11.5 Diseases of affluent societies and the need f

11.5 Diseases of affluent societies and the need for dietary change 1891

ESSENTIALS Nutritional problems of many countries depend increasingly upon the stage of technical and economic development rather than geo- graphical location. People in affluent societies have ready access to food all year round. Traditional dietary patterns are being eroded and in many affluent societies replaced by diets which are higher in fats and/or free sugars and consequently more energy dense. Obesity is the most obvious and important nutritional disease in affluent soci- eties, with comorbidities including type 2 diabetes, coronary heart disease, hypertension, some cancers, gallstones, osteoarthritis, and obstructive sleep apnoea. Obese people may also be disadvantaged by social, economic, and psychological effects. Particular dietary constituents promote or protect against coronary heart disease by their effect on cardiovascular risk factors, and some promote or pro- tect against various cancers. While those at the highest personal risk are likely to show the greatest individual benefit from dietary and lifestyle changes, national rates of nutrition-related diseases will best be reduced if changes are made by the population at large. The main purpose of such recom- mendations is to reduce the risk of morbidity and mortality in those who are in the prime of life. Even greater reduction in morbidity and mortality and an improvement in life expectancy may occur in suc- ceeding generations if they have reduced lifetime exposure to risk factors related to lifestyle. Many different dietary patterns are compatible with widely ac- cepted nutritional recommendations for overnourished societies. More recent recommendations include a wider acceptable range of macronutrient intakes than had previously been suggested. There are multiple sources of nutritional advice, not all based on sound science. The following statements are representative of universally accepted advice: • Energy balance is essential for body weight control, hence energy- dense foods high in fat and sugars should be restricted to avoid excess weight gain • Wide ranges of fat (25–40% total calories) and carbohydrate (45– 60% total calories) intakes are acceptable provided appropriate food sources predominate • Saturated fat and trans-unsaturated fat intake should together provide less than

10% total energy, the remainder being from cis mono- and polyunsaturated fat • Vegetables, fruit, and pulses (which are also sources of protein) and whole-grain cereals should provide most of the carbohydrate • Intake of salt and foods rich in salt should be reduced so that so- dium intake is below 100 mmol/d (6 g NaCl) • Alcohol should be consumed in moderation (1 to 2 drinks/day) by those who choose to drink All dietary patterns should include a wide variety of foods to en- sure a nutritionally adequate diet Introduction The current nutritional problems of many countries depends more upon the stage of technical and economic development than geo- graphical location (Table 11.5.1). Until about 10 000 years ago our ancestors were hunter-gatherers, most of whom collected a variety of plant foods, but also ate meat and fish. They ate little or no salt, alcohol, milk (other than breast milk), cereal, and no free sugar apart from honey. Studies of contemporary hunter-gatherers suggest that malnutrition was uncommon unless illness or injury supervened, and that the noncommunicable diseases (NCDs) of affluent societies were rare. However, the facts that life expectancy was short and that today's food supply bears little resemblance to the food consumed by our ancestors suggest that the diet of hunter-gatherers does not ne- cessarily provide indicators as to appropriate dietary practices in the 21st century. Furthermore, a hunter-gatherer type diet would not be sustainable today. In most parts of the world hunter gatherers acquired a more set- tled existence and became predominantly agriculturists and pastor- alists. Where traditional dietary patterns based on a variety of food sources were retained, diet-related NCDs and premature mortality from them remained relatively uncommon. However where there has been reliance on a single staple food which may lack essential nutrients or fail, malnutrition may result. A similar situation may occur, in developing countries where many people live in urban slums or periurban shanties, often in overcrowded insanitary ac- commodation, or in relatively affluent societies where poverty and affluence may co-exist. People have lost contact with the land and their food traditions. Food is expensive. Mothers of young chil- dren are required to go out to work and often cannot breastfeed. Undernourished young children are susceptible to infectious diseases
11.5 Diseases of affluent societies and
the need for dietary change J.I. Mann and A.S. Truswell

 and may develop marasmus. Some adults are excessively lean, others become obese, and excessive intake of alcohol is frequent. A totally different situation prevails among the prosperous people in affluent societies, especially when traditional dietary practices have been abandoned or modified and increasingly among the affluent in developing countries. They have access to a wide variety of safe, relatively cheap foods all year round. The environment is generally conducive to the consumption of a diet which is typically high in fats and or free sugars and dense in energy. Among such societies deaths from infectious diseases are infrequent, life expectancy has increased, and noncommunicable diseases, many linked in some way to in- appropriate nutrition, are the major causes of premature death and ill health. Malnutrition occurs mainly in frail elderly people and the sick. Approaches to the study of nutrition-related diseases The nutritional component of noncommunicable diseases is more difficult to study than classical nutrition deficiency diseases because noncommunicable diseases develop slowly and are multifactorial. The practice of evidence-based medicine ideally requires that an inter- vention recommended for the treatment or prevention of a disease should be proved to be of benefit in one or more randomized con- trolled trials. The evidence for the benefit of an intervention may be strengthened by the aggregation of the results of like studies in a meta- analysis. However, trials involving nutritional interventions and clinical endpoints are much more difficult to undertake than those involving drugs. It is possible to study the effects of a food component given as a

pharmaceutical (e.g. antioxidant nutrients given as a tablet), and some trials have been undertaken to study the effects of dietary manipulations on relatively common nutrition-related diseases (e.g. diabetes, hyper- tension). Clinical trials to demonstrate the effect of various dietary measures on the risk of coronary heart disease have been undertaken and have generated meaningful results when considered in aggregate, but the magnitude and duration of trials required to demonstrate the ef- fect of dietary change on cancer are such that few have been attempted. Much research involving the role of diet in chronic degenerative disease has centred around the effects of diet on modifying risk fac- tors rather than the disease itself. For many chronic diseases there are biochemical or clinical markers of risk. High plasma cholesterol is an important risk factor for coronary heart disease, for example, and high blood pressure is a major risk factor for strokes. Innumerable studies have examined the role of different nutrients and foods on plasma cholesterol, blood pressure, or other risk factors. Such studies are easier to undertake and cheaper than population-based studies because far fewer people are studied over a relatively short period of time. They have shown which foods lower cholesterol and so should help protect against coronary heart disease. Epidemiological studies have also played a pivotal role in establishing the role of nutritional factors as determinants of NCDs and the poten- tial of dietary modification in risk reduction. The first clue to the as- sociation between a food, or nutrient, and a disease often comes from observing striking differences in disease incidence between countries (or groups within a country) that correlate with differences in nutri- tional intake. Sometimes, dietary changes over time in a single country have been found to coincide with changes in disease rates. Such obser- vations give rise to hypotheses about possible diet–disease links, ra- ther than proof of causation, because many potential causative factors may be confounded by parallel dietary changes. Case–control studies have sometimes been used as a rapid and inexpensive way of testing hypotheses. These have been of value when comparing biochemical markers of nutrient intakes in the blood of people with (cases) and without (controls) various diseases, but they are of limited value when nutrient intake has been determined by recall of dietary intake. Prospective or cohort studies avoid the biases involved in asking people to recall past eating habits as information about food intake and other characteristics are collected well before onset of the disease. These have played a key role in identifying nutritional factors involved in coronary heart disease and cancer, but even strong nutrient or food and disease associations can be explained by confounding (i.e. the as- sociation between the nutritional factor and some other disease deter- minant). Sometimes the confounding can be quantified, but it is never possible to be absolutely certain that it has not been explained by some other factor which has not been measured, viz residual confounding. Given the difficulty in establishing casual associations between nutrients or individual foods and disease states, a decision as to whether or not to recommend dietary change needs to be based on a portfolio of evidence when evidence from randomized controlled trials is not available. Such evidence might include consistent and strong associations in longitudinal studies, biological plausibility, and corroborative experimental evidence in animals and humans. Obesity Obesity (see also Chapter 11.6) is the most obvious and important nutrition-related disease in affluent societies, its comorbidities Table 11.5.1 Nutrition issues at different stages of technical and economic development Hunter-gatherers Occasional seasonal hunger Malnutrition uncommon General nutritional health good No obesity No hypertension Low serum cholesterol Peasant agriculturists Single crop staple Clinical disorders may result from single or multiple nutrient deficiencies Hypertension may occur Obesity rare Urban slum and periurban shanty town dwellers Inadequate breastfeeding Inadequate food security Diarrhoea and other infective disorders, especially in young children Marasmus Obesity and alcoholism may occur

Affluent societies Energy-dense diets, high in fats and sugars Physical inactivity Obesity, coronary heart disease, and hypertension common Malnutrition may occur in frail elderly and sick people

11.5 Diseases of affluent societies and the need for dietary change 1893 including type 2 diabetes, coronary heart disease, hypertension, some cancers, gallstones, osteoarthritis, and obstructive sleep ap- noea. Obese people may also be disadvantaged by social, economic, and psychological effects. The psychological well-being of children may be particularly affected, and childhood obesity has recently been recognized as a risk factor for fractures in children. Most of the adverse consequences of obesity are appreciably reduced by weight loss, though gallstone formation may not be reduced. Although the genetic component of obesity is acknowledged, its dramatic increase in virtually all westernized countries and many developing countries in recent years provides ample evidence of overwhelming environmental factors. Physical inactivity is an im- portant cause, but frequent consumption of large portions of readily available energy-dense foods (high in fats and/or sugars) which contribute to an energy intake in excess of expenditure probably accounts for much of the global increase in obesity. Frequent con- sumption of sugar-sweetened soft drinks (and fruit juice) appears to enhance excessive weight gain, especially among children. Whole- grain cereals and cereal products, nonstarchy vegetables, and dietary fibre help to reduce the energy density of the diet, promote satiety, and thus reduce the risk of inappropriate weight gain. Current pharmaceutical measures have little to offer in the man- agement of obesity. While bariatric surgery results in weight loss and reduction of comorbidities for some obese people, it seems unlikely that the epidemic of obesity will be reversed unless the environ- ment in which we live is made more conducive to appropriate food choices and more opportunities for physical activity are provided. Such measures will also facilitate attempts by individuals to achieve weight reduction. Reducing the obesogenic environment requires commitment from national and local governments. Despite all of the aforementioned points, there is some cause for optimism in that obesity rates are relatively low among those of a higher socioeconomic status, and the increase in rates of childhood obesity appears to have been halted in countries where preventative measures have been implemented. Coronary heart disease The totality of evidence from experimental, epidemiological, and clinical trial data provide strong evidence for the role of nutritional factors in the aetiology of coronary heart disease and the potential for dietary modification to reduce cardiovascular morbidity and mortality in the population as a whole, in individuals at high risk, and in those who have already experienced a cardiovascular event. Prospective and experimental studies suggest a variety of foods and nutrients that may be involved (Table 11.5.2). Foods that in- crease the risk of coronary heart disease when consumed in large amounts probably do so because they are rich in saturated or trans- unsaturated fatty acids. ‘Protective’ foods contain several different nutrients that may reduce cardiovascular risk. Vegetable oils and nuts contain several potentially ‘protective’ fatty acids (linoleic and oleic acids). Oily fish is rich in very long-chain polyunsaturated fatty acids (eicosapentaenoic and docosahexaenoic acids). Fruit and vegetables are good sources of antioxidant nutrients, folate, potas- sium, and other biologically active substances. Whole-grain cereals are good sources of dietary fibre as well as of some unsaturated oils. Data presented in Table 11.5.3, derived from well-known pro- spective studies of cardiovascular disease, provide an indication of the extent of the potential cardioprotection and risk afforded by some foods and nutrients. Each of the foods and nutrients listed in Tables 11.5.2 and 11.5.3 has an appropriately favourable or adverse ef- fect on one or more of the cardiovascular risk factors (Table 11.5.4). As the global prevalence of obesity increases, risk factors associated with excess adiposity (notably dyslipidaemia, dysglycaemia, inflam- mation) contribute increasingly as ‘causes’ of coronary heart

disease. Thus, it may be appropriate to also consider nutrition-related causes of obesity as causes of coronary heart disease. The strongest evidence for recommending dietary change to re- duce risk of coronary heart disease relates to replacing an appreciable proportion of dietary saturated fat with polyunsaturated fatty acids. Systematic reviews and meta-analyses of both prospective cohort studies and randomized controlled trials suggest a reduction in car- diovascular events which, in the case of the randomized controlled trials, is proportional to the reduction in total and low-density lipo- protein (LDL) cholesterol achieved and comparable with benefit predicted from epidemiological data. LDL cholesterol appears to be a key risk factor for coronary heart disease, and a similar ‘dose re- sponse’ benefit in terms of cardiovascular risk reduction is seen when LDL cholesterol is lowered by means of statin drugs. It is unclear why meta-analyses do not show similar cardioprotection when saturated fatty acids are replaced by monounsaturated fats or carbohydrate, Table 11.5.2 Foods and nutrients which may protect against or promote coronary heart disease Protective Promoting Foods Nutrients Foods Nutrients • Fruits • Vegetables • Antioxidant nutrientsa, folatea • Dietary fibre (nonstarch polysaccharide) • High-fat dairy products • Fatty meats Saturated fatty acids (especially myristic and palmitic acids) Whole-grain cereals • Dietary fibre (nonstarch polysaccharides) • Unsaturated fatty acids Eggs Dietary cholesterol Vegetable oils (e.g. sunflower, safflower, olive, and canola) Unsaturated fatty acids (linoleic, oleic, α-linolenic) Some margarinesb, cooking oils, confectionery, and manufactured foods • Trans-unsaturated fatty acids • Saturated fatty acids Oily fish Eicosapentaenoic and docosahexaenoic acids Nuts Unsaturated fatty acids (oleic, linoleic), vitamin Ea Alcohol (moderate amounts only) a When present in foods, not supplements. b When containing appreciable quantities of trans-unsaturated fatty acids.

 although it may be relevant that monounsaturated fat replacement was undertaken in only a small number of trials, and trials involving carbohydrate replacement did not emphasize the need to consume minimally processed carbohydrate-containing foods rich in dietary fibre which would be more likely to be cardioprotective than rapidly digested carbohydrate. It is noteworthy that the reduction in coronary heart disease which has been occurring in most Western countries since the 1970s has paralleled the reduction in saturated and increase in polyunsat- urated fats, although improved medical management of the disease and its risk factors and reduction in smoking in some sections of the populations will also have contributed to the trend. Trials that have examined potential benefits of dietary manipulations other than those designed to lower plasma cholesterol suggest that fur- ther clinical benefit might accrue from favourable changes in other risk factors, but the evidence is less conclusive. Consumption of oily fish two or more times per week, or a small amount of fish oil taken as a supplement, has been shown to reduce cardiovascular death in those with pre-existing coronary artery disease. Although increased intakes of vegetables and fruit may confer a cardioprotective effect, there is no convincing evidence from clinical trials of benefit asso- ciated with the use of antioxidant nutrient supplements. The role of margarines rich in plant sterols and stanols, which may further lower dietary cholesterol by preventing absorption and reabsorption of dietary cholesterol, is yet to be established with certainty. Community programmes aiming to change diet along the lines indicated here have been shown to reduce cardiovascular risk fac- tors and one, the North Karelia Project in Finland, has shown that cardiovascular disease mortality in the intervention county de- creased to a greater extent than might have been expected on the basis of experience in other Finnish counties. The availability of appropriate food choices at reasonable cost is an essential compo-nent of any programme aimed at reducing cardiovascular risk, since rates are highest in people of

the lowest socioeconomic status. While those at the highest personal risk are likely to show the greatest indi- vidual benefit from dietary and lifestyle changes, national coronary heart disease rates will best be reduced if changes are made by the population at large. The main purpose of such recommendations is to reduce the risk of morbidity and mortality from coronary heart disease in those who are in the prime of life. Even greater reduction in morbidity and mortality and an improvement in life expectancy may occur in succeeding generations if they have reduced lifetime exposure to risk factors related to lifestyle. Hypertension and stroke Salt (sodium chloride) Three dietary factors are well established as raising blood pressure. The longest known is salt, sodium chloride. In a few isolated com- munities salt was not available until recently, and there high blood pressures were rare or absent. Usual sodium intakes of around 150 mmol/(8.8 g NaCl) or more per day are about six times more than the physiological requirement (human milk contains only 7 mmol sodium/litre). Salt used to be important for preserving food before canning, re- frigeration and rapid transport, and people are now habituated to its flavour in foods like bread. The relationship between sodium intake Table 11.5.3 Age adjusted relative risk of coronary heart disease according to quintile of intake of certain foods or nutrients Study population Relative risk according to quintile of intake p for trend 1 2 3 4 5 43 757 male health professionals (40–75 years) Total dietary fibre (Rimm et al., 1996) 1.00 0.97 0.91 0.87 0.59 <0.001 75 521 female nurses (38–63 years) Whole-grain consumption (Liu et al., 1999) 1.00 0.87 0.82 0.72 0.67 <0.001 84 136 female nurses (30–55 years) Nuts <0.001 (Bernstein et al., 2010) 1.00 0.73 0.91 0.76 0.68 43 757 male health professionals (40–75 years) Trans fatty acids (Ascherio et al., 1996) 1.00 1.24 1.33 1.40 1.57 <0.001 Table 11.5.4 Some effects of nutrients which promote or protect against coronary heart disease on cardiovascular risk factors Nutrient Effect Promoting Saturated fatty acids ↑ total and LDL cholesterol ↑ thrombogenesis ↓ insulin sensitivity Trans-unsaturated fatty acids ↑ total and LDL cholesterol and Lp(a) Dietary cholesterol (when taken in large amounts) ↑ total and LDL cholesterol Protective Dietary fibre ↓ total and LDL cholesterol ↑ insulin sensitivity Antioxidant nutrientsa ↓ oxidation of LDL Unsaturated fatty acidsb ↓ total and LDL cholesterol ↓ arrhythmias, thrombogenesis ↑ increase; ↓ decrease a When consumed in fruits and vegetables, rather than supplements b C18:1, n-9: oleic acid C18:2, n-6; linoleic acid C18:3, n-3; α-linolenic acid C20:5, n-3: eicosapentaenoic acid C22:6, n-3; docosahexaenoic acid

11.5 Diseases of affluent societies and the need for dietary change 1895 and blood pressure cannot be studied by asking people to recall dietary intake of salt. Most of the salt consumed (c.85%) is added at the time of manufacture, rather than during food preparation or at the table, hence 24-h urinary sodium excretion rather than dietary intake measurements are needed to accurately assess salt intake, al- though sodium measurements in spot urine samples are considered to be adequate for assessing intakes of populations. Sodium excretion (reflecting intake) and blood pressures fluc- tuate markedly, and some individuals are more salt-sensitive than others. Nevertheless, surveys within one country (e.g. the 1986–1987 British National Dietary and Nutrition Survey) and internationally (the Intersalt Study involving 10 000 people in 32 countries) have shown a clear relationship between urinary so- dium and blood pressure, and a Finnish cohort study found an increased risk of cardiovascular disease in those who had high 24-h urinary sodium. There is strong confirmation from carefully controlled primate research that salt is causally related to essential hypertension. Blood pressure rose significantly over an 18-month period when salt was added to the diet of chimpanzees that nor- mally eat a vegetarian and fruit diet, and fell again when the salt was stopped. Several controlled clinical trials in humans have shown that when salt intakes are reduced to around 70 mmol/litre, blood pressure falls more in people with mild to

moderate hyper- tension. Salt restriction can contribute to the treatment of hyper- tension, but major dietary change is needed because so much is derived from manufactured food, emphasizing unprocessed foods and low-salt bread (ordinary bread contains over 100 times more salt than wheat flour). Weight reduction Overweight and obese people have higher blood pressures than those who are lean and, if they lose weight, blood pressure falls even if the usual salt intake is maintained. An Australian clinical trial showed that weight reduction (maximum loss 7.4 kg) compared favourably with metoprolol in the treatment of mild hypertension, and diet was associated with an improved plasma lipid profile not seen on the drug. Alcohol Alcohol intake is emerging as the third of the important envir- onmental factors associated with raised blood pressure. In epi- demiological studies, blood pressure, especially systolic, increases progressively when reported alcohol intake increases above three drinks per day. Several intervention studies have shown that reduc- tion of alcohol intake can produce an appreciable reduction in blood pressure among hypertensive heavy drinkers. For example, one trial showed that replacing standard beer (5% alcohol) with a reduced- alcohol beer (0.9% alcohol) produced a reduction in alcohol intake from 450 to 64 ml/week and a significant fall in blood pressure. Other factors Other components that may lower blood pressure are not as clearly established. Potassium, probably acting as an antagonist to sodium, has been shown in controlled trials to lower blood pressure modestly, but this was when given in pharmacological doses. Potassium may have been one of the operative factors in the Dietary Approaches to Stop Hypertension (DASH) trials that have shown an appreciable blood pressure lowering effect of substantial quantities of fruits and vegetables. In these trials the addition of low-fat dairy foods pro- duced additional blood pressure lowering, but the effects of calcium have been less marked in controlled trials. Blood pressure is an important determinant of ischaemic stroke and cerebral haemorrhage, so that all the nutritional determinants of hypertension may be regarded as relevant to their cause. In add- ition, prospective studies have consistently demonstrated that fruit and vegetables protect against ischaemic stroke. Although it appears that most categories of fruit and vegetables are protective, the effect is particularly striking for cruciferous vegetables, green leafy veget- ables, and citrus fruits. Diabetes mellitus and the metabolic syndrome Rates of type 2 diabetes have escalated in most affluent societies to the extent that the condition is considered to have reached epidemic proportions in many countries. The precise nature of the metabolic syndrome is not clearly understood, and some have questioned whether it should be regarded as a clinical entity. Nevertheless, the constellation of abnormalities (including abdominal obesity, hyper- glycaemia, raised blood pressure, dyslipidaemia, and increased in- sulin levels, which constitute the ‘syndrome’), does identify people likely to develop type 2 diabetes and who are at appreciably increased risk of cardiovascular disease. Where information is available, it ap- pears that the frequency of the ‘syndrome’ has also increased and that risk factors are the same as for type 2 diabetes. Epidemiological evidence suggests that type 2 diabetes is un- common in people eating a range of ‘traditional diets’ high in fresh fruit, vegetables and minimally processed cereals, and relatively low in fat, especially saturated fat. Diabetes prevalence seems to increase rapidly when traditional lifestyles are exchanged for the Western way of life, particularly when such transitions occur over a short time span. Such changes are occurring in China and India, where type 2 diabetes has already created an enormous public health problem. Similar findings had been noted earlier in Micronesians, Polynesians, American Indians, and Aboriginal Australians, as well as in Asian Indian immigrants to Fiji, South Africa, the United Kingdom, and Mauritius and among Chinese in Singapore, Taiwan, Hong Kong, and Mauritius. The change from traditional to a Western way of life is gener- ally associated with a reduction in physical activity and an increase in the energy density of the diet, resulting from increased intakes

of fats and sugars, with the resultant energy imbalance leading to increasing rates of overweight and obesity. Lack of physical ac- tivity and increasing degrees of obesity (especially abdominal adi- posity) have consistently been shown in longitudinal studies to be associated with the risk of developing type 2 diabetes. Globally and nationally, rates of type 2 diabetes have increased in parallel with increasing obesity. Genetic determinants of diabetes should not be underestimated, but they clearly cannot explain the exponential in- crease in so many countries. While any cause of energy imbalance leading to excessive weight gain will increase the risk of type 2 diabetes, there is less certainty re- garding the role of individual macronutrients in the aetiology. Excess sucrose has not been clearly established as an important dietary factor, except when high intakes contribute to an increase in energy density. However excessive energy intakes though sugar-sweetened

SECTION 11 Nutrition 1896 beverages have been implicated as a cause of type 2 diabetes inde- pendent of an effect on body fatness. A high intake of saturated fatty acids increases insulin resistance, an underlying abnormality in type 2 diabetes and the metabolic syndrome, independently of an effect of excess adiposity, hence sat- urated fats are regarded as a probable contributor. One large pro- spective study of health professionals in the United States of America has found that a high intake of low glycaemic index foods tends to protect against type 2 diabetes and that this effect is independent of other individual dietary attributes. A high intake of dietary fibre has been shown to enhance insulin sensitivity in insulin-resistant indi- viduals, so that foods rich in dietary fibre and with a low glycaemic index are probably protective. Thus, it seems most likely that a com- bination of factors is responsible. Although we do not fully understand the complex mechanisms by which genes and environment interact to result in type 2 diabetes, randomized controlled trials among individuals with impaired glu- cose tolerance carried out in Finland, the United States, China, and India provide strong support for the suggestion that lifestyle modi- fication can help to prevent or at least appreciably delay the onset of type 2 diabetes. Interventions in the Finnish Diabetes Prevention Study (Box 11.5.1) resulted in an approximately 60% reduction in rate of progression from impaired glucose tolerance to type 2 dia- betes, a benefit which has persisted for well beyond the six-year dur- ation of the Study. Of particular interest is the fact that remarkably few of those individuals who complied with at least four of the five target interventions progressed from impaired glucose tolerance to type 2 diabetes. The benefits appear to accrue principally from re- duction in excess body fat rather than a change in any of the indi- vidual nutrients. The United States, Chinese, and Indian studies have reported comparable results. Similar lifestyle interventions have been shown to increase insulin sensitivity in insulin-resistant individuals prior to the development of impaired glucose tolerance or diabetes. Convincing randomised controlled trial evidence has shown that appreciable weight reduction (around 15kg), especially when achieved relatively soon after diagnosis can result in remission of established type 2 diabetes even in those already on drug treatment. It is yet to be established for how long remission can be sustained. Less marked weight loss and appreciable dietary change (high in- takes of dietary fibre, especially soluble forms, low glycaemic index, minimally processed wholegrain foods and reduced saturated fats) even in the absence of appreciable weight loss, can improve gly- caemic control (often with reduced requirements for hypoglycaemic drug treatment) and cardiovascular risk factors in those with type 2 diabetes regardless of the duration of the disease. Although diet is important in the management of type 1 diabetes, nutritional factors do not appear to have contributed to the aeti- ology of the disease to the same extent as for type 2 diabetes. Genetic and other environmental factors are believed to be more important. Some studies have suggested, however, that infants who have been breastfed may have a reduced risk

of type 1 diabetes in later life, and this observation could be linked with immune mechanisms known to be associated with this condition. Cancers The development of cancer involves several stages and occurs over a long period of time. Nutritional factors may operate at one or more of these stages, but given the time scale of cancer development it is hardly surprising that few data from intervention trials are available, and data relating dietary factors to various cancers are principally derived from epidemiological associations and animal experiments. Causal factors Despite the difficulty in assessing dietary intake over the prolonged period during which cancer develops, it has been estimated that about one-third of all cancers in Western countries may be asso- ciated with diet. The dietary and nutritional factors that may play a role in human cancer are listed in Table 11.5.5. Restriction of total energy intake, provided that nutrient requirements are met, has been clearly shown to reduce the risk of cancer in experimental animals, and obesity in humans is one of the most powerful and consistent epidemiological associations with cancers. Obesity is as- sociated with insulin resistance and increased levels of inflamma- tory markers and insulin-like growth factors, which may increase cancer risk. These effects are reversed by weight loss. High intakes of total fat are strongly correlated with colorectal, breast, prostatic, and pancreatic cancer in ecological (between coun- tries) studies. The effects of alcohol as a risk factor for breast cancer as well as cancer of the mouth, larynx, and pharynx are consistent and strong. Alcohol probably increases the risk of liver cancer because large intakes lead to cirrhosis of the liver, which is associated with liver cancer, regardless of cause. High intakes of red meat have been associated with an increased risk of colon cancer. Processed meats (the definition of which differs in different countries) have also been linked with colorectal cancer. Haem rather than iron per se is one possible explanation since it is susceptible to endogenous nitrosation by bacterial flora in the colon, and nitroso compounds can increase the likelihood of neoplastic change. Such effects are not seen with fish or poultry, which appear to be protective against colorectal cancer. Other clearly established nutrition-related promoters of cancers tend to operate regionally: salt and salted fish increase the risk of stomach and nasopharyngeal cancer in Japan and China, and maté, consumed at high temperatures in Brazil, is an important cause of oesophageal cancer. Protective factors Vegetables and fruit are generally accepted as important protective factors, particularly against lung, stomach, and colorectal cancers. Antioxidants (especially vitamin C, vitamin E, carotenoids, and Box 11.5.1 Lifestyle modifications, targets for the intervention group in the Finnish Diabetes Prevention Study • Weight loss of 5–7% initial body weight or a weight loss of 5–10 kg depending upon degree of obesity. • Reduce total and saturated fat by encouraging low-fat dairy and meat products. • Prefer unsaturated soft margarines and vegetables oils rich in mono- unsaturated fatty acids. • Increase whole grains, vegetables, and fruit. • Physical activity, at least moderate intensity for a minimum of 30 minutes daily.

 flavonoids), glucosinolates (found in brassica vegetables), sulphur components (in Allium species—onions and garlic) and folates have all been shown to have anticancer properties, which may explain the protective effects that have been demonstrated mainly in case– control studies. Possibly of even greater importance is the protection (particu- larly against colorectal cancer) conferred by dietary fibre (nonstarch polysaccharide) present in many minimally processed cereal foods, as well as fruits and vegetables. Dietary fibre and resistant starches escape digestion in the small intestine and are fermented in the large bowel by the colonic microbial flora. Short-chain fatty acids are pro- duced, one of which, butyric, is an antiproliferative agent. Dietary fibre may further reduce the risk of large-bowel cancer by increasing stool bulk and decreasing transit time, which in turn reduces the

op- portunity for colonocytes to be in contact with carcinogens. While there is renewed interest in the potential protective ef- fects of selenium, tomatoes, and lycopene against prostate cancer, and possible protection of folate and calcium against cervical and colorectal cancers, respectively, it seems likely that reducing rates of obesity may have a more marked effect on reducing nutrition-related cancers than modifying intakes of individual nutrients or foods. Dental caries Archaeological evidence shows that in ancient times dental caries was exceptionally rare in young people. In a recent survey of children's dental health in the United Kingdom the overall preva- lence of dental caries was 43% and the average number of decayed missing and filled teeth among 12-year-olds (permanent teeth) was 0.8. Among adults in the United Kingdom there appears to be an improvement. The Adult Dental Health Survey 2009 reported that in the 85 plus age group, 30% were edentulous (compared with al- most 80% in 1988). In the 75–84-year-old group 15% are edentu- lous and only 5% of those aged 65–74 years. The extent to which dental caries contributes to ill health throughout the life course has resulted in the condition being considered as a major chronic non- communicable disease. Several strands of evidence suggest a nutritional cause. Dental caries once had a very low prevalence among the indigenous populations of many countries where unrefined foods form the bulk of the diet (e.g. China, Uganda), and this increased rapidly within a few years of the addition of sugar and other refined foods. A similar change has been shown experimentally in monkeys. In a classical experiment carried out in a Swedish mental hospital, vo- lunteers given toffee apples, chocolate, and caramel in addition to their controlled diet had a 13-fold greater number of tooth surfaces becoming carious each year, compared with those eating the con- trolled diet alone. A 2013 meta-analysis of cohort, population and cross-sectional studies, and a small number of intervention trials conducted on behalf of the World Health Organization (WHO), found evidence of moderate quality that caries is lower when free sugars intake is less than 10% total energy and some evidence of a further reduction when intakes are below 5%. Although fluoride in the water at one part per million or in toothpaste can appreciably reduce the risk of dental caries, the association between free sugars and dental caries remains even in areas where fluoridated water is consumed. Diverticular disease of the colon The first suggestion that deficiency of dietary fibre may be impli- cated in the aetiology of diverticular disease of the colon came from geographical variations and trends over time in several countries which were compatible with a causative link with low-fibre diets, but such associations could also be explained by several alternative dietary and other environmental influences. The best-documented evidence comes from comparisons of asymptomatic groups of vege- tarians and meat eaters who volunteered to undergo radiological imaging. Radiological diverticular disease was found more fre- quently among omnivores than vegetarians, who had appreciably higher intakes of dietary fibre. Furthermore, when comparing in- dividuals with and without diverticular disease, in both the vege- tarian and nonvegetarian groups those with diverticular disease had appreciably lower intakes of dietary fibre than those with no evidence of diverticula. An increase in dietary fibre intake is widely recommended to patients with symptomatic diverticular disease, a treatment justified by the findings of some (but not all) controlled clinical trials. Table 11.5.5 Nutritional associations of various cancers Factor Causal (↑) Cancers Protective (↓) Obesity ↑ Postmenopausal breast, colorectum, endometrium, gallbladder, kidney, oesophagus, pancreas, prostate Processed and red meat ↑ Colorectum Alcohol ↑ Liver, breast, mouth, larynx, pharynx, oesophageal Salt ↑ Stomach Salted fish (Cantonese style) ↑ Nasopharynx Hot drinks (maté) ↑ Oesophagus Vegetables ↓ Colorectum, oesophagus, stomach Fruits ↓ Lung, stomach, oesophagus Nonstarch polysaccharide/dietary fibre ↓ Colorectum Selenium ↓ Prostate

SECTION 11 Nutrition 1898 Plausible theories concerning pathogenesis have been suggested; small, hard faeces associated with a fibre-deficient diet lead to nar- rowing of the colon and the formation of closed segments in which pressure increases. Additional work is needed by colonic muscles to provide the pressure to move the more solid faeces, producing muscular hypertrophy in addition to diverticula at sites of weakness where blood vessels penetrate the muscular coat. Constipation and the irritable bowel syndrome Ninety-nine per cent (99%) of a large population sample studied in the United Kingdom reported that they defecated at least three times per week but perceived constipation as a frequent com- plaint. Approximately 3% of all prescriptions written in the United Kingdom's National Health Survey were for purgatives and laxatives, at a cost of around £4 000 000, and many times this amount must have been spent in over-the-counter purchases. In another survey, 6% of people aged 18 to 80 years described straining when passing stools. By contrast, constipation is uncommon in populations with a high intake of dietary fibre. In Britain, stool weights in omnivores are usually around 100 g (with a very wide range), whereas in vegetarians with a high fibre in- take, the average stool weight is over 200 g. Furthermore, vegetarians and omnivores with high average daily fibre intakes have transit times of less than 75 h and rarely report constipation, whereas those with lower fibre intakes have transit times ranging from 20 to 124 h and frequently complain of constipation. Controlled clinical trials con- firm that increasing dietary fibre (especially that derived from cereals) relieves the symptoms of constipation. Diets rich in dietary fibre are widely recommended in the treatment of irritable bowel syndrome, despite the absence of good evidence from formal clinical trials. Osteoporosis Osteoporosis is an important cause of morbidity among elderly people, especially women, and the incidence of osteoporotic frac- tures is increasing steadily as people are living longer. The aetiology of osteoporosis is complex; women have a lower peak bone mass in their twenties than men and lose bone rapidly after the meno- pause in association with a decline in oestrogens. Women lose ap- proximately one-half their trabecular bone and one-third of their cortical bone, while men lose one-third of their trabecular bone and one-fifth of their cortical bone. Genetic factors influence peak bone mass and bone loss. These may operate by some established risk factors: family history of osteoporosis, short stature, early meno- pause, white or Asian race, and leanness. However, there are also environmental factors, including cigarette smoking, excessive salt and alcohol intakes, and lack of vitamin D, especially in housebound people with little sun exposure. The role of dietary calcium has been uncertain but there is now convincing evidence that the best way of avoiding osteoporotic fractures in later life is to achieve optimal skeletal mass for one's genetic potential and to retain this as long as possible. The best means of doing so is by ensuring lifelong adequate consumption and maximum absorption and retention of calcium. The need for substantial amounts of dietary calcium, in conjunc- tion with adequate vitamin D, is particularly important during the periods of growth, pregnancy, lactation, and in the postmenopausal years. Fruit, vegetables, and adequate physical activity have also been identified as protective factors. Other diseases Gallstones, appendicitis, haemorrhoids, varicose veins, and hiatus hernia all occur frequently in developed countries and rarely in developing countries, but the evidence linking these diseases to a nutritional cause is tenuous. Gