy lifestyle, readers must be aware that aspects such as the role of smoking and dietary salt intake and the contribution of genetic background also need to be considered.

A number of European experts addressed issues related to the intake and health aspects of fat, antioxidants, dietary fibre, fluid and alcohol and also of physical activity and oral health. Suggestions for further reading are given in each chapter.

Public health authorities, regulators at national and European levels as well as representatives from academia, national nutrition foundations, consumer organisations and industry will find this informative publication a useful tool. With improved understanding of the scientific basis of the relationship between nutrition and health, people in all of these groups can apply the knowledge presented in these pages in ways that can contribute to current efforts to promote healthy lifestyles.

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### ***Note of the Scientific Editor***

The editor's main role in preparing this multiauthor report has been to ensure reasonable consistency of style, content and presentation across all chapters, while allowing authors a certain individuality of approach. In several areas of diet and health, there is an extensive scientific literature providing a vast amount of information. The information is there for all to read; however, the interpretation of that information is by no means always without contention. The scientific editor wishes to point out that whereas he has liaised closely with several authors to ensure a balanced assessment and presentation of topics that are open to different interpretations, the interpretation in each chapter of this booklet is the author's and not necessarily that of the scientific editor or indeed the referees.

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# 1. INTRODUCTION

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As long ago as 480 BC, Hippocrates recognized that several aspects of what we now call “lifestyle” must come together to produce a healthy body. He said: “Positive health requires a knowledge of man’s primary constitution and the powers of various foods, both those natural to them and those resulting from human skill. But eating alone is not enough for health. There must be exercise, of which the effects must likewise be known. The combination of these two things makes regimen, when proper attention is given to the season of the year, the changes of the winds, the age of the individual and the situation of his home. If there is any deficiency in food or exercise the body will fall sick”.

What Hippocrates called “man’s primary constitution” we today call “genetics”, and we can infer that “foods resulting from human skill” can be equated with today’s processed foods. In the generally affluent European continent, overall health has never been better and life expectancy at birth is at its highest ever. However, chronic diseases still exist and many have been associated with deficiencies in the lifestyle factors perceptively referred to by Hippocrates so long ago.

Given the importance of food in health, the European food industry has a major role to play in contributing to healthy lifestyles through the foods it produces and the scientific knowledge it helps disseminate.

A healthy lifestyle is defined here as one that gives an individual his or her optimal level of physical and mental health. People respond differently to environmental and lifestyle factors, depending on their individual genetic makeup. Every individual inherits a set of genes which contain information that determines certain characteristics. However, the effects of these genes are modified by

interaction with environmental factors, including diet and exercise. This monograph recognizes the importance of interactions between genetic and environmental factors in determining an individual’s health.

This monograph is for the benefit of personnel in the food, health and related industries, those in academia, members of consumer organizations and regulators at both the national and the European levels. Its aim is to provide a brief, accurate summary of the state of the science in regard to those components of a healthy lifestyle in which ILSI has scientific expertise. A very large number of factors contribute to health, not all of which are within ILSI’s expertise.

This concise monograph is therefore not intended to be exhaustive, but concentrates on, as indicated in the table of contents, a selected number of important aspects of health covered in existing ILSI publications and other consensus documents. For example, whereas it is clear that improvements in many aspects of health could be made by the single act of reducing smoking, a discussion of the health effects of smoking is not within the remit of this booklet. Furthermore, most readers will be aware that a discussion of the role of salt in health and disease is an outstanding omission. There is still considerable scientific debate about the role of sodium intake in the development and the management of hypertension in a normal population. Scientific evidence is still being collected and needs to be further evaluated.

It is not the purpose of this concise monograph to make recommendations about how to achieve optimal health. Its main objective is to provide readers with a summary of current knowledge of the underlying science. On the basis of this understanding, individuals can make their own decisions about how best to improve their health, and health professionals and educators can be guided in their task of formulating public health recommendations.

Chapter 2 summarizes current knowledge of general nutritional principles in relation to health, and Chapters 3-10 review current knowledge in specific aspects of nutrition and other lifestyle factors affecting health. Chapter 11 summarizes and draws together all these topics and discusses limitations of the scientific knowledge and ways in which it can be translated into practical application.

## ***2. THE ROLE OF NUTRITION IN A HEALTHY LIFESTYLE: CURRENT CONCEPTS OF NUTRITION AND HEALTH***

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### ***What constitutes a healthy lifestyle?***

Health is a condition of physical, mental and social well-being and implies the absence of disease. A major effect of disease in human society is a high rate of premature deaths, and therefore an important aim in the improvement of public health is to reduce the number of premature deaths and increase the population's total life expectancy. Unfortunately, increased life expectancy often brings with it poorer health as the elderly become less active, less independent and more prone to chronic degenerative diseases. For many, ageing is accompanied by decreased quality of life arising from many disabilities and chronic degenerative diseases including dementia, arthritis, osteoporosis, diabetes, cancer and circulatory diseases. Attention must therefore be given to increasing the quality of life.

### ***Factors affecting health***

The interaction of genetics and environment ("nature and nurture") is the basis of all health and disease. The state of people's health is the product of their genetic endowment, their age, nutrition, other aspects of lifestyle (such as physical activity and smoking habits), social environmental factors (such as housing conditions, sanitation and hygiene) and many other aspects of their social and cultural environment, such as stress, working conditions and family support.

An individual's metabolism is determined mainly by his or her genes. Genetic factors interact with nutrients, and

knowledge of these interactions at the molecular level is increasing as a result of intense research. In recent years, the genetic basis of several inherited diseases has been identified (e.g. familial hypercholesterolaemia, some types of cancer), confirming that the nature of the genes can predispose to the development of certain diseases in later life.

Whereas the relatively rare “genetic diseases” result from defects in single genes, the techniques of molecular biology are now revealing genetic variations that influence susceptibility to common multifactorial diseases including coronary heart disease, hypertension, diabetes, osteoporosis, cancer and arthritis. These conditions are almost certainly not due to defects in a single gene but to small changes in many genes.

Such genetic variance in a population explains why people are not susceptible to chronic diseases to the same degree even though their environments and diets may be similar.

### ***Current health status in Europe***

Health status can be assessed by measuring life expectancy, mortality rates, illnesses and disabilities, and recording subjective perceptions of “health” and various measures of the quality of life. Life expectancy at birth has frequently been used as the main global indicator of health as it relates to mortality patterns in populations. A major achievement of the 20th century has been the doubling of life expectancy at birth, which has now reached (with some regional variations) averages of 70 and 76 years in Eastern and Western Europe, respectively. In the West it ranges from 74.4 years in Portugal to 78.8 years in Iceland, where it has surpassed the World Health Organization’s “Health for All” target of 75 years. Life expectancy for women is in general several years longer than for men. Increased life expectancy and birth control measures have resulted in

an increasing proportion of elderly people in the population. This has important consequences for public health budgets.

According to European mortality statistics, major health problems are cancer, diabetes and cardiovascular diseases. Although death rates from the latter are declining steadily in Western European countries, the reverse is true for many Eastern European countries, and this is a cause for concern. Lung cancer is a typical example of a disease that can be reduced substantially by a single lifestyle change, namely, a reduction in smoking.

The increasing prevalence of diabetes, osteoporosis and obesity and of different forms of cancer, and the still high level of cardiovascular diseases, in the population are of major concern. Obesity (see Chapter 3) may increase the chance of developing other chronic diseases including diabetes, hypertension, stroke, coronary heart disease (CHID), arthritis and possibly some forms of cancer.

In industrialized countries with a seasonally independent food supply and a wide range of affordable high-quality foods, classic deficiency diseases from undernutrition, which are common in developing countries have been largely eliminated as public health problems. Nevertheless, diseases associated with some kind of nutritional imbalance still exist in Europe, and these will be described later in this chapter. There is increasing concern about undernutrition, especially in childhood, in many Eastern European countries.

### ***What is nutrition?***

Nutrition is the sum total of the processes involved in taking in nutrients and assimilating them into the body so that the body functions properly and health is maintained. Nutrition, however, means more than just the adequate provision of essential major and minor nutrients; there is

more to eating than mere nourishment. It is an important source of pleasure as well as social interaction and so has cultural significance. Throughout Europe characteristic regional traditions, cuisines, and food cultures have evolved to satisfy these needs.

The intake of food and water and the availability of oxygen are basic determinants for the proper functioning of the human body. Diet provides fuel as well as components for the essential structural elements of the body. Together with appropriate physical activity and suitable hygiene, these represent the main contributors to good health. The balance between food intake that provides fuel to the body (energy intake) and physical activity (energy expenditure) is important for well-being (see Chapters 3 and 9), although within limits, the body can adapt readily to changes in food supply and energy demands.

### ***Nutrient requirements and healthy eating***

Healthy nutrition requires appropriate dietary intake of energy in the form of macronutrients as well as the adequate intake of essential nutrients, comprising vitamins, trace elements, minerals, essential fatty acids and essential amino acids. A person's requirements for the different nutrients are related to his or her energy requirements, age, height and weight. They will differ according to different physiological conditions, such as whether the person is growing or not and whether, for example, a woman is pregnant or lactating. Lifestyle factors such as level of physical activity, stress, smoking and alcohol consumption also affect nutrient requirements. It is almost impossible to establish an individual's precise nutrient requirements, and each individual certainly has different requirements. "Dietary allowances" can therefore give only general guidance about population needs.

No individual food contains all nutrients in the optimum quantities and in the correct ratios essential for maintaining health under all conditions, with the possible exception of mother's milk during a short period of early life.

Therefore, healthy nutrition is possible only by eating a variety of different foods to maintain the required balance of nutrients. In this sense there are no good or bad foods: each has its part to play in providing this balance, but clearly some foods are better suppliers of certain nutrients than others.

### ***Current nutrition status in Europe***

Malnutrition has traditionally been regarded as the outcome of a lack of sufficient energy or specific nutrients (undernutrition). Although widespread undernutrition is no longer seen in Europe, some nutritional deficiencies nevertheless still exist.

Iron and iodine deficiencies exist in different parts of Europe and in different population groups. The former may be especially prevalent among menstruating women. The mean prevalence of anaemia in women between the ages of 15 and 49 is estimated to range from 10% in Western Europe to 17% in the economies that are still developing. The prevalence of anaemia in pregnant women is a reasonable approximation of the rate at which anaemia occurs in young children. Thus, about 7 million Western European children under 5 are probably anaemic from iron deficiency, a condition that is likely to continue during the early school years. The situation is likely to be more serious in Eastern Europe, but there are fewer data. Iron deficiency may impair learning ability among affected school children. A primary source of dietary iron is meat, but where meat consumption is considered inappropriate or meat is unavailable, new approaches to iron fortification have the potential to reduce iron deficiency in Europe.

Iodine deficiency diseases have been eliminated in European countries where universal salt iodization has been implemented, such as Switzerland and Austria, or where seafood is a dietary staple, such as the Scandinavian countries. With the wider availability and use of iodized salt in other countries, the chances of preventing and eliminating iodine deficiency disorders in Europe have increased.

A significant number of cases of rickets is still being reported in Northern Europe, pointing to a low vitamin D status. Vitamin D status is often low in the elderly and in Asian immigrants to Europe. Many nutritionists think a case can be made for supplementation programmes in certain situations where exposure to sunlight is inadequate.

Whereas the classic deficiency diseases are relatively easy to recognize and, in theory, straightforward to eliminate, there is more general concern about the potential, and less easily recognized, effects of deficiencies of a wide variety of nutrients including folates, magnesium, copper and antioxidants, among others. These are not generally within the scope of this concise monograph but the ramifications of inadequate intakes of antioxidants, for example, are discussed in Chapters 4 and 5.

Much more common than undernutrition in Europe are the results of overnutrition: the consumption of too much food energy. There is particular concern about high average intakes of fat, and some results of this are more fully described in other chapters, particularly Chapters 3 and 4.

Eating disorders such as bulimia and anorexia nervosa are not uncommon in adolescent girls (and becoming more common in boys) as a result of extreme nutritional and behavioural disorders. These can result in unbalanced nutrition (bulimia) or undernourishment (anorexia).

## ***Food supply and lifestyle***

Changes in lifestyles, urbanization and industrialization, in the structure of the family and in demographic trends during this century have also brought about changes in the food supply. Perhaps even more drastic is the reduction in daily physical activity partly as a result of the trend towards more sedentary occupations and the adoption of sedentary leisure time pursuits. Many traditions of family life are changing as more mothers go out to work and spend less time on food preparation. Increasingly, food preparation and cooking have been moved from the kitchen to the food-processing industry and to caterers in canteens and restaurants. The importance of factors such as ease of local production has decreased with improvements in food transport and distribution.

Developments in food processing have helped manufacturers to respond to today's dynamic lifestyles and the changing demands of the consumer. Now consumers can choose from a rich assortment of affordable foods available throughout the year. Choices include the traditional ranges as well as a wide selection of ready-to-eat meals including frozen or chilled menus and special nutritionally modified foods. The contribution of the food industry (both manufacturers and food service operators) is crucial to achieving the objectives of improving the quality of eating in a population.

Such contributions include not only the provision of a variety of high-quality nutritious foods, specifically fortified where appropriate, but also the clear labelling of foods with nutritional content.

## ***Eating for a healthy lifestyle: current trends***

Despite incomplete information and many differences of opinion about how changes in eating habits may influ-

ence public health, it is nevertheless possible to discern some consensus about healthy eating. There is little disagreement that individuals should eat a balanced and varied diet, decrease the consumption of total fat, increase the consumption of fruit, vegetables and cereal grains and maintain energy balance. Physical activity on a regular basis, even at moderate intensity, improves physical fitness and helps maintain a stable body weight and is to be encouraged. However, the full benefit of nutritional recommendations can be expected only with a move from the sedentary lifestyle towards a more active lifestyle on a day-to-day basis.

National nutrition and health surveys and food supply data from different European countries have shown that some aspects of this advice have already been taken up by the population. In particular, there have been increases in the consumption of vegetables and especially fruits in some European countries. Nevertheless, groups having a less than adequate supply of minerals and vitamins or those with a higher demand for antioxidant vitamins (for example, smokers) undoubtedly need additional appropriate information on the best ways to improve diets and lifestyles. Food manufacturers have the opportunity to contribute to improved choice through the provision of a wide variety of fortified foods.

### **Further reading**

Anderson GH, (ed.) Diet and behavior: multidisciplinary approaches. *ILSI Human Nutrition Reviews*. Heidelberg and London: Springer Verlag, 1990

Garrow JS, James WPT, (eds.) Human nutrition and dietetics, 9th ed. Edinburgh: Churchill Livingstone, 1993

International Life Science Institute. Present knowledge in nutrition 7th ed. Washington, DC: ILSI Press, 1996

Langseth L. Nutritional epidemiology, possibilities and limitations. ILSI Europe Concise Monograph Series, Brussels and Washington, DC: ILSI Europe/ILSI Press, 1996

National Research Council (US). Diet and health: implications for reducing chronic disease risk. Washington, DC: National Academy Press, 1989

Simopoulos AP, (ed.) Declaration of Olympia on nutrition and fitness. *Journal of the American Dietetic Association* 1992,92:1282–3

Simopoulos AP. Genetic variation and nutrition. *Nutrition Today* 1997;30:157–67, and 168–206

World Health Organisation. Health in Europe: the 1993/1994 Health for All monitoring report. WHO Regional Publications: European Series No 56. Copenhagen: WHO, 1994

### 3. DIETARY FAT AND ENERGY BALANCE

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#### *Energy balance and obesity*

The chemical energy we ingest in food is used to perform three kinds of work: mechanical (muscular contraction), electrical (maintaining ionic gradients across membranes) and chemical (synthesis of new molecules). Some energy is dissipated as heat during these interconversions. Energy balance means the difference between energy intake and its utilization. If energy balance is positive over a period of time, the excess energy is deposited as fat in adipose tissue and eventually the individual becomes obese. A state of obesity is generally defined as a body mass index (BMI = weight in kg/height in m<sup>2</sup>) greater than 30. A normal range of BMI is considered to be 20–25 and the 25–30 region is regarded as “overweight”.

#### *Obesity and risk of chronic disease*

It is now generally recognized that obesity is a health hazard. No single disease accounts for all of the excess mortality among obese people, but obesity increases the risk of adult onset diabetes and high blood pressure and exacerbates a tendency to have higher than normal concentrations of lipids in the blood (hyperlipidaemia). Even though coronary heart disease (CHD) is often considered to be the main condition associated with obesity, the additional heart disease risk associated with overweight or mild obesity is in fact small. Severe obesity (BMI > 35) is associated with hypertensive heart disease, congestive heart failure, sudden cardiac death and stroke. Recent research has highlighted the importance of body fat distribution as distinct from total body fat: a higher risk is associated with a fat distribution centred around the abdomen compared with the hips and thighs.

#### *Fat intake: a major determinant of body weight*

With few exceptions, epidemiological studies have shown a positive association between fat intake and the degree of obesity in populations (Figure 3.1). Three lines of physiological evidence support the concept that dietary fat intake has a major influence on body weight.

1. The excessive body weight gain of obese individuals is due mainly to an increase in the adipose tissue mass, 85% of which is composed of fat. The fatty acid composition of the adipose tissue fat is influenced by the fatty acid composition of the diet, suggesting that dietary fat makes an important contribution to body weight gain in obese subjects.
2. Immediately after consumption of a meal rich in fat, the absorbed fat is transported in the bloodstream to the adipose tissue in the form of particles called chylomicrons. Metabolic studies show that insulin secreted in response to such a meal inhibits the release of fatty acids from adipose tissue and enhances the activity of the enzyme lipoprotein lipase (LPL). This enzyme is located in the blood capillaries supplying the adipose tissue, where it hydrolyses the triglycerides in chylomicrons and favours fatty acid uptake by adipose cells. This research demonstrates that the major metabolic fate of dietary fat is not uptake by energy-consuming tissues and oxidation but storage in adipose tissue. This concept is also supported by the observation that after a high-fat meal, fat oxidation is not stimulated but, rather, carbohydrate and protein oxidation are enhanced.
3. In human beings, the synthesis of fatty acids from glucose is not an important metabolic pathway. This is due to the fact that the activities of enzymes that convert carbohydrate into fatty acids are intrinsically low and, moreover, are normally “switched off” because of the considerable amount of fat that is normally

present in the Western diet. Even after consuming a high carbohydrate breakfast, only 2% of the palmitic acid (one of the commonest fatty acids) present in the very-low-density lipoproteins produced by the liver is derived by synthesis from carbohydrate. Thus, dietary carbohydrates are normally less important than dietary fats as contributors to an increase in body fat content. However, when carbohydrates represent a very large proportion of energy intake and intake exceeds overall energy expenditure, an increase in body fat is bound to occur.

Additional observations help explain the association between dietary fat and obesity.

A high-fat diet tends to stimulate overeating. Subjects spontaneously have a higher energy intake with a higher-fat meal than with a high-carbohydrate/low-fat meal. Most studies have concluded that high-fat meals induce less intense satiety than high carbohydrate meals of equal energy value. Thus, when people are given meals equal in energy but rich in either fat or carbohydrate, and then allowed free access to foods of their own choice in a subsequent meal, they tend to consume less energy after the high-carbohydrate than the high-fat meal.

However, several studies have found that some individuals are much better at compensating for the energy consumed in the first meal, so that their overall energy consumption would be no greater on a high-fat than on a high-carbohydrate diet.

The overconsumption of energy stimulated by fat may be explained in part by another important attribute of fat, namely, its high “energy density”. In other words, it provides a concentrated form of energy, with 9 kcal of energy for every gram of fat compared with carbohydrate’s 4 kcal. High-carbohydrate foods tend to be bulkier, making it more difficult to consume as much energy as with

fat-rich foods. In experiments where fat was varied to contribute up to 60% of energy, spontaneous energy intake was positively correlated with the energy density of the food.

Fats often improve flavour perception and impart a pleasing texture to foods, thereby improving palatability. Many obese individuals express a liking for fatty foods and may choose such foods in preference to foods with a different taste and texture. Recent studies indicate that a preference for high-fat foods may in part be of genetic origin. The fact that for many people, excess fat intake results in fat deposition in adipose tissue without feedback on subsequent food intake clearly suggests that a preference for high-fat foods is a major mechanism for inducing obesity. By contrast, high-carbohydrate meals promote carbohydrate oxidation, and carbohydrate balance is usually reached within 24 hours. Similarly, high-protein meals stimulate protein oxidation with no net gain of proteins. This means that people are less likely to become obese if their diets are rich in carbohydrate or protein.

### ***Body weight regulation and fat balance***

Prevention of obesity requires that energy balance be maintained over a period of time. While carbohydrate and protein balances are usually adjusted within 24 hours, fat balance (i.e., the difference between fat intake and fat oxidation) requires a longer time to adjust and is thus the critical parameter of body weight regulation. There are two ways to avoid a positive fat balance: a moderate fat intake or increased fat oxidation.

Moderate fat intake, with a compensatory increase in the proportion of carbohydrate, is an effective means of avoiding body weight gain. Several randomized clinical trials reporting the effect of a calorically unrestricted low-fat diet on weight change showed modest weight reductions in the range of 3 to 4 kg after six months (Table 3.1). The low-

fat diet has therapeutic potential provided that people can maintain their weight loss for periods longer than a few months. The evidence available so far suggests, however, that weight loss occurs mainly within the first three months of the lowfat diet, but after that most people are less able to maintain their dietary changes in the long term.

Regarding the other side of the energy balance equation, namely, energy expenditure, there is much evidence to suggest that low levels of physical activity are important in the development of obesity (see also Chapter 9). Some researchers have concluded that to be useful the exercise must be of high intensity. Most, however, argue that exercise of low intensity and of long duration is to be preferred, since it favours mobilization of adipose tissue fat and the utilization of the fatty acids that are released as an energy substrate by the muscles. Thus, it may play an important role in everyday fat balance.

It must be emphasized that the duration of low-intensity exercise must be of several hours per day to have a significant influence on fat balance. An individual spends about 3 kcal/minute while walking at a rapid pace; at rest, energy expenditure is close to 1 kcal/minute. Thus, walking induces an increase in energy expenditure of 2 kcal/minute; a 3-hour walk results in a supplementary energy expenditure of 360 kcal, which corresponds to 40 g fat. Consequently, a 3-hour exercise period should be carried out each day for 25 days to lose 1 kg fat. These theoretical calculations do not take into account a possible increase in appetite that could to some degree compensate for the small amount of lipid oxidized by exercise; the duration of the exercise is important and is further discussed in Chapter 9. Another beneficial effect of exercise is to increase the basal metabolic rate by increasing lean body mass; whether this is accompanied by an increased capacity to oxidize fat is still an open question that needs further research.

Notwithstanding the controversy over whether low- or high-intensity exercise is to be preferred, these examples illustrate the need for a dramatic change in lifestyle if a sustained fat loss, or more importantly prevention of fat gain, is to be achieved solely by exercise.

However, moderate activity has other health benefits (see Chapter 9) so all levels and types of activity are to be encouraged, combined with a diet that does not include too much fat. A major problem in today's society is that exercising for several hours a day may not be practical, and recommendations for exercising must clearly take into account factors such as age and general fitness.

### *The influence of alcohol*

Alcohol may provide up to 10% of an adult's energy intake and considerably more in some populations. It provides an immediate source of energy and consequently decreases fat oxidation. The overall effect of alcohol in the diet, if taken in addition to normal fat intake, may therefore favour lipid storage. However, as Chapter 8 makes clear, some published studies appear to have found no influence of alcohol on weight gain, and this is a matter that requires more research.

### *Conclusions*

In most European countries, the proportion of food energy derived from fat is close to 40%, which is higher than the upper limit of the recommended fat intake (i.e., 30–35%). Although some evidence suggests that there may have been a marked increase in fat consumption during the first part of this century during the past 30 years, fat consumption has not increased as a proportion of energy whereas obesity has increased markedly in most European countries suggesting an important contribution from lack of exercise. During thousands of years of human evolution, the capacity to store fat was likely advantageous

for survival when food was limited. Nowadays this metabolic feature becomes a risk for people living in affluent countries, and obesity results from a chronic imbalance between fat intake and fat oxidation.

Moderate fat and low alcohol intakes, consumption of a diet with a high proportion of mainly complex carbohydrate to fat and enhanced physical activity are the best recommendations to avoid body weight gain. These dietary principles and behavioural modifications are not new since they correspond to the lifestyle of most people at the beginning of this century. In the 100 years since then, humans have not been able to adapt their behaviours and lifestyles to rapid environmental change. It is therefore important that people understand the reasons why body weight gain can occur so easily. A better understanding of these mechanisms should help in the prevention and treatment of obesity.

### **Further reading**

Blundell JE, Burley VJ, Cotton JR, Lawson CL. Dietary fat and the control of energy intake on meal size and postmeal satiety. *American Journal of Clinical Nutrition* 1993;57(suppl):772S–8S

Flatt JP, Ravussin E, Acheson KJ, Jéquier E. Effects of dietary fat on postprandial substrate oxidation and on carbohydrate and fat balances. *Journal of Clinical Investigations* 1985;76:1019–24

Gurr M. Nutritional and health aspects of sugars: evaluation of new findings. ILSI Europe Concise Monograph Series. Brussels and Washington, DC: ILSI Europe/ILSI Press, 1995

Jéquier E. Nutrient effects: postabsorptive interactions. *Proceedings of the Nutrition Society* 1995;54:253–65

Lissner L, Heitmann BL. Dietary fat and obesity: evidence from epidemiology. *European Journal of Clinical Nutrition* 1995;49:79–90

Prentice AM, Jebb SA. Obesity in Britain: gluttony or sloth? *British Medical Journal* 1995;311:437–39

Rolls BJ, Kim-Harris S, Fischman MW, et al. Satiety after preloads with different amounts of fat and carbohydrate: implications for obesity. *American Journal of Clinical Nutrition* 1994;60:476–87

Tremblay A, Almeras N, Boer J, et al. Diet composition and postexercise energy balance. *American Journal of Clinical Nutrition* 1994;59:975–9

Weststrate JA. Fat and obesity. *International Journal of Obesity*, 1995;19:S38–S43

## 4. DIETARY FAT AND CORONARY HEART DISEASE

### *Basic facts about coronary heart disease*

Coronary heart disease (CHD) is a major health problem in most industrial countries. The predominant clinical symptoms are myocardial infarction (heart attack), angina and sudden cardiac death. In CHD the arteries supplying blood to the heart are narrowed by the disease of atherosclerosis, in which “plaque” builds up under the inner lining of the artery over many decades (Figure 4.1). Later, the plaque may rupture. This triggers a series of processes to repair the wound, and a blood clot or thrombus is formed. If this completely blocks the artery (thrombosis), the blood, and thus the oxygen supply to the heart, is cut off and the result is a heart attack that may be fatal. The amount and type of fat in the diet may influence both the process of atherosclerosis (which takes many years) and that of thrombosis (which may occur in hours or even minutes).

### *Risk factors*

Although some people are more genetically predisposed to develop CHD than others, environmental factors have a major impact on the risk of CHD. Immigrant groups from countries with low CHD rates often become more susceptible to the disease in their new host country, and during this century there have been large changes in deaths from CHD which cannot be explained by genetic factors. Many risk factors have been identified. Those factors with the strongest scientific support are a high concentration of low density lipoprotein cholesterol in the blood, elevated blood pressure and smoking. Others, which may be related to these in complex ways, include obesity (see Chapter 3), diabetes, physical inactivity (Chapter 9), a high concentration of certain blood-clotting fac-

tors (see a later section in this chapter) and a poor capacity for the body to defend itself against oxidation (Chapter 5). Recent studies have implicated infection with the organism *Chlamydia pneumoniae* as a possible risk factor. There is currently much interest in the influence of maternal nutrition on fetal growth in relation to later CHD risk.

Dietary fat is widely accepted as one of the fundamental environmental risk factors, operating through blood cholesterol and possibly other physiological and biochemical functions. This view originates from a classic epidemiological study by Ancel Keys and coworkers, the Seven Countries Study. This study found that the differences in mean blood cholesterol concentrations among 16 cohorts could be predicted from the intake of saturated fatty acids and that differences in CHD rates were consistent with differences in saturated fatty acid intakes and blood cholesterol concentrations. The differences in blood cholesterol concentrations associated with diet were, however, much larger than has been observed in subsequent controlled metabolic studies. Evidently, other environmental or lifestyle factors played a role in influencing cholesterol concentrations in this study. Nevertheless, the Seven Countries Study has triggered much research, both epidemiological and experimental, on the link between dietary fat, blood cholesterol and CHD.

### *Diet, blood cholesterol and CHD*

A large body of epidemiological data indicates a direct association between the total concentration of cholesterol in the blood and the incidence of CHD. The evidence includes studies within populations as well as comparisons between populations. The strongest evidence for a link between cholesterol concentrations and CHD comes from randomized clinical trials. Such studies have shown that lowering blood cholesterol concentration can reduce the risk of CHD in persons without prior CHD (primary pre-

vention trials) as well as the risk of recurrent CHD in patients (secondary prevention trials). From the results of clinical trials of a relatively short duration, it has been calculated that each 1% reduction in total cholesterol concentration should result in about a 2% reduction in CHD incidence. Observational studies suggest that the reduction in CHD risk with long-term cholesterol lowering may be substantially greater.

The idea that diet is related to CHD has relied largely on the observation that blood cholesterol concentrations predict CHD risk. In numerous metabolic studies on the effect of diet, blood cholesterol has served as an intermediate marker for CHD. The evidence that links diet directly to CHD, however, is limited. Cholesterol lowering in clinical trials was achieved by diet, drugs or a combination of the two. Modern drugs (statins) have proved to be the most powerful cholesterol-lowering agents and to reduce CHD risk substantially. Nevertheless, analyses that pool the results of dietary trials demonstrate that dietary therapy alone can also be effective (see Further reading).

The evidence suggests that the type of fat consumed—that is, replacement of saturated by unsaturated fatty acids—rather than reducing the total amount of fat is important in lowering CHD risk. Animal experiments support the view that diets rich in saturated fatty acids result in elevated blood cholesterol concentrations and eventually severe atherosclerosis.

It is important to distinguish between the types of cholesterol. About 70% of total blood cholesterol is carried in low-density lipoproteins (LDL). This form of cholesterol has been found to increase CHD risk: the higher the blood LDL cholesterol concentration, the greater the risk. A large proportion of the remaining blood cholesterol is carried in high-density lipoproteins (HDL), which, by contrast, protect against CHD. Epidemiological studies have also

shown that people with high fasting concentrations of triglycerides (carried together with some cholesterol on very-low-density lipoproteins [VLDL]) have an enhanced CHD risk. However, it is not clear whether a high triglyceride concentration is itself a causal factor or simply coincides with other risk factors such as low HDL levels or proneness to diabetes. In addition to the risk associated with fasting concentrations of LDL or VLDL, much research is now focusing on the concentrations of blood lipids immediately after a meal. This is discussed in a later section of this chapter.

### ***Basic facts about dietary fats***

Dietary fats consist largely of fatty acids which may differ from each other in their chain length (number of carbon atoms), the number and position of double bonds and the geometry (*cis* or *trans*) of the double bonds. Table 4.1 shows the fatty acid intakes of men in four countries in the 1960s collected during the Seven Countries Study. These data cannot be taken as representative of current intakes because of changes in eating patterns over the intervening years. In particular, the intake of linoleic acid in Western Europe and the United States has risen in recent decades, with a commensurate decrease in saturated fatty acids intake. Nevertheless, Table 4.1 serves to illustrate how the relative contribution of different fatty acids to dietary intakes differs widely among countries. These data were collected during a similar period by comparable methods; although current data would be preferable, there is a major problem in obtaining up-to-date comparative information on fatty acids because of the widely differing methods used to collect and analyse data in different countries.

Most diets contain a variety of saturated fatty acids with different chain lengths. The major saturated fatty acid in the diet is palmitic acid (16:0)\*, followed by stearic (18:0), myristic (14:0) and lauric (12:0) acids. Short- and medium-

chain fatty acids (4:0–10:0), mainly occurring in dairy fat, palm kernel oils and coconut oils, also contribute to total intake of saturated fatty acids.

Palmitic acid is found in all edible fats and oils and is particularly abundant in palm oil (a vegetable oil used in frying fats, hard margarines and baked goods) and in butter, milk, cheese and meats. Stearic acid is found predominantly in cocoa butter used in chocolate and in the fats from beef and sheep. Lauric oils, such as coconut and palm kernel oils used in confectionery and dairy fats contain relatively high amounts of lauric and myristic acids. The major monounsaturated fatty acid in human diets is oleic acid (*cis*-18:1, *n*-9), and the principal polyunsaturated fatty acid is linoleic acid (*cis,cis*-18:2, *n*-6); each has a chain length of 18 carbon atoms. Liquid vegetable oils such as soybean, corn and sunflower oils are rich in linoleic acid. Oleic acid is present in all types of edible fats and oils but olive and rapeseed oils are particularly rich sources.

*Trans* isomers of monounsaturated fatty acids (*trans*18:1) are produced during the industrial hydrogenation of polyunsaturated vegetable oils or by the biohydrogenation of polyunsaturated fatty acids in the rumen of ruminant animals (cows and sheep). Major sources of *trans* unsaturated fatty acids are partially hydrogenated fats in the form of shortenings and frying fats used in industrial food preparation and in fast-food restaurants, and hard margarines. However, the level of *trans* fatty acids in these food fats may vary widely depending on the raw materials and processes used.

### ***Dietary fats and blood lipoproteins***

The best information regarding the effects of fatty acids on blood lipids and lipoproteins derives from metabolic

studies in human beings. Fatty acids cannot be simply classified as “cholesterol lowering” or “cholesterol raising”. The addition of a particular fatty acid to the diet involves removing some other energy-yielding food component; otherwise subjects will gain weight. Thus, the effect of a certain amount of fatty acid can be expressed only relative to a similar quantity of energy provided by another dietary component that serves as a reference.

The choice of the reference component is arbitrary. Figure 4.2 indicates changes that would occur in serum LDL and HDL when 1% of dietary energy in the form of carbohydrate is replaced by a particular class of fatty acid. The choice of a reference can be a major source of confusion. For example, the effect of olive oil—predominantly oleic acid—on LDL cholesterol is small when it replaces carbohydrates, but olive oil clearly lowers LDL cholesterol when it replaces saturated fatty acids.

Saturated and *trans* unsaturated fatty acids strongly raise LDL cholesterol, and oleic and linoleic acids moderately lower it relative to carbohydrates. Linoleic acid is slightly more effective in lowering LDL than oleic acid. The long-chain highly polyunsaturated fatty acids of the *n*-3 family from fish oil have relatively little effect on LDL cholesterol (not shown in Figure). Fatty acids may raise HDL cholesterol when they replace carbohydrates, but notable exceptions are the *trans* fatty acids; unlike other fatty acids, they lower HDL cholesterol relative to carbohydrates. Surprisingly, eating fat decreases the concentration of triglycerides in fasting blood; long-chain *n*-3 polyunsaturates from fish are particularly effective in this regard.

As a result of the effects shown in Figure 4.2, oils rich in linoleic acid produce the most favourable lipoprotein pro-

\* The number before the colon indicates the number of carbon atoms in the fatty acid chain, and the number after the colon indicates the number of double bonds. Double-bond geometry is indicated by the prefix *cis* or *trans*. The family to which an unsaturated fatty acid belongs is indicated by *n*-3, *n*-6 or *n*-9.

file in regard to CHD risk: they simultaneously lower LDL and raise HDL. However, the difference between their effects and those of monounsaturated oils rich in oleic acid is small. Fats rich in saturated fatty acids are unfavourable in risk terms because they raise LDL cholesterol. The saturated fatty acids used in the studies depicted in Figure 4.2 comprise lauric (12:0), myristic (14:0), palmitic (16:0) and stearic (18:0) acids. Stearic acid has been found to be “neutral” for total and LDL cholesterol compared with carbohydrates, whereas lauric, myristic and palmitic acids are the fatty acids responsible for the cholesterol-raising effect of saturated fat. These three fatty acids make up 70% of total fatty acids in the diets in the metabolic studies (Figure 4.2), and they represent the major portion of total saturated fatty acids in most practical diets in European countries (Table 4.1).

Some investigators have reported that palmitic acid from palm oil does not raise total and LDL cholesterol, but in most well-controlled studies, fats rich in palmitic acid have clearly raised LDL cholesterol.

Myristic acid raises cholesterol more than lauric and palmitic acids, but for practical purposes these three cholesterol-raising fatty acids may be conveniently grouped together. Although stearic acid does not raise total and LDL cholesterol, there is some evidence that it may lower HDL and so might not be as favourable for the lipoprotein profile as oleic or linoleic acids.

As well as raising LDL and lowering HDL cholesterol, the unfavourable effects of *trans* unsaturated fatty acids are compounded by their apparent elevation of lipoprotein (a), another type of blood lipoprotein that is associated with atherogenesis. Many people eat no more than a few grams of *trans* unsaturated fatty acids per day (Table 4.1). Thus, the overall effect of *trans* fatty acids on CHD risk in the population may be less important than that of the satu-

rated fatty acids, which are consumed in much higher amounts. In summary, replacement of hard fats rich in saturated and *trans* unsaturated fatty acids with soft fats or liquid oils rich in oleic or linoleic acids improves the lipoprotein risk profile for CHD.

It is emphasized that the above-mentioned effects derive from strictly controlled dietary studies. The effects of dietary fatty acids on blood cholesterol observed in large trials with free-living subjects receiving dietary advice have generally been smaller. This may have been due to poor dietary compliance or to the complex interactions of uncontrolled environmental variables. Thus, dietary advice may not be the most effective way to improve blood lipoprotein profiles and reduce CHD risk in a population. Changing the fatty acid composition of widely consumed food fats is an alternative strategy.

### ***Dietary fat and thrombosis***

In contrast to the focus on lipoproteins and atherosclerosis or overall CHD, there is comparatively little information on the effects of dietary fats on thrombosis, mainly because it is difficult to study in living human beings. Thrombosis is a complex process of aggregation of blood platelets and the formation of a clot (thrombus) that involves the participation of many blood proteins (clotting factors).

Several metabolic studies on platelet function *in vitro* and on clotting factors suggest that unsaturated fatty acids promote the thrombotic process less than saturated fatty acids do, but the evidence is not conclusive. For example, a high concentration or activity of a clotting factor called factor VII is regarded as a putative risk factor for CHD. The total amount of dietary fat markedly raises the concentration of factor VII after a meal, whereas the fatty acid composition of the meal seems to be without effect. There

are some indications that dietary fat may raise the fasting concentration of factor VII.

Other research has shown that the activity of factor VII is closely linked to the concentration of blood triglycerides after a meal. Normally, the triglycerides in the circulation immediately after a high-fat meal are cleared rapidly, but in some individuals they remain high for longer. They also remain relatively high in overweight or diabetic persons, and this may explain the increased risk associated with these conditions. Some clinical trials and epidemiological studies have shown that regular consumption of fish reduces the risk of CHD (see Further reading). It has usually been assumed that this is due to their content of *n*-3 polyunsaturates, but whether through an effect on triglyceride concentrations, platelet aggregation or clotting factors, or by some other mechanism, remains unclear.

### **Recent research developments**

During the past decade, two lines of research in particular have influenced thinking about the role of dietary fats in CHD. The first is the discovery that a form of LDL chemically modified by oxidation, and not the unmodified form, might be important in the development of atherosclerosis. Because *in vivo* tests to measure oxidative modification of LDL are not yet available, we do not know exactly how dietary fat may influence this putative risk factor. In tests *in vitro*, diets very rich in linoleic acid produced LDL that were more susceptible to oxidation than did diets rich in *cis* monounsaturated fatty acids. This has shifted the emphasis in dietary recommendations to lower CHD risk from the *n*-6 polyunsaturates towards the *cis*

monounsaturates. However, the relevance of these experiments to the living body is not entirely clear. Furthermore, animal experiments and clinical trials with human subjects show a lower risk of CHD when linoleic acid replaces saturated fatty acids, which are not prone to oxidation.

It is important to note that most vegetable oils rich in linoleic acid naturally contain high levels of vitamin E, an antioxidant that may counteract the possible prooxidant activities of linoleic acid. Recent epidemiological studies and one intervention trial suggest that a high intake of vitamin E may indeed lower the risk of CHD.

The second line of research is concerned with the idea of "insulin resistance". This means that metabolic reactions dependent on the action of insulin are less responsive than they should be to the hormone. Among these reactions is the uptake of fat-rich lipoproteins by adipose tissue after a meal, which results in elevated blood concentrations of small, dense LDL particles. These particles have been associated with increased CHD risk, even when overall blood cholesterol is within the normal range. This abnormality may be influenced by dietary fat: increasing the intake of *n*-3 polyunsaturates reduces the concentration of fat-rich lipoproteins and small, dense LDL after a meal. Insulin resistance is also associated with increased blood pressure, an important risk factor for CHD. However, there is little sound scientific evidence for a strong effect of either the amount or type of dietary fat on blood pressure.

## Further reading

Ashwell M, (ed.) Diet and heart disease—A round table of factors. London: *British Nutrition Foundation*, 1993

Barker DJR Fetal origins of adult disease. *British Heart Journal* 1993;69:195–6

Burr ML, Fehily AM, Gilbert JF, et al. Effects of change in fat, fish and fibre intakes on death and myocardial reinfarction: Diet and Reinfarction Trial (DART). *Lancet* 1989;ii:757–60

Griffin BA, Zampelas A. Influence of dietary fatty acids on the atherogenic lipoprotein phenotype. *Nutrition Research Reviews* 1995;8:1–26

Gurr MI. Dietary lipids and coronary heart disease: old evidence, new perspective. *Progress in Lipid Research* 1992;31:195–243

Katan MB, Zock PL, Mensink RP. Dietary oils, serum lipoproteins and coronary heart disease. *American Journal of Clinical Nutrition* 1995;61(suppl):1368S–73S

Knapp HR. Dietary fatty acids in human thrombosis and haemostasis. *American Journal of Clinical Nutrition* 1997;65(suppl):1687S–98S

Steinberg D, Parthasarathy S, Carew TE, et al. Beyond cholesterol: modifications of low density lipoprotein that increase its atherogenicity. *New England Journal of Medicine* 1989;320:915–24

Truswell AS. Dietary fat—some aspects of nutrition and health and product development. ILSI Europe Concise Monograph Series. Brussels and Washington DC: ILSI Europe/ILSI Press, 1995

## 5. ANTIOXIDANT NUTRIENTS

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### Reactive oxygen species and free radicals

Oxygen is essential to human life, but paradoxically, highly reactive forms of oxygen are produced during normal aerobic metabolism which have the potential to cause extensive damage to the body. It has been proposed that this damage is an important contributor to ageing, and there is evidence that many degenerative diseases associated with ageing, including cardiovascular disease, cancer, cataracts, a decline in the immune system and degenerative diseases of the nervous system, may involve activated oxygen among their causative factors.

The reactive types of oxygen are collectively termed “reactive oxygen species” (ROS). Many are “free radicals” (see Glossary), but the term ROS is used in this chapter. ROS are not always harmful; indeed, for example, they play a vital role in the destruction of pathogenic microbes by specialized blood cells called phagocytes, as well as other beneficial functions. However, because their chief characteristic in the health context is that they can attack several intracellular and extracellular constituents, thereby impairing cellular functions, their formation and activity must be controlled.

Recent research also indicates that several free radical species that contain nitrogen (“reactive nitrogen species”; RNS) also contribute to the detrimental processes in which ROS are involved.

### Antioxidant defence mechanisms

Fortunately, ROS are normally kept in check by the body’s intricate and diverse defence systems. These systems are complementary because they act on different ROS or in

different cellular compartments. For example, enzyme systems such as the superoxide dismutases, glutathione peroxidase and catalase are capable of removing ROS. These enzymes can also prevent their leakage from one cellular compartment to another, thereby minimizing damage to cellular components like DNA, protein and lipids, and prevent the formation of more active ROS by removing the reactants from which they are formed.

The effectiveness of the antioxidant defence system is dependent on an adequate dietary intake of foods containing antioxidants such as vitamins E and C and several essential trace minerals. Selenium, copper, manganese and zinc are involved in the structure or catalytic activity of the protective enzymes. The antioxidant status of the body can be considerably influenced by diet. Should the normal defence mechanisms be impaired by nutritional deficiencies, pathological conditions can occur. Other compounds which act as antioxidants in the body include glutathione, ubiquinone and uric acid, all of which are produced during normal metabolism.

### ***Oxidative stress***

Figure 5.1 illustrates the delicate balance between prooxidant factors and antioxidant defences. An increase in the production of ROS or a deficiency in the nutritional defence systems can disturb their balance, causing “oxidative stress”. Much research activity is focusing on the role of ROS and RNS in human disease and the measurement of sensitive indicators of antioxidant status and oxidative stress.

### ***Food sources of antioxidants***

There is now abundant evidence, both experimental and epidemiological, that the antioxidant nutrients and a wide range of non-nutrient substances in the normal diet can exert protective effects against several diseases. Many of

the non-nutrient compounds are of plant origin, and individual foods frequently contain as much as several percent by weight of these naturally occurring constituents. Plant leaves are exposed to visible and ultraviolet light and other radiation and are especially susceptible to damage by activated forms of oxygen. Hence there are numerous natural antioxidant constituents which can counter ROS. Similarly, all plant seeds have efficient systems to protect germination from ROS.

Much scientific research has focused on three key nutrients: vitamin E, vitamin C and the carotenoid pigments (e.g.  $\beta$ -carotene, which the body turns into vitamin A). These nutrients work together very effectively to scavenge the harmful ROS and RNS molecules that can cause damage and disease.

Vitamin E is the collective name for the tocopherols. These are major fat-soluble antioxidants found in all cellular membranes. They protect polyunsaturated fatty acids against oxidation. Good sources of vitamin E include vegetable and nut seed oils, wheat germ, vegetables, meat and fish. Vitamin C or ascorbic acid is the most important water-soluble antioxidant substance found in the body’s extracellular fluids. Fruits and vegetables, and especially citrus fruits, are good sources. Important dietary carotenoids include:  $\beta$ -carotene present in yellow and orange vegetables and fruits and dark green vegetables; lycopene, the red colour in tomatoes; and lutein, the dark green colour in broccoli and other leafy vegetables.

Fruit and vegetables are the principal sources of these major dietary antioxidants (Table 5.1) and there is now compelling evidence to support the assertion that beneficial effects result from consuming larger amounts of these foods. The focus here on vitamins C and E and  $\beta$ -carotene does not diminish the importance of the dietary minerals necessary for the activities of the metalloenzyme

antioxidants. However, in Europe there is little evidence for deficiencies of these metalloenzyme systems; moreover, most research to date has focused on vitamins C and E and  $\beta$ -carotene.

The plant world is full of antioxidant substances, and in addition to their individual effects, they interact synergistically. For example, vitamin C may reinforce the antioxidant effect of vitamin E *in vivo* by regenerating the active form of the vitamin after it has reacted with a free radical as it does *in vitro*. A positive message of special public health significance is to eat more fruit and vegetables, fresh and preserved, every day, and five or more portions (or “servings”) are recommended by several authorities.

Plant foods also contain polyphenolic compounds, such as those present in red wine, that can behave as antioxidants (Table 5.2). However, these have not been traditionally regarded as nutrients (although they may eventually be so) and are beyond the scope of this chapter.

## ***Antioxidants and disease***

### **Cardiovascular disease**

Evidence continues to accumulate that antioxidants may play an important role in reducing the risk of cardiovascular disease. It is now recognized that oxidized low-density lipoprotein (LDL, the main carrier particle for cholesterol in the bloodstream) is involved in the development of atherosclerosis, a progressive degeneration of arteries characteristic of cardiovascular diseases.

Oxidative modification of LDL particles in the damaged arterial walls (not in the plasma) results in structural changes which can contribute to the initiation and progression of atherosclerosis. This initial oxidation step has been shown to be inhibited by a high serum concentra-

tion of antioxidant nutrients such as vitamin E. Low concentrations of antioxidants within LDL particles are associated with an increased tendency for oxidation to occur within those particles, so that enhancement of the dietary intake of vitamin E may be expected to lower the risk of atherosclerosis. However, the levels of dietary vitamin E required to be protective and the relative roles of other antioxidants remain to be demonstrated.

A key discovery is that oxidized LDL particles are no longer recognized by normal LDL receptors on the surface of most cells throughout the body, but are bound instead by “scavenger receptors” on macrophages, which then engulf them. The macrophages become laden with fatty materials, primarily cholesterol and cholesterol esters to form “foam cells”. These are trapped in the damaged lining of the artery walls, creating atherosclerotic fatty streaks. The naturally occurring antioxidants, particularly tocopherols and ascorbate, may be beneficial as anti atherosclerotic agents because they prevent the formation of oxidized LDL.

It has also been suggested, and there is some evidence to support it, that antioxidants may positively influence various other heart disease risk factors, such as serum levels of high-density lipoprotein (HDL) cholesterol, platelet adhesion and hypertension. HDL has also recently been shown to be an antioxidant in its own right. Furthermore, it appears that antioxidants may slow the progression of heart disease and reduce the severity of angina, reperfusion injury and ischaemia. The immediate cause of tissue damage during a heart attack or stroke results from the supply of oxygen being cut off (ischaemia), and irreversible damage can be prevented only by the restoration of blood flow (called reperfusion). This latter action involves the reintroduction of oxygen, which can damage the heart tissues further because harmful ROS are formed during the reperfusion process.

An increasing body of mechanistic and epidemiological evidence now links dietary antioxidant intake with reduced risk of cardiovascular diseases. Definitive data on these potential benefits are likely to emerge over the next few years, including results of dietary intervention trials. Double-blind placebo-control trials are examining the effect of antioxidant supplements to the diet on risk of cardiovascular disease. Such information will allow public health recommendations which could further reduce morbidity and mortality from cardiovascular disease in most developed countries.

### Cancer

Cancer is a disease in which disorder occurs in the normal processes of cell division, which are controlled by the cell's genetic material (DNA). The transformation of a normal cell into a cancerous cell is believed to proceed through many stages over a number of years or even decades. The stages of carcinogenesis include initiation, promotion and progression. Preventing initiation is an important anti-cancer strategy, as are the opportunities to inhibit cancer throughout the latter stages. One of the most important contributors to cancer is considered to be oxidative damage to DNA. If a cell containing damaged DNA divides before the DNA can be repaired, the result is likely to be a permanent genetic alteration, the first step in carcinogenesis. Body cells that divide rapidly are more susceptible to carcinogenesis because there is less opportunity for DNA repair before cell division.

Although the mechanism of the protective effect is unclear, the fact that the consumption of fruit and vegetables lowers the incidence of carcinogenesis at a wide variety of sites is broadly supported. The epidemiological evidence suggests protection against a wide array of cancers (Table 5.3), particularly those of the respiratory and digestive tracts and, to a lesser extent, the hormone-related cancers.

A host of plant constituents could be responsible for the protective effects, and it is likely that several of them play a role under some circumstances. Most of the non-nutrient antioxidants in these foods are phenolic or polyphenolic compounds such as the isoflavones in soy beans, catechins in tea, phenolic esters in coffee, phenolic acid in red wine, quercetin in onions and rosmarinic acid in rosemary (Table 5.2).

Of the many anti-carcinogens already detected in plant foods, the antioxidant vitamins C and E and the pro vitamin  $\beta$ -carotene have received most attention. Although there has been considerable enthusiasm for the potential anti-carcinogenic properties of  $\beta$ -carotene, research findings suggest that several different carotenoids are likely to be associated with reduced cancer risks.

In two intervention trials to investigate the potential protective effects of  $\beta$ -carotene against cancer, an unexpected, significantly higher incidence of lung cancer was found among men taking supplements compared with those not taking additional  $\beta$ -carotene. These men were long-time heavy smokers and may represent a special case in that their lung cancer may have been initiated many years before the study took place. These results cause concern and need serious consideration and further investigation. They do not invalidate the concept of the importance of antioxidant nutrients but do underline the need to examine the relative influence of supplements of a single antioxidant nutrient (as distinct from complex mixtures of antioxidants in foods) as well as interactions between the effects of smoking, antioxidant nutrients and disease progression.

Vitamin C is known to interfere with the action of nitrites, and further dietary intervention studies are under way to test the ability of ascorbic acid to reverse precancerous

lesions of the stomach. Vitamin E is a lipid-phase scavenger of nitrite, oxygen and oxygen-derived free radicals. However, the evidence linking vitamin E and reduced cancer risk is still inconclusive.

The mechanisms for the protective effects of fruits and vegetables and the antioxidant nutrients appear to involve the early rather than the later stages of carcinogenesis. Hence, there are theoretical and practical reasons for suggesting that dietary advice to increase consumption should be started from an early age. The evidence for protective effects from individual antioxidant nutrients is less definitive, whereas the evidence for an overall nutritional benefit from increased fruit and vegetables consumption is overwhelming.

### **Eye diseases**

Oxidative processes have been implicated in cataract and diseases of the retina of the eye. These age-related diseases are major health problems, and recent evidence suggests that high dietary intakes of antioxidants may help delay or prevent them. The eye has defence systems which protect the lens from oxidative damage, but as people age the defence mechanisms become less effective and damage to lens proteins may become irreversible. Further research is needed to determine conclusively whether improved nutrition can reduce the risk of these common causes of blindness in the elderly.

### **Other diseases and pathological conditions needing further research**

*Rheumatoid arthritis.* The products of free-radical reactions have been detected in the blood and joints of patients with inflammatory diseases such as rheumatoid arthritis.

*Decreases in immune function.* The decrease in immunity that occurs with age may be partly offset by dietary antioxidant supplementation.

*Exercise-induced oxidative stress.* Exercise leads to increased oxygen consumption and formation of ROS. Some studies suggest that antioxidant supplementation has a beneficial effect, but it is unclear whether the effect lies in the direct enhancement of physical performance or in protection from injury.

*Neurological disorders.* Studies suggest that antioxidants may be helpful in slowing the progression of several disorders of the brain and nervous system.

## Further reading

Ames BN, Shigenaga MK, Hagen TM. Oxidants, antioxidants and the degenerative diseases of aging. *Proceedings of the National Academy of Sciences USA* 1993;90:7915–22

Block G, Patterson B, Subar A. Fruit, vegetables and cancer prevention: a review of the epidemiological evidence. *Nutrition and Cancer* 1992;18:1–29

British Nutrition Foundation. Antioxidant nutrients in health and disease. *Briefing Paper 25*. London: BNF, 1991

Bruckdorfer KR. Free radicals, lipid peroxidation and atherosclerosis. *Current Opinion in Lipidology* 1990;1:529–35

Diplock AT. Antioxidant nutrients and disease prevention. *Molecular Aspects of Medicine* 1994;15:293–376

Duthie GG, Wahle KW, James WPT. Oxidants, antioxidants and cardiovascular disease. *Nutrition Research Reviews* 1992;2:51–62

Halliwell B. How to characterize a biological antioxidant. *Free Radical Research Communications* 1990;9:1–32

Hill MJ. Diet and cancer: a review of scientific evidence. *European Journal of Cancer Prevention* 1995;4:2–42

Langseth L. Oxidants, antioxidants and disease prevention. ILSI Europe Concise Monograph Series. Brussels and Washington, DC ILSI Europe/ILSI Press, 1995

Riemersma RA, Wood DA, MacIntyre CCA, et al. Risk of angina pectoris and plasma concentrations of vitamins A, C and E and carotene. *Lancet* 1991;337:1–5

## 6. DIETARY FIBRE

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### Types of carbohydrates

Carbohydrates in foods that are digested and absorbed in the small intestine provide energy to body tissues mainly in the form of glucose and, to some extent, fructose and galactose. As a consequence of their chemical or physical structure, some carbohydrates are not digested and therefore pass into the large intestine, where they may serve, to varying degrees as substrates for the maintenance and growth of the intestinal microflora. Among the fermentation products of these carbohydrates are the short-chain fatty acids; those that are absorbed provide some energy to body tissues, notably to the mucosal cells of the large intestine.

The fundamentally different effects of digestible and undigestible carbohydrates on body functions were recognized more than 100 years ago and led to the differentiation of “available” and “unavailable” carbohydrates. For practical purposes, the unavailable carbohydrates are more or less equivalent to dietary fibre, which was first defined as “the remnants of plant cell walls resistant to digestion in the small intestine”. Cellulose, hemicelluloses and pectins are the main components of dietary fibre. Lignin, although not a carbohydrate, is also generally included as a fibre component.

The present interest in dietary fibre arose around 1970 when researchers proposed that this component of foods could be protective against many non infectious diseases, such as cardiovascular disease, diabetes and disorders of the gastrointestinal tract, including cancer of the colon. Much research has since been devoted to substantiating the initial epidemiological observations (“the dietary fibre hypothesis”) and to understanding mechanisms by which dietary fibre could be protective.

## *Satiating effect*

Foods rich in dietary fibre usually need more chewing than corresponding low-fibre foods. This, together with the water that is bound to fibre in the stomach, makes fibre-rich foods more satiating than low-fibre alternatives. Therefore, the tendency to ingest excess energy may be reduced.

## *Effects in the small intestine*

Inclusion in diets of purified viscous types of dietary fibre, such as pectin or guar gum, reduces blood glucose and insulin concentrations after a meal and also the concentration of low-density lipoprotein (LDL) cholesterol. There may be some health benefits, especially for people with diabetes or milder disorders of carbohydrate metabolism or for those with elevated LDL concentrations.

After a meal, the rate at which glucose is absorbed influences the secretion of insulin, which is needed to metabolize glucose. This in turn determines the change in blood glucose concentration following the meal. In diabetic patients, it is particularly important to regulate metabolism by achieving a low postprandial blood glucose concentration. There is much scientific evidence that foods containing slowly released carbohydrates (so-called low-glycaemic-index foods) may also be generally beneficial, not simply to diabetics, but this hypothesis needs further testing. The addition of viscous types of dietary fibre ("soluble fibre") to foods is one way of reducing their glycaemic index. However, factors such as particle size of cereal grains, intact cellular structure in legumes, the presence of a protein matrix in pasta, the type of starch and the way it has been heat treated, the fat content of the meal and the content of organic acids (such as lactic and propionic) may influence the overall glycaemic response at least as much as the natural dietary fibre content.

Foods with a very high content of soluble viscous fibre, notably oat bran, also help reduce LDL cholesterol concentration in the blood. However, the content and composition of the fat in a mixed diet may be as important in this respect. Indeed, diets selected from foods rich in fibre tend to be low in fat, making it difficult to single out which benefits are directly related to fibre. Substances such as plant sterols also seem to be partly responsible for the LDL-lowering effect of diets rich in vegetables. Thus, the apparent protective effects of dietary fibre against cardiovascular disease, as indicated in some epidemiological studies, may be related to a combination of factors in high-fibre diets.

The bioavailability of minerals from some foods rich in fibre, such as whole-grain cereal products and legumes, is low. The comparatively low absorption of, for example, nonhaem iron and zinc from such foods, is due to their phytic acid content rather than the dietary fibre.

In many instances, the higher mineral content of foods rich in fibre compensates for their lower absorption. If required, the phytic acid content of foods can be reduced by suitable processing, notably sour dough fermentation in bread baking. However, we rarely eat single foods in isolation. In a mixed diet many other factors enhance or inhibit mineral bioavailability. For example, vitamin C promotes iron absorption, whereas drinking tea with a meal provides tannins that reduce iron absorption. The effects of fibre must therefore be seen in the context of the whole diet.

## *Effects in the large intestine*

The most important physiological effects of dietary fibre probably occur in the large intestine. Faecal output is highly correlated with dietary fibre intake and inversely correlated with the time taken for materials to pass

through the alimentary tract (“transit time”). Thus, stools formed when the diet is rich in fibre are softer and more voluminous, and pass more rapidly through the gut, than when the diet contains little fibre. When the daily stool weight is 150 g or more, not only is constipation alleviated but the risk of colon cancer is reduced, supporting the original hypothesis.

Fibre that is relatively resistant to fermentation, for example, that found in whole-grain cereals, has the most pronounced faecal bulking effect. This is due to the ability of the fibre to bind water throughout the intestinal tract. Fibre components that are fermented are partly transformed into bacterial biomass, which also contributes to the faecal bulk.

The short-chain fatty acids formed during the fermentation of fibre (Figure 6.1) reduce the pH in the content of the large intestine. This seems to protect against cancer in several ways: by reducing the conversion of bile salts into co-carcinogens and decreasing their solubility by reducing the formation of ammonia and by providing specific nutrition to the cells lining the large intestine (Table 6.1). Some evidence also indicates that one of the short-chain fatty acids, propionic, may be responsible at least in part for the cholesterol-lowering effects of fibre.

Constituents of plant cell walls (non-starch polysaccharides) generally provide most of the carbohydrates fermented in the colon. Some foods, such as onions and Jerusalem artichokes, contain inulin and other fructooligosaccharides that are also undigestible and share some physiological properties with other fermentable fibres. Such carbohydrates are increasingly used as food ingredients. In starchy foods such as bread, cornflakes and legumes, some of the starch is undigestible (“resistant starch”).

In summary, the “unavailable” carbohydrate in foods is the total of non-starch polysaccharides, oligosaccharides and resistant starch, and all are important determinants of health in the large intestine.

### *Sources and intakes of dietary fibre*

Average diets in different European countries have similar dietary fibre contents of about 20 g/day, according to a recent survey. Fibre was determined by the AOAC (Association of Official Analytical Chemists) gravimetric method and represents fibre in food available for consumption. The sources of fibre differed in that 50% or more of the total fibre came from bread in the northern countries whereas fruit and vegetables were the predominant sources in the south. There are large differences among individuals and groups of people; for example, the diets of vegetarians contain three to five times as much dietary fibre as the average Western diet.

### *What are appropriate intakes?*

Several countries have made recommendations on dietary fibre intake usually in the range of 25–35 g/day, based on the scientific evidence summarized in this chapter. In the Nordic countries the recommendation is also expressed as >3g/MJ. Dietary fibre intakes could be increased by greater consumption of whole-grain bread and other cereal products, fruits and vegetables, and reduced consumption of low-fibre foods. Such dietary changes would also serve to decrease fat intakes and provide balanced intakes of many micronutrients.

## **Further reading**

Cummings JH, Frohlich W, (eds.) Dietary fibre intakes in Europe. COST 92. Brussels: Commission of the European Communities, Directorate-General Science, Research and Development, 1993

Gurr MI, Asp NG. Dietary fibre. ILSI Europe Concise Monograph Series. Brussels and Washington, DC: ILSI Europe/ILSI Press, 1994

Kritchevsky D, Bonfield C, (eds.) Dietary fiber in health and disease. St Paul, MN: Eagan Press, 1995

Schweizer TF, Edwards CA, (eds.) Dietary fibre—a component of food: nutritional function in health and disease. *ILSI Human Nutrition Reviews*. Heidelberg and London: Springer-Verlag, 1992

Joint FAO/WHO Expert Consultation on

Carbohydrates in Human Nutrition. Rome, Italy, 1998

## **7. FLUID INTAKE**

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### ***Importance of water in the body***

The consumption of fluids is a fundamental human need. A person might survive for more than 50 days without eating but can live only a few days without drinking. Water is the body's principal constituent amounting to about 60% of body weight (42 litres) in a 70kg man and slightly less in a woman because she has a greater proportion of body fat. The proportion of water varies throughout life, being about 75% in an infant's body and only 55% in the elderly.

All biological reactions take place within our body water. These require that dissolved molecules and electrolytes maintain a relatively constant concentration and osmotic pressure (see Glossary). Deviations from the normal electrolyte concentrations in body fluids are not well tolerated and need immediate correction by processes like drinking, urine production and sweating (see below). Electrolyte and fluid balance need to be considered together, since the dissolved electrolytes (the most important are sodium [Na<sup>+</sup>], potassium [K<sup>+</sup>] and chloride [Cl<sup>-</sup>]) are responsible for maintaining osmotic pressure and the electrical potential across cell membranes. The latter is important for many cell functions such as the conduction of nerve impulses.

### ***The main body water compartments***

There are two main water compartments in the body: the intracellular and extracellular compartments.

#### **Intracellular water**

Water within the cells accounts for about 50% of body water. Cells may either expand or shrink as they take up

or lose water. The intracellular fluid is isotonic mainly as a result of the concentration of  $K^+$  ions and is in osmotic equilibrium with the extracellular compartment, where the dominant ion is  $Na^+$ .

### **Extracellular water**

The extracellular compartment comprises the large and somewhat ill-defined spaces between cells and the arteries, veins and lymph vessels. It also includes the large volume of the intestines. The vascular and extravascular compartments communicate and balance their fluid contents. In contrast to the intracellular compartment, the extracellular compartment tolerates more deviations from normality in either direction, which gives it the role of a reservoir, yielding water to or recovering it from cells, thus maintaining the water content of the intracellular compartment at an appropriate level.

### ***Requirements for water and dietary sources***

In northern countries under ordinary conditions, consumption needs to be about 2 litres; per day but as much as 10 litres may be needed during strenuous exercise in hot climates. Water intake comes from the liquids we drink and to a lesser extent from the foods we eat. A small amount is produced by the metabolism of foods, particularly of fat (Table 7.1).

### ***Water losses and dehydration***

Water is lost from the body all the time. Regular water losses are associated with several vital functions. The air we breathe becomes saturated with water in the lungs before being expired. Digestion requires a large volume of fluid for the digestive enzymes to work in and to assist the transfer of substances through the gut into the bloodstream. Some of this fluid is lost in faeces. Much is ab-

sorbed and is important in helping the kidneys excrete waste products in urine. Such regular losses have to be regularly compensated for.

Variable or irregular fluid loss in addition to the regular losses is dictated mainly by environmental rather than internal conditions. The chief variable loss is sweating, which helps cool the body and regulate body temperature. Sweat is not simply water. It contains salt ( $Na^+Cl^-$ ) at a concentration of about half that of internal body fluids, but may be less than a third in people who are adapted to warm climates or who exercise regularly. Therefore, if excessive amounts of fluid have been lost (e.g. during strenuous exercise in high temperatures and high humidity), it is not sufficient simply to drink more water, but additional electrolytes are also needed. The amount of fluid intake needed and the appropriate concentration of electrolytes in a drink depend on individual circumstances.

Other conditions also require more fluid intake. For example, the kidneys need more water to eliminate the waste products from high protein intake, and the gut may need more water to digest certain types of foods. Children, for example, metabolize and need a lot of water, whereas the elderly tend to have a relatively lower body water content and different needs. Conditions such as vomiting and diarrhoea also put extra demands on water and electrolyte intake.

The term dehydration is generally applied to states of combined water and sodium depletion, whereas water depletion refers to pure water losses. Maintenance of body water and mineral balance is essential. Depletion of only a few percent of extracellular fluid is sufficient to cause at best some discomfort and at worst mental and bodily tiredness, followed by fever, vertigo, headache, vomiting, delirium and eventually coma. Although severe dehydra-

tion is seldom encountered, mild dehydration is common in everyday life, for example among labourers, soldiers, sportsmen and even tourists unfamiliar with fluid needs in warm climates.

The main causes of dehydration are outlined in Table 7.2. As can be seen, these may be due to age, illness, clinical interventions and environmental or self-imposed conditions.

### ***Maintenance of fluid balance***

Maintaining a constant water and mineral balance requires the coordination of sensitive detectors at different sites in the body linked by neural pathways with integrative centres in the brain that process this information. Instructions from the integrative centres to the “executive organs” (kidney, sweat glands and salivary glands) and to the part of the brain responsible for corrective actions such as drinking are conveyed by certain nerves and by several hormones and neuroactive substances.

Most of the components of fluid balance outlined in Table 7.1 are controlled by homeostatic mechanisms responding to the state of body water. These mechanisms are exquisitely sensitive and precise, and are activated with deficits or excesses of water amounting to only a few hundred millilitres.

A water deficit produces an increase in the ionic concentration of the extracellular compartment, which takes water from the intracellular compartment, causing cells to shrink. This shrinkage is detected by two types of brain sensors, one controlling drinking (see the next section) and the other controlling the excretion of urine by sending a message to the kidneys mainly via the antidiuretic hormone vasopressin to produce a smaller volume of more concentrated urine. When the body contains an excess of

water, the reverse processes occur: the lower ionic concentration of body fluids allows more water to reach the intracellular compartment. The cells imbibe, drinking is inhibited and the kidney excretes more water.

The kidney thus plays a key role in regulating fluid balance. In general, the kidneys function more efficiently in the presence of an abundant water supply. If the kidneys economize on water, producing a more concentrated urine, there is a greater cost in energy and more wear on their tissues. This is especially likely to occur when the kidneys are under stress, for example, when the diet contains excessive amounts of salt or toxic substances that need to be eliminated. This can eventually weaken the kidneys and increase the risk of hypertension later in life. Consequently, drinking plenty of water helps protect this vital organ.

### ***Drinking behaviour***

#### **Regulatory drinking**

Apart from urinary excretion, the other main fluid regulatory process is drinking, mediated through the sensation of thirst. There are two distinct mechanisms of physiological thirst: the intracellular and the extracellular mechanisms.

When water alone is lost, ionic concentration increases. As a result, the intracellular space yields some of its water to the extracellular compartment. Once again, the resulting shrinkage of cells is detected by brain receptors that send hormonal messages to induce drinking.

When water losses are accompanied by losses of ions (for example,  $\text{Na}^+\text{Cl}^-$  in sweating and diarrhoea), the intracellular compartment remains isotonic (retains a steady ionic concentration) although reduced in volume. Compensating for this isotonic state by drinking pure water causes over-dilution of the intracellular compartment. Thirst as-

sociated with receptors that govern extracellular volume is therefore accompanied by an enhancement of salt appetite. Thus, people who have been sweating copiously prefer drinks that are relatively rich in  $\text{Na}^+$  salts rather than pure water. It is always important to supplement drinks with additional salt when excessive sweating is experienced.

Sweat has only about half the ionic concentration of body fluids, so replacement of fluid lost as sweat by drinking the same volume of pure water will restore the water volume but at the same time will also cause the salt concentration of the extracellular compartment to be reduced. As a result, the cells swell up, causing a marked inhibition of the desire to drink. Further sweating will remain uncompensated because continued shrinkage of the extracellular compartment will be counteracted by signals to the brain that the intracellular compartment is overhydrated.

This problem may be experienced by some elderly people. It is not appropriate for them to consume too much extra salt because of the risk of hypertension and heart problems. Because of their low water reserves, it may be prudent for the elderly to learn to drink regularly even when not thirsty and to moderately increase their salt intake when they sweat. Better education on these principles may help prevent sudden hypotension and stroke or abnormal fatigue that can lead to a vicious circle and eventually hospitalization.

The brain's decision to start or stop drinking and to choose the appropriate drink is made long before the ingested fluid can reach the intra- and extra-cellular compartments. The taste buds in the mouth send messages to the brain about the nature, and especially the saltiness, of the ingested fluid, and neuronal responses are triggered as if

the incoming water had already reached the bloodstream. These are the so-called anticipatory reflexes: they cannot be entirely "cephalic reflexes" because they arise from the gut as well as the mouth.

### **Drinking that is independent of physiological need**

Although everyone experiences thirst from time to time, it plays little day-to-day role in the control of water intake in healthy people living in temperate climates. We generally consume fluids not to quench our thirst, but as components of everyday foods (e.g. soup, milk), as beverages used as mild stimulants (tea, coffee) and for pure pleasure. A common example is alcohol consumption which can increase individual pleasure and stimulate social interaction. Drinks are also consumed for their energy content, as in soft drinks and milk, and are used in warm weather for cooling and in cold weather for warming. Such drinking seems also to be mediated through the taste buds, which communicate with the brain in a kind of "reward system" the mechanisms of which are just beginning to be understood. This bias in the way human beings rehydrate themselves may be advantageous because it allows water losses to be replaced before thirst-producing dehydration takes place.

Unfortunately, this bias also carries some disadvantages. Drinking fluids other than water can contribute to an intake of caloric nutrients in excess of requirements, or in alcohol consumption that in some people may insidiously bring about dependence.

### ***Further Reading***

Fitzsimons JT. The physiology of thirst and sodium appetite. Monograph of the physiological society N° 35. Cambridge: Cambridge University Press, 1979

Nicolaidis S. Physiologie et pathologie de la soif. In: Encyclopedie médico-chirurgicale. Paris: 1987;1–20

Wrong O. Water and monovalent electrolytes. In: Garrow JS, James WPT, (eds.), Human nutrition and dietetics, 9th ed. Edinburgh: Churchill Livingstone, 1993;146–61

## **8. ALCOHOL CONSUMPTION AND HEALTH**

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### ***Alcohol in human society***

For millennia the consumption of alcoholic beverages has been part of many cultures in the world. Beers, wines, spirits and liqueurs contribute to the pleasure of eating and drinking and to social interaction. Most alcoholic beverages are produced by the fermentation of extracts of fruits and grains during which carbohydrates are transformed into alcohol. This simple natural organic compound is a source of metabolic energy and has physiological and psychotropic effects depending on the level of intake. Most people who consume alcoholic beverages do so responsibly and in moderation. Excessive drinking of alcoholic products, however has been recognized throughout the ages as presenting serious health and social risks, and the problems of misuse have led to controversy about the appropriate place of alcoholic beverages in society.

This brief review focuses on the effects on human health of moderate alcohol consumption. The main sources of scientific literature regarding possible associations of alcoholic beverage consumption and health are epidemiological studies, but some clinical and animal studies have investigated particular causal relationships.

### ***Alcohol intake***

There is no agreed definition of “moderate” alcohol intake, although the health authorities in some countries provide guidelines for their populations (see Further reading). The results of many epidemiological studies suggest that moderate consumption corresponds to a daily intake of 15–40 g alcohol for an adult man and 10–30 g for a woman. However, the health and behavioural conse-

quences of consuming alcoholic beverages vary considerably among individuals, depending on age, sex, body weight, overall health condition, eating habits and drinking pattern.

One barrier to understanding is that the accurate measurement of alcohol intake in epidemiological studies is particularly difficult. In studies relying on self-reporting by the subjects, underestimates of up to 40% are not uncommon, and improved methods of data collection are needed. Underestimates of alcohol consumption will tend to lead to conservative estimates of amounts that can be safely consumed, whereas overestimation will result in safe levels that are lower than has been assumed.

In this concise monograph, moderate consumption generally means a range of alcohol intake between 10 and 40 g/day. *Light* drinking is consumption of less than 10 g/day. *High* consumption is intakes in excess of 40 g/day on individual occasions, and heavy or excessive consumption is regular intakes in excess of 40 g/day for extended periods.

### ***Inherited factors***

Genetic studies indicate that individuals differ markedly with respect to how they are affected by alcohol. Alcoholism is recognized as a disease that may run in families. A person's genetic background may interact with environmental factors to influence how he or she will respond to the consumption of different quantities of alcohol and the extent to which consumption is under control. Today's understanding of the multiple genetic factors underlying the response to alcohol is incomplete. Several "biological markers" that may indicate susceptibility to an alcohol-related disease have been identified, and it may soon be possible to use them to predict an individual's risk of acquiring the disease as mediated by alcohol.

### ***Alcohol metabolism***

Alcohol is absorbed via the stomach and small intestine and diffuses rapidly throughout the body fluids (Figure 8.1). It is metabolised mainly by the liver at the rate of 5–7 g alcohol per hour. Thus, if the consumption of, say, four drinks (with one drink containing about 10 g alcohol) is spread over the day, the blood alcohol concentration at any one time will be very low.

The main route for alcohol metabolism in the body is by oxidation to acetaldehyde and then to acetate by means of the enzymes alcohol and acetaldehyde dehydrogenase, respectively, which are active mainly in the liver. At high levels of intake, an alternative route through the so-called microsomal ethanol-oxidizing system may be induced. The acetate that results from either of these pathways will be further degraded to carbon dioxide or used as acetyl-CoA building blocks in the biosynthesis of lipids.

### ***The energy value of alcohol***

One important aspect of the metabolic fate of alcohol concerns its energy value and whether alcoholic beverage consumption increases the risk of overweight or obesity. One gram of alcohol supplies about 29 kJ (7 kcal) of energy. In several studies, people who consumed moderate amounts of alcohol tended to have total energy intakes that were higher than those who did not drink; they appeared to add the energy from alcohol to their normal energy intake rather than replace food with alcohol. Yet in the same population groups, relative body fatness decreased with increasing alcohol intake.

Thus, despite the commonly held belief that alcohol consumption automatically results in weight gain, it has been surprisingly difficult to demonstrate such an association in epidemiological surveys. There have been too many confounding factors in the studies published so far (in-

cluding the fact that alcoholic beverages contain sources of energy other than alcohol) to enable clear conclusions to be drawn. What happens to the energy supplied by alcohol remains a metabolic mystery. Carefully designed metabolic studies are required to provide a more satisfactory explanation of a relationship that is far from clear.

### *The digestive system*

Epidemiological evidence suggests that there is an association between alcohol consumption and the occurrence of cancers of the mouth, oesophagus and larynx and that the risk increases in a dose-related manner. Cigarette smoking in combination with alcohol intake significantly increases the risk. In the absence of smoking, the risk of these forms of cancer from alcohol consumption is small, except in very heavy drinkers or where nutrition is inadequate.

There is no evidence that alcohol consumption is associated with cancers of the stomach or pancreas. Some studies have indicated a possible association with cancers of the colon and rectum, but the risk is considered very small and in many cases may be due to other factors in the large intestine.

Alcohol itself is not a carcinogen, but in animal experiments it may enhance the carcinogenic process without itself having an initiating capability. It thus acts as a co-carcinogen.

### *Alcohol and the liver*

The liver is the principal site of alcohol metabolism in the body. It is well able to metabolize moderate amounts of alcohol over a long period of time but can be adversely affected by heavy drinking. The occurrence of toxic intermediates such as acetaldehyde and peroxides may be

possible causes of cellular damage in the liver during excessive consumption.

Liver diseases develop in stages. Fatty liver and fibrosis may occur without symptoms in the early stages. In alcoholic hepatitis, local cellular death occurs and malfunctions become apparent. Liver cirrhosis is the more advanced stage of cellular degeneration and fibrotic scarring for which chronic excessive alcohol consumption is one of a number of causative factors. Habitual heavy consumption is often accompanied by a state of malnutrition which further contributes to cirrhosis. It is uncertain whether there is any direct association between alcohol and cancer of the liver, but liver cirrhosis certainly carries a greater risk of cancerous change.

### *The circulatory system*

Alcohol affects a variety of functions of the blood and blood vessels that have a bearing on diseases such as hypertension, stroke and coronary heart disease (CHD).

There is evidence that the chronic intake of more than 30 g alcohol per day may result in hypertension and that certain individuals are particularly susceptible. About 10% of hypertension cases in North America and Europe are believed to be due to alcohol intake levels exceeding 40 g/day. Such evidence is confirmed by intervention studies in which the effects of reduced alcohol consumption are studied.

Prospective epidemiological studies provide no evidence for a strong association between alcohol intake and total incidence of stroke. There are some indications that moderate drinking may reduce the risk of ischaemic stroke whereas heavy drinking may increase the risk of haemorrhagic stroke.

Notwithstanding the adverse effects on blood pressure in some people, many scientific studies have associated moderate alcohol intake with a significant reduction in the overall risk of coronary heart disease (Figure 8.2). This applies to both sexes at all ages but is most evident in men over 50, where CHD risk is highest. The reason for this is not known but several possibilities are under investigation. Clinical studies show that alcohol increases the level of high-density lipoproteins in the blood. These lipoproteins inhibit or reverse the atherogenic process. Laboratory experiments provide evidence that alcohol may reduce platelet aggregation and decrease the risk of arterial blockage (thrombosis). Other substances present in alcoholic beverages, such as antioxidants in red wine, may have additional benefits. A detailed analysis of epidemiological data has provided no conclusive evidence for differences in the beneficial effects observed among the various types of alcoholic beverages.

## *The reproductive system*

### **Pregnancy**

Foetal abnormalities known as “foetal alcohol syndrome” have been observed in babies born of alcohol-dependent women. High blood alcohol levels may damage the development of the central nervous system in the foetus, but numerous confounding factors such as poor diet and illicit drug use often found in this condition question the existence of a simple relationship between alcohol and foetal alcohol syndrome. There is little evidence of an increased risk of foetal abnormalities associated with light or even moderate drinking, but a safe limit has not been determined.

### **Breast cancer**

Numerous epidemiological studies have revealed a weak and inconsistent association between moderate alcohol

intake and the risk of breast cancer. The association might be more consistent in women consuming in excess of 40 g alcohol per day. Definitive conclusions about the role of alcohol in this disease cannot be drawn because of the existence of other nutritional and hormonal factors in most studies.

## *Conclusions*

There can be little doubt that light to moderate consumption of alcoholic beverages (not exceeding 40 g alcohol/day) is associated with benefit to health and social interactions. It is also clear that excessive consumption, either acutely or over a long period of time, increases morbidity from a variety of causes and can result in accidents. Because the vast majority of drinkers do not proceed to alcohol abuse or dependency, when the transition to abuse or dependency occurs, it may be the result of genetic predisposition reinforced by environmental factors. Community health programmes might reduce the risk of such adverse trends by educational activities in early adulthood.

### **Further reading**

Colditz GA. The Nurses' Health Study: findings during 10 years of follow-up of a cohort of US women. *Current Problems in Obstetrics, Gynecology and Fertility* 1990;13:135–74

Gurr MI. Health Issues related to alcohol consumption. ILSI Europe Concise Monograph Series. Brussels and Washington, DC: ILSI Europe/ILSI Press, 1996

Jackson R, Scragg R, Beaglehole R. Alcohol consumption and risk of coronary heart disease. *British Medical Journal* 1991;303:211–6

James WPT Alcohol: its metabolism and effects. In: Garrow JS, James WPT, eds. *Human nutrition and dietetics*, 9th ed. Edinburgh: Churchill Livingstone, 1993;103–18

Klatsky AL. Alcohol and hypertension. *Clinica Chimica Acta* 1996;246:91–105

Verschuren PM, (ed). Health issues related to alcohol consumption. Brussels and Washington, DC: ILSI Europe/ILSI Press, 1993

## **9. PHYSICAL ACTIVITY**

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### **Overall health implications**

Physical inactivity confers a substantial public health burden. On the basis of several studies published in the United States, it has been estimated that between 9 and 16% of deaths can be attributed to a sedentary lifestyle. No comparable published data are available for Europeans, but there is no reason to expect that the European situation differs substantially from that in the United States. If the proportion of men classified as sedentary in the United States could be decreased to 25% from the current estimate of about 37% conservatively estimated, death rates could be reduced by between 3 and 6%. This represents an appreciable number of people.

It follows that people who are physically active tend to be healthier than their sedentary peers: they experience fewer chronic degenerative diseases, especially coronary heart disease (CHD), hypertension, stroke, osteoporosis and possibly non-insulin-dependent (adult-onset) diabetes. There are also indications that exercise is associated with improved immune function and decreased risk of colon cancer. Regular exercise may also bring about an increased sense of well-being.

### **Effects on metabolism**

Physically active people are characterized by an elevated concentration of high-density lipoprotein (HDL) cholesterol in the blood and an enhanced capacity to metabolize triglycerides and their constituent fatty acids, whether they originated from the diet or were produced in the liver. Such individuals are also particularly effective in metabolizing glucose and controlling its concentration in the blood in response to the action of the hormone insulin.

The significance of HDL cholesterol is that its concentration in the blood is inversely associated with CHD risk. A ratio of total cholesterol to HDL cholesterol greater than 5 has been regarded as unfavourable in this regard. Differences in blood HDL concentrations between physically active and sedentary people are typically about 20–30% and are independent of age, body mass index, alcohol consumption and cigarette smoking. Aerobic exercise that involves a minimum expenditure of about 1200 kcal/week over a period of several months increases HDL concentration, especially when the exercise programme results in weight loss.

Walking may be as effective as more strenuous (and for many people less acceptable) forms of exercise. In one study of more than 3,500 men and women, those who habitually walked for 2.5–4 hours/week (say 13–19 km) were twice as likely to have a favourable total-to-HDL cholesterol ratio as those in a comparable group who exercised little.

Glucose tolerance—the body’s ability to regulate the concentration of glucose in the blood in response to the secretion of insulin—is improved for up to 48 hours after exercising, because the body’s tissues become more responsive to the effects of insulin. Skeletal muscle and adipose tissue are the body’s principal insulin-responsive tissues and exercising the whole body may affect some 30 kg of muscle tissue. Exercising regularly and over a long period results in increases in muscle mass and decreases in adipose tissue mass.

### ***Non-insulin-dependent diabetes***

Poor glucose tolerance, excessive adipose tissue and inactive muscles are associated with increased risk of developing non-insulin-dependent diabetes. Exercise may therefore be expected to reduce the risk of this form of

diabetes. Indeed, long-term studies show that the incidence of non-insulin-dependent diabetes decreases by some 6% for every 500 kcal expended per week in physical activity; with an indication that the influence of activity level is strongest in those who are overweight.

### ***Bone health***

Mechanical loading stimulates new bone formation and inhibits bone loss. As might be expected, bone mass in some skeletal sites is markedly higher in athletes than in sedentary people. The effect is clearly a local response, with changes occurring only at the skeletal sites loaded in exercise and is most effective with high loading, for example, in strength training.

Regular physical activity during childhood and adolescence can increase peak bone mass substantially and if continued into adulthood can confer a significant advantage in later life, when bone loss begins. Exercise that is not weight bearing, such as swimming, does not influence bone mass.

Exercise in pre-menopausal women increases bone strength. In the short term, the effects are small and probably do not influence fracture risk, but if continued over years, the accumulated small improvements may have a clinically important effect.

In the first five or so post-menopausal years, when the loss of bone is greatest owing to oestrogen deficiency, physical activity may have little impact on bone loss. After this, exercise may be able to slow the rate of bone loss. Whereas it is to be expected that activities that exert a high strain on bones should be most effective, they would tend to increase the risk of further fracture in women with established osteoporosis, typically the elderly who may also have problems with balance. Overall, the most appropri-

ate exercise prescription for this group may be regular brisk walking.

Extreme levels of exercise may have potentially detrimental effects in other age groups. Low bone mineral densities have been reported in young women athletes whose menstrual cycles and reproductive function are disturbed by high levels of training. Decreasing the intensity of training reverses bone loss but the long-term effects of repeated spells of amenorrhoea induced by exercise are unknown.

### ***Weight regulation***

Any physical activity whatever its intensity, contributes to energy expenditure. Moreover, physical activity is the only way in which energy expenditure can be increased voluntarily. Moderate activity, for example, walking 3 km three times a week, is not associated with changes in body fatness over 3–6 months, but at higher levels there tends to be a consistent loss of body fat. In general, studies on the effect of exercise on fat loss in which spontaneous increases in intake may occur owing to the stimulation of appetite, report an average loss of about 0.12 kg/week for men and a little less for women, with total exercise energy expenditure being the most important determinant of weight loss. Less is known about the long-term implications of inactivity for weight regulation, but one recent study in the United States found that men and women with low levels of recreational physical activity at both initial and followup examination were more than three times as likely to experience major weight gain over a 10 year period as were people with high levels of such activity.

One contributory factor in the relationship between physical activity and avoidance of weight gain may be the body's ability to match food intake to energy expenditure more precisely at high levels of energy turnover (i.e. when intake and expenditure are both high).

### ***Hypertension***

Exercise may play a role in both the prevention and treatment of hypertension. An analysis of 48 studies found that increased physical activity level resulted in falls in systolic and diastolic blood pressure of 3 mm Hg in people with blood pressure in the normal range. The corresponding falls were (systolic/diastolic) 6/7 mm Hg in borderline hypertensives and 10/8 mm Hg in hypertensive subjects. These results certainly suggest that endurance exercise is an effective way of reducing hypertension that does not involve drugs and that even low-intensity exercise, such as walking, may cause a worthwhile improvement in people with high blood pressure.

### ***Coronary heart disease***

Physical inactivity has been estimated to double the risk of a heart attack. This is the same order of increased risk as is associated with high blood pressure, smoking more than 20 cigarettes a day or having a high blood total cholesterol concentration. The effect is independent of other major risk factors and is graded. Only current activity confers protection: regular exercising is probably as important in risk reduction as stopping smoking.

Regular physical activity in occupational work or in leisure time decreases risk. In one landmark study of Harvard graduates, the risk of a first heart attack was one-quarter to one-third lower in men who expended more than 2,000 kcal/week in leisure time physical activity (sports, gardening, walking, climbing stairs, etc.) than in men with lower exercise energy expenditure.

Some, but not all, studies suggest that a threshold level of exercise may be needed for a cardioprotective benefit. In a study of British civil servants, only men reporting vigorous exercise (sports and games, fast walking at more than 6 km/h, frequent cycling) showed heart attack rates

about half those experienced by sedentary men (Figure 9.1). There was no such effect for non vigorous exercise even at high cumulative total activity levels.

Some studies have measured the outcome of regular exercise, or fitness. The Cooper Institute for Aerobics Research studied more than 10,000 men and 3,000 women over 8 years. All-cause mortality—predominantly from heart disease—was greater in men and women with lower fitness levels. The most marked difference in risk was evident between the least active men and the second lowest group (Figure 9.2), suggesting that the greatest public health benefit may arise when the most sedentary individuals become somewhat fitter.

The evidence that physical activity protects against CHD is convincing, but there is not yet consensus regarding the amount and intensity of activity needed to be effective, as the foregoing paragraphs illustrate. Although several key studies have shown substantial reductions in risk with large amounts of moderate-intensity activities, other findings suggest that more vigorous activity may provide unique benefits.

Mechanisms by which regular exercise might confer lower risk of CHD include effects on blood pressure, weight regulation, lipoprotein metabolism and insulin sensitivity or even an effect on the acute phase of the disease—thrombosis, for example.

### ***Functional capacities in the elderly***

In the elderly, and in some younger patients, functional capacities (including strength, stamina and mobility) decline to the point where they fall below thresholds that are important for the quality of life. As at younger ages, aerobic training increases functional capacities in the elderly. For the elderly, as for the majority of middle-aged

sedentary individuals, brisk walking is a suitable and effective form of exercise if it can be done regularly. Regular physical activity can decrease the rate of decline in functional capacity with age. For example, in some studies, older men who maintained their activity levels experienced a rate of decrease about half that found in sedentary men.

In the very old, muscular strength is even more important than endurance. Leg muscle strength in particular influences independence; if it is too low, it becomes difficult to lift body weight against gravity, for example, when rising from a low chair or toilet seat. Muscle mass will increase, particularly with training designed to increase strength, even in the very old (Figure 9.3).

Another benefit of exercise for the elderly is that it stimulates appetite: the consumption of more food leads to higher (micro) nutrient intakes, which may be crucially important for some elderly people.

### ***Exercise in the management of patients with existing disease***

Physical activity contributes to the management of patients with existing disease (e.g. heart disease, chronic lung disease, asthma, adult-onset diabetes, osteoporosis) because it maximizes residual functional capacities and increases independence. Evidence of benefit is most extensive for myocardial infarction; an overview of randomized trials of rehabilitation that included an exercise component (many also included other health advice, for example, on smoking and diet) suggested a reduction in mortality of some 20% after 3 years.

Aerobic exercise, which includes brisk walking, jogging, swimming, dancing and cycling, requires additional effort by the heart and lungs to meet the increased demand

of the muscles for oxygen. Low to moderate aerobic exercise involves the muscles working at up to about half their maximum oxygen uptake ( $<50\% \text{VO}_{2\text{max}}$ ). For patients, this level of exercise is effective and can be prescribed with safety. The activities described above have the advantage of involving most of the body's large muscles, thereby maximizing the benefits. Regular aerobic exercise of this kind that is sufficient to expend a substantial amount of energy, preferably on a daily basis, is also important in weight regulation, which can be a major problem for many patients.

Exercise that is too strenuous, however, is not without its risks as discussed above and in a later section. For exercise to be beneficial for health, it does not have to be at a level traditionally regarded as "training". Benefits for blood pressure and metabolism are evident after single exercise sessions. Cumulatively, these small immediate effects make an important contribution to decreasing the risk of, for example, cardiovascular diseases, demonstrating the importance of the frequency and regularity of exercise. Routine physical activities which are habit forming are therefore particularly to be recommended to help patients increase their physical activity levels and then maintain, within the limits of their condition, a generally more active life.

### ***Exercise and feelings of well-being***

Anecdotally, exercise has long been associated with "feeling good". Recent objective studies have demonstrated beneficial effects on measures of anxiety and reactions to stress as well as on self-esteem. Exercise has an antidepressant effect in healthy people as well as in patients diagnosed as clinically depressed.

### ***Risks associated with exercise***

Although mortality is significantly greater in men and women who are unfit and who lead sedentary lives and regular physical activity improves health in many respects, exercise also carries risks. It can result in injuries (see the earlier section on Bone health), trigger heart attacks and at high levels may decrease resistance to infection. These risks appear to depend mostly on the intensity of exercise: longer-duration or more frequent bouts of moderate intensity exercise may result in sufficient levels of fitness to confer worthwhile health gains while avoiding much of the risk. People who engage in exercise of lower-intensity for longer duration or in frequent sessions, rather than in higher-intensity shorter sessions, probably decrease these risks and will be likely to find the exercise more acceptable. It is desirable to follow a varied activity programme to avoid injury from over use and to encourage the training of different muscles to maximize metabolic advantages. Varying the type of activity may also increase motivation.

Strenuous exercise also has implications for drinking and the maintenance of water balance (see Chapter 7).

## Further reading

Arroll B, Beaglehole R. Does physical activity lower blood pressure? A critical review of the clinical trials. *Journal of Clinical Epidemiology* 1992;45:439–47

Berlin JA, Colditz GA. A meta-analysis of physical activity in the prevention of coronary heart disease. *American Journal of Epidemiology* 1990;132:612–28

Blair SN. Physical activity physical fitness and health. *Research Quarterly for Exercise and Sport* 1993;64:365–76

Blair SN, Hardmann AE, (eds.). Physical activity, health and well-being: Papers from an international scientific consensus conference, Quebec, May 1995. *Research Quarterly for Exercise and Sport* 1995;66 (no 4).

Bouchard C, Shephard RJ, Stephens T, (eds.) *Physical activity, fitness and health: International proceedings and consensus statement*. Champaign, IL: Human Kinetics, 1994

Brooke-Wavell K, Jones PRM, Hardmann AE. Brisk walking reduces calcaneal bone loss in post-menopausal women. *Clinical Science* 1997;92:75–80

Haskell WL. Health consequences of physical activity: understanding the challenges regarding dose-response. *Medicine and Science in Sports and Exercise* 1994;26:649–60

O'Connor GT, Buring JE, Yusuf S, et al. An overview of randomized trials of rehabilitation with exercise after myocardial infarction. *Circulation* 1989;80:234–44

Williamson DF, Madans J, Anda RF, et al. Recreational physical activity and ten-year weight change in a US national cohort. *International Journal of Obesity* 1993;17:279–86

## 10. ORAL HEALTH

Good teeth and a healthy mouth are maintained by daily attention to oral hygiene, by the frequent presence in the mouth of fluoride, by a balanced, nutritious diet and by regular professional dental care.

### Dental caries

Dental caries (tooth decay) is started by dissolution (“demineralisation”) of the tooth enamel by acid. The acid is produced by bacteria that ferment carbohydrates present in the diet. These dietary carbohydrates include sugars, both refined and in fruits and other plant foods in their natural form, as well as cooked starches. Bacteria are always present in the mouth, normally in thin layers. However, if the teeth are not cleaned regularly, they may build up in a thick and sticky layer of plaque on the tooth surface, and a large plaque volume gives rise to potentially harmful quantities of acid.

Decay will occur only if acid challenges following the intake of carbohydrate take place too frequently. Even so, decay is not inevitable. After an acid challenge, saliva gradually neutralises the acid, and by virtue of its calcium and phosphate content, saliva returns mineral to the tooth enamel to start the repair process (“remineralisation”). Carious holes in teeth occur only if the loss of mineral after acid challenges is not sufficiently repaired by remineralisation. This repair is greatly enhanced by fluoride. The many interacting factors that affect caries development are shown in Figure 10.1.

A number of factors about the tooth itself affects the likelihood of its becoming decayed. Primary (baby) teeth and newly erupted secondary (adult) teeth are more susceptible because the enamel is less dense. Some tooth sur-

faces are more likely to decay than others. For example, deep fissures on the biting surfaces of the back teeth can make it difficult to remove the plaque there. However, under all circumstances, the resistance and survival of teeth are greatly favoured by fluoride because it reduces demineralisation (decay) and enhances remineralisation (repair).

Because the progress of decay depends on the balance between demineralisation and remineralisation, carious holes form under two preconditions: when a person fails to clean the teeth (and thus to remove plaque) with a fluoride toothpaste and when carbohydrate is consumed too frequently so that demineralisation will predominate. In countries where fluoride toothpaste is used, the number of children and adults with decay has declined dramatically over the last 20 years and is now at a very low level even though the intake of sugars has remained the same. For someone with good oral hygiene (brushing twice a day with fluoride toothpaste), an intake of carbohydrate food or drinks six or seven times a day is consistent with a very low risk of decay.

### ***Periodontal disease***

The supporting structures for the teeth—the jaw bones and the soft tissues of the gums—are every bit as important as the teeth. Plaque tends to build up at the margin of the gum and the tooth. If it is allowed to accumulate, the gum tissue becomes inflamed, and this can lead to serious damage to the gums, which recede down the teeth. Eventually even the supporting bone can be damaged by infection, resulting in loosening of the teeth until, finally, they fall out.

### ***Tooth wear***

Tooth tissue can be damaged by factors not related to decay. The most important cause of loss of tooth substance

is erosion. Food acids, for example from lemons, oranges or apples, their juices or other acidic beverages, can lead to dissolution of the surface layers of enamel. Acid may also enter the mouth by the reflux of stomach contents, by vomiting, through eating disorders and, rarely, from airborne industrial sources (e.g. in battery production facilities). As with the caries process, the saliva forms the main line of protection in the mouth. However, if the acid challenge is severe or prolonged, the saliva cannot provide adequate protection. Individuals with reflux disease, vomiting or eating disorders clearly need medical intervention. In most cases excessive intake or unusual use of acidic foods and beverages leads to erosive enamel loss. However, erosion rarely occurs alone but in combination with too forceful toothbrushing after softening by acid or with attrition resulting from chewing forces.

### ***Sucrose and the prevalence of caries***

For a long time, it was stressed that refined sugar consumption was the single most important factor in promoting the high prevalence of tooth decay in developed countries and that avoidance of sugar was the most effective way of preventing caries. This association was certainly a strong one before the widespread use of fluoride and other improvements in oral hygiene. A reviewer who analysed data from 47 nations up to the 1970s concluded that half the variability in dental caries prevalence could be explained by sucrose availability.

This situation has changed conspicuously during the last 20 years, especially in countries where fluoride toothpaste and/or fluoridated water are widely used and children have been taught good toothbrushing habits. Recent studies have highlighted the fact that caries prevalence correlates well with sucrose consumption in communities where oral hygiene is poor and where fluoride is absent, but not elsewhere. Sucrose has been particularly targeted because the stickiness of some products that contain sucrose can

result in prolonged adherence to tooth surfaces. Nevertheless, starches which lodge in niches of the teeth can be readily hydrolysed and fermented in the mouth and can have caries-promoting properties equal to those of sucrose in certain circumstances. Again, the role of oral hygiene in dislodging sticky or otherwise firmly adhering food residues cannot be stressed too strongly.

### ***Preventive strategies for oral health***

Effective oral hygiene can help protect the oral structures. Decay of the teeth as well as inflammation of the gums can be triggered by the presence of bacteria. Without bacterial plaque, carbohydrate cannot be turned into demineralising acid and the gums also remain healthy. Regular toothbrushing helps achieve freedom from plaque and maintain oral health.

Fluoride significantly reduces caries risk. Fluoride has revolutionised dental health, and experts think that its wide use in toothpastes in highly developed countries is the main factor responsible for the decline of dental decay in the last 20 years. Fluoride increases the resistance of enamel to decay and enhances remineralisation of enamel following an acid challenge. Fluoride can be supplied to the tooth in two ways. When fluoride is ingested, for example, with drinking water, it enters the body and, during tooth development in childhood, is incorporated into the tooth enamel. This is called systemic fluoride. Other ways of providing systemic fluoride are in salt, milk or other food products.

In Europe, methods of systemic fluoridation have met continuous opposition which has jeopardized implementation in spite of strong WHO recommendations. Fortunately, epidemiological evidence has shown that topical fluoride, applied directly to the tooth surface after eruption, for example as fluoride toothpaste or as fluoride gel applied by the dentist, is at least as effective as systemic

fluoride administration. This is demonstrated by the caries decline in The Netherlands (Figure 10.2), where water fluoridation was stopped in 1973; this event stimulated personal oral hygiene and the use of fluoride toothpastes which were found to be highly effective although sugar consumption is still high.

Both systemic and topical fluoride are effective, but topical fluoride use combined with added oral hygiene benefit of regular toothbrushing makes the use of fluoride toothpaste the most popular and effective means of prevention. Infants' first teeth should be cleaned with a tiny amount of fluoride toothpaste, and children up to the age of about 6 years will need help with toothbrushing. The general level of personal hygiene is higher today than ever before so toothbrushing has become universally popular and accepted except in certain ethnic groups who prefer more traditional methods. Unfortunately, most traditional remedies do not involve fluoride.

Although the benefits of fluoride are established beyond doubt, some authorities caution against too much fluoride. In some parts of the world, high concentrations of fluoride resulting from a highly fluoridated water supply combined with the ingestion of fluoride, such as found in Tanzania and Kenya where a natural mineral of volcanic origin is used as a food tenderizer, can cause severe "fluorosis"—a mottling of the tooth enamel which weakens the tooth structure. Mild fluorosis affects the appearance of teeth but not their structure. Fluorosis is rare in Europe.

Attention to diet is also important. Oral health, just like overall health, is affected by diet. A balanced diet rich in starches, fruits and vegetables and moderate in fat is widely recommended. To reduce the chance of both caries and erosion, it may be prudent for individuals at risk to limit the intake of carbohydrate or acidic foods and beverages to six or seven portions per day. Constant nib-

bling and sipping should be avoided. The beneficial influence of eating a small amount of cheese at the end of a meal is now well established. Several mechanisms have been suggested for this effect; the most likely explanation is the calcium content of milk aiding remineralization. Chewing gum after meals also helps neutralize acid in plaque by stimulating the production of saliva.

Infants should never be put down to sleep with a bottle containing anything but plain water. Even milk can cause caries during sleep when salivary flow is low. Bedtime use of carbohydrate-rich foods and beverages should also be avoided.

Another very important facet of good oral health is regular visits to the dentist. Infants should be taken to the dentist at the time the teeth first appear. The dentist can not only identify early signs of decay and periodontal disease but also detect other problems such as oral infections or even oral cancer.

### Further reading

Fédération Dentaire Internationale Working Group on Nutrition. Diet and oral health. *International Dental Journal* 1994;44:599–612

König KG. Changes in the prevalence of dental caries: how much can be attributed to changes in diet? *Caries Research* 1990;24(suppl 1):16–8

Navia LJ. Nutritional role of sugars in oral health. *American Journal of Clinical Nutrition* 1995; 62(suppl):275S–83S

Pollard MA, Duggal MS, Fayle SA, et al. Caries preventive strategies. ILSI Europe Concise Monograph Series. Brussels and Washington, DC: ILSI Europe/ILSI Press, 1995

Sieber R, Graf H. Hemmt Käse die Zahnkaries? *Ernährung* 1990;14:63–70

## 11. SUMMARY AND CONCLUSIONS

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Many factors influence the adoption and maintenance of healthy lifestyles. The reader should be aware that only a selection of these factors have been addressed in this booklet. Allergies, osteoporosis, the long-term implications of experiences in early life (e.g. infant feeding practices), the health effects of smoking and many other issues, have not been addressed here. In particular, abundant evidence indicates that social and economic inequalities in society play a large part in health status. Thus, in many industrialized countries there is a steep gradient of mortality and life expectancy from the professional classes to the unskilled classes, and the gap appears to be widening. Although these matters are beyond the scope of this booklet, they should not be forgotten or ignored.

Many nutritional deficiencies have been successfully eliminated in Europe during this century, and life expectancy at birth has doubled. Chapter 2 concluded that problems of overnutrition now play a greater role in poor health than nutritional deficiencies, although clearly identified deficiencies (e.g. iron, iodine, folic acid, calcium) in specific population groups still exist and need to be addressed.

It is appropriate that the topic of energy balance and obesity was introduced early in this booklet (Chapter 3), since it is now becoming an important issue in many countries. Overconsumption of fat relative to carbohydrate was identified as a major contributory factor in the high (and increasing) prevalence of obesity, and the importance of increased physical activity as part of preventive measures was stressed. Fat reduction from current intakes of 35–45% to about 30% is the cornerstone of many recommendations, which implies an increased intake of carbohydrates not only as cereals but as fruits and vegetables as well (Chapter 6).

Coronary heart disease (CHD) remains the major cause of death in many European countries, even though death rates from CHD are falling steadily in many countries including those in Western Europe. The fact that this booklet has chosen to focus on the association between dietary fat and CHD (Chapter 4) should not obscure the fact that many other dietary factors (including fibre, several minerals and vitamins and several non-nutrient substances) also influence the development of this disease as well as many non-dietary environmental factors such as smoking and exercise. At the present time the known risk factors appear to account for only about 50% of total risk.

Chapter 4 concluded that dietary fat can modify the risk of CHD through effects on the concentrations of LDL and HDL cholesterol in the blood and possibly also through other mechanisms. High intakes of saturated and *trans* unsaturated fatty acids can increase risk, and replacement of these fats by oleic, linoleic and *n*-3 polyunsaturates from fish oils reduce it. Among these fatty acids, linoleic acid is the most effective in lowering blood LDL cholesterol. Public health measures to lower CHD risk should aim to encourage reduction of intakes of the cholesterol-raising saturated fatty acids and perhaps *trans* fatty acids as well. In practical terms, this could be achieved by replacing most hard fats in the diet by unsaturated oils or carbohydrate-rich foods.

Although the processes of oxidation and the body's defence against it (Chapter 5) have been studied for a long time, only comparatively recently has so much emphasis been placed on their role in the development of chronic diseases such as CHD and cancer. Although the scientific basis for the role of dietary antioxidants in the prevention of disease has yet to be fully established, the soundest advice at present is to ensure a plentiful intake of fruit and vegetables. These not only supply a wealth of antioxidant nutrients and non-nutrient substances but also provide a wide range of micronutrients and dietary fibre.

The energy density of fruit and vegetables is also lower than that of most other food items.

In Chapter 6 it was concluded that dietary fibre has important effects on satiety. Owing to its bulk, fibre helps limit the amount of fat energy that can physically be consumed. Fibre also influences the regulation of glucose and fat metabolism. In its intact form, fibre's physical properties influence processes of digestion, absorption and rate of passage through the small intestine, whereas in the large intestine degradation of some forms of fibre by endogenous microorganisms generates substances such as volatile fatty acids that also influence metabolism in important respects. Each of these effects may be relevant to a role for fibre in protecting against a variety of chronic diseases. Highest intakes of fibre are achieved by individuals who consume abundant fibre-rich foods such as fruit and vegetables and whole-meal products.

Chapter 7, on fluids provides a timely reminder that drinking is at least as important as eating and that water is a vital nutrient. Normal consumption is about 2 litres per day for the average adult, and at least half this needs to come from beverages.

In industrialized societies, even though dehydration is not a common problem, care has to be taken in special circumstances, as when participating in strenuous exercise, adapting to very hot climates or when severe diarrhoea has occurred. Special attention should be given to adequate fluid intake by particular groups of the population notably children and the elderly.

Social drinking may involve the consumption of alcoholic beverages (Chapter 8). The serious adverse effects on the health of individuals of chronic excessive alcohol consumption are well documented: it is associated with a variety of debilitating diseases and may result in addiction with consequential social and mental problems. The

potential social costs of even moderate consumption cannot be discounted in regard to increased risk of accidents on the roads and in the home. Nevertheless, low or moderate consumption adds pleasure to eating and aids social interaction, and there is little evidence of ill effects in most people. Indeed, there is consistent evidence of a benefit from moderate consumption of alcoholic beverages in respect of CHD.

A significant trend in industrialized societies is the decreased daily energy expenditure as occupations and leisure activities become more sedentary. There is strong evidence that physical inactivity confers a substantial public health burden (Chapter 9) in terms of increased prevalence of obesity, diabetes, hypertension, cardiovascular disease and osteoporosis. Clearly the importance of even moderate levels of physical activity need to be recognized in health promotion programmes.

Chapter 10 presents an overview of current understanding of oral health. A clear message emerges about the importance of regular oral hygiene with fluoride toothpaste for the prevention of tooth decay and gum disease. Regular professional dental care is also recommended. As far as diet is concerned, some individuals at high risk still need to be advised to limit their frequency of consumption of carbohydrate-rich or acidic foods and drinks.

There are considerable gaps in knowledge on most of the issues reviewed here. A few have been pointed out in these chapters, but research continues and continual updating is essential. The science needs to be continually and critically assessed. The communication of science-based messages and the application of this knowledge are equally important.

Individuals may acquire their knowledge about a healthy lifestyle through schooling, the media (newspaper, magazines, television, radio), medical practitioners, information

from industry, product labels, and public health campaigns and other sources. It is vital, therefore, that these communication efforts provide the correct information in a form understandable to the targeted audience. For example, dietary guidelines are usually formulated in terms of nutrients, but consumers buy foods and need to be able to translate nutrient recommendations into how best to purchase foods. Better interaction among scientists, educators and professional communicators, journalists, medical doctors, industrialists and legislators is therefore essential to achieve improved communication of important messages concerning a healthy lifestyle.

## GLOSSARY

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**Adipose tissue:** Tissue, found throughout the body, in which fat is stored as an energy reserve. Excessive expansion leads to obesity.

**Alcohol:** The common name for the substance with the chemical names *ethyl alcohol* or *ethanol*. Produced during fermentation of sugars by yeasts, it is the characteristic component of alcoholic beverages.

**Anion:** A negatively charged ion such as a chloride ion, Cl<sup>-</sup>.

**Antioxidant:** Any substance which can delay or prevent oxidation in the presence of oxygen.

**Atherosclerosis:** A degenerative disease of arteries in which there is thickening caused by an accumulation of material (plaque) beneath the inner lining, eventually restricting blood flow. The material characteristically contains cholesterol and macrophage cells.

**Blood pressure:** The pressure exerted by the circulating blood on the walls of arteries and veins and in the heart chambers. When the heart contracts, the maximum pressure exerted is the systolic pressure; during relaxation, the minimum pressure is the diastolic.

**Carbohydrates:** Components of foods containing carbon, hydrogen and oxygen. The term *carbohydrate* encompasses simple sugars, monosaccharides (e.g. glucose) and disaccharides (e.g. sucrose), oligosaccharides (containing several monosaccharide units) and polysaccharides. Starch is the only important food polysaccharide that can be digested in the small intestine. The indigestible polysaccharides are the main components of “dietary fibre”.

**Cardiovascular disease (CVD):** Any one of numerous abnormal conditions characterized by dysfunction of the heart and blood vessels (see also *coronary heart disease and stroke*). Other types of CVD mentioned in this book are *congestive heart failure* and *sudden cardiac death*.

**Carcinogen:** A substance capable of inducing cancer; *carcinogenesis* is the complex, multistep process of cancer development.

**Carotenoids:** A group of red, orange and yellow pigments found in plant foods and in the tissues of organisms that consume plants. Carotenoids have antioxidant activity. Some, but not all, can act as precursors of vitamin A; the principal of these is  $\beta$ -carotene, the most common of the carotenoids.

**Case-control study:** A type of epidemiological study in which the exposure of patients to factors that may cause their disease (cases) is compared with the exposure of subjects without the disease (controls) to the same factors.

**Cation:** A Positively charged ion such as a sodium ion, Na<sup>+</sup>.

**Cholesterol:** A lipid (sterol) made in the body from acetyl-CoA and present in the diet; a constituent of cell membranes (especially in nervous system tissues), blood and atherosclerotic plaques.

**Chylomicrons:** Large particles composed mainly of triglycerides stabilized with a protein and phospholipid surface, which carry fats recently absorbed from a meal in the bloodstream.

**Cis fatty acid:** The form of most naturally occurring unsaturated fatty acids, where the hydrogen bonds are on adjacent sides of double bonds, resulting in a bend in the hydrocarbon chain at that point.

**Cognitive ability:** Knowledge, perception.

**Cohort or follow-up study:** Type of epidemiological study which measures exposure to factors that may affect health in a group of people (cohort) and relates these factors to the subsequent disease experience (during follow-up).

**Congestive heart failure:** See *cardiovascular disease*.

**Coronary heart disease (CHD):** Restriction of blood flow to the coronary arteries, often characterised by chest pains (angina); may result in a heart attack. The main cause of reduced blood flow is the accumulation of atherosclerotic plaque, a disease known as *atherosclerosis*.

**Dental caries:** A disease affecting the hard tissues of the teeth resulting in progressive decay. Bacteria that accumulate in a dense mass known as plaque on the surface of the teeth ferment dietary carbohydrates (see *fermentation*) to form acids that demineralize the hard tissues underneath. Hence, *cariogenicity* refers to the capacity of a food or drink to lead to caries in those who consume it. Periodontal disease is a related bacterial infection that affects the softer supporting tissues of the teeth.

**Diabetes mellitus:** A metabolic disorder in which the hormone insulin is ineffective either because of a failure of the pancreas to secrete it (type I insulin-dependent, [IDDM], or juvenile onset) or because target tissues are relatively insensitive to its action (type II, non-insulin-dependent [NIDDM], or maturity-onset, diabetes).

**DMFS, DMFT:** Number of decayed, missing and filled permanent teeth surfaces or permanent teeth.

**Electrolytes:** Positively or negatively charged ions dissolved in the extracellular or intracellular water (see also *anion* and *cation*).

**Energy expenditure:** Utilization by the body of chemical energy from food components or body stores during the processes of metabolism which is eventually dissipated as heat plus the heat generated by muscular activity, either in shivering or in physical activity. Usually used to mean the day's total energy (calorie) loss as heat.

**Energy intake:** The chemical energy in foods that can be metabolized to produce energy available to the body; usually used to mean the day's total energy (calories) supplied by all the food and drink consumed.

**Energy density:** Energy content per gram of food.

**Enzymes:** Proteins which catalyse the reactions of metabolism, speeding them up without themselves being used up in the reaction. Each enzyme is specific for a given substrate and/or reaction.

**Epidemiology:** The study of associations between patterns of disease in populations and environmental, lifestyle or genetic factors.

**Extracellular:** Inside the body but not within cells; hence, *extracellular compartment* refers to the space, mainly water, outside the cells.

**Fat:** Triglycerides (triacylglycerol) that are either solid (e.g. in margarine, shortening, lard, etc.) or liquid (e.g. vegetable or fish oil) at room temperature (see also *Oil*)

**Fat-soluble vitamins:** Vitamins A, D, E and K are the fat-soluble vitamins (see also *vitamins*).

**Fatty acids:** Organic acids with a hydrocarbon chain of varying length; constituents of triglycerides and related lipids.

**Fermentation:** Metabolism to extract energy from substrates. In the context of dietary fibre, fermentation involves anaerobic (without oxygen) degradation of indigestible carbohydrates by the microflora (mainly bacteria) of the large intestine. Fermentation may also involve the degradation of sugars or starches (following enzymic digestion by saliva) in the mouth by cariogenic bacteria.

**Free radical:** Any chemical species capable of independent existence that contains one or more impaired electrons. Most free radicals are unstable and highly chemically reactive.

**Gastrointestinal tract:** The organ along which food travels from the mouth until the undigested remnants emerge as stools. Mixing and some digestion occur in the stomach where the environment is acidic; most digestion and absorption of nutrients occurs in the small intestine; the large intestine, principally the colon, contains very large numbers of microorganisms capable of fermenting food components that have escaped digestion in the small intestine.

**Genetic:** Inherited; a *genetic disease* is one that is inherited via a faulty gene.

**High-density lipoproteins (HDL):** Plasma lipoproteins containing relatively low concentrations of cholesterol and other lipids; thought to be beneficial because they cycle cholesterol out of tissues.

**Homeostasis:** A relative constancy in the internal environment of the body, naturally maintained by adaptive responses that promote healthy survival.

**Hypercholesterolaemia:** Concentrations of cholesterol in the blood higher than normal (or reference) values. Causes include dietary and genetic.

**Hyperglycaemia:** A greater than normal concentration of glucose in the blood, most frequently associated with diabetes mellitus.

**Hyperlipidaemia:** A condition arising from an increased concentration in the blood of cholesterol triglycerides or both. These lipids are in the form of lipoproteins.

**Hypertension:** A disorder characterized by elevated blood pressure persistently exceeding systolic/diastolic pressures of 140/90 mm Hg.

**Hypertriglyceridaemia:** Concentrations of triglycerides in the blood higher than normal (or reference) values.

**Insulin:** A hormone secreted by the pancreas in response to consumption of food. It circulates in the blood and assists in the transport of glucose into cells and activates or suppresses the activities of various enzymes.

**Intervention trials:** Trials in which one or more factors that may affect health are altered, with the aim of demonstrating beneficial effects compared with a control group not receiving the intervention.

**Intracellular:** Within cells; the *intracellular compartment* refers to the space, mainly water, inside cells.

**In vitro:** From the Latin meaning “in glass”. The term is applied to biological processes studied experimentally in isolation from the organism, as distinct from *in vivo*, which refers to the study of processes in the living organism.

**In vivo:** A reaction or study carried out in the living organism.

**Ischaemia:** Reduced or inadequate blood, and thus oxygen, supply to a part of the body.

**Life expectancy:** Expected *further* number of years of life measured at any age.

**Lipid:** General name for fatty materials insoluble in water (nonpolar), including fats, oils, phospholipids and cholesterol.

**Lipoproteins:** Particles composed of specialized proteins and lipids (triglycerides, phospholipids and cholesterol) which enable lipids (which are water-insoluble) to be carried in blood plasma.

**Low-density lipoproteins (LDL):** Plasma lipoproteins containing high concentrations of lipids (which are low in density compared to that of water), including cholesterol. Increased concentrations are a risk factor for coronary heart disease.

**Macronutrients:** Carbohydrates, fats and proteins.

**Medium-chain fatty acids:** Fatty acids with chain lengths generally of eight and ten carbon atoms.

**Meta-analysis:** Quantitative literature review that combines the results from published epidemiological and intervention studies, thus allowing greater statistical power. In a good meta-analysis, quality criteria for the studies to be included are applied.

**Metabolism:** Complex interacting network of biochemical reactions within living organisms.

**Micronutrients:** Vitamins, mineral salts and trace elements.

**Nutrient density:** Nutrient content of a food expressed in relation to energy content (e.g. mg per 1000 kcal).

**Obesity:** An excessive accumulation of body fat, often defined as a body mass index (BMI) greater than 30. BMI is the ratio of body weight in kilograms to height in metres squared.

**Oestrogen (estrogen):** One of a group of hormonal steroid compounds that promote the development of female secondary sex characteristics; sometimes used in the prevention of osteoporosis.

**Oil:** Triglycerides (fats) that are liquid at room temperature.

**Oligosaccharide:** See *carbohydrate*.

**Osmotic pressure:** The pressure exerted when ions flow across a membrane from one cellular compartment to another to equalize the ionic concentration, which is normally close to what is obtained by adding 9 g salt ( $\text{Na}^+\text{Cl}^-$ ) to 1 litre distilled water.

**Osteoporosis:** A disorder characterized by an abnormally low density of the bone mineral. The condition predisposes to fracture of certain vulnerable bones and occurs most frequently in women after menopause.

**Oxidation:** A chemical reaction that involves the loss of electrons; it usually but not always involves direct participation of oxygen and is an important process by which food components are utilized by the body's metabolism.

**Oxidative stress:** A condition in which the production of oxidants and free radicals exceeds the body's ability to inactivate them.

**Phospholipid:** A lipid that contains phosphorus, most commonly a compound of glycerol with two fatty acids and one phosphate group. Normally the phosphate is also linked to a small molecule such as choline, serine, ethanolamine or inositol.

**Polysaccharide:** A carbohydrate polymer formed by the linking of many monosaccharides.

**Reactive oxygen species (ROS):** Forms of oxygen that have enhanced chemical reactivity compared with the normal oxygen molecule; many are free radicals (see *also free radical*).

**Risk factor:** Physical condition or lifestyle that in epidemiological studies appears to increase susceptibility to a particular disease.

**Saturated fat:** Triglyceride with a preponderance of saturated fatty acids.

**Saturated fatty acid:** Fatty acid whose hydrocarbon chain contains no double bonds.

**Short-chain fatty acids:** Fatty acids with chain lengths of two to six carbon atoms.

**Stroke:** Interference with blood circulation in the brain, starving one or more parts of the brain of oxygen. The medical term is *cerebrovascular accident*.

**Sucrose:** A disaccharide of glucose and fructose.

**Sudden cardiac death:** See *cardiovascular disease*.

**Sugars:** Simple carbohydrates, generally with a sweet taste and soluble in water. The term is usually reserved for mono- and disaccharides (see *carbohydrates*). In non-scientific English, the disaccharide sucrose is referred to as sugar.

**Thrombosis:** Blockage of an artery resulting from aggregation of platelets and fibrin, a fibrous protein.

**Tocopherols, tocotrienols:** Forms of vitamin E.

**Trans fatty acid:** A form of unsaturated fatty acid that is straight (rather than bent, i.e. *cis*) at the double bond; not abundant in natural edible oils but occurring in ruminant fats and formed during some manufacturing processes.

**Triglyceride:** See *Oil*.

**Unsaturated fat:** Triglyceride with a preponderance of unsaturated fatty acids.

**Unsaturated fatty acid:** Fatty acid whose hydrocarbon chain contains at least one double bond. Monounsaturated fatty acids contain one double bond; polyunsaturated fatty acids contain two or more.

**Vitamins:** Organic substances that cannot be produced in the body but are essential for cellular functions and must be obtained from the diet.

**Water-soluble vitamins:** Vitamins of the B group and vitamin C are the main water-soluble vitamins.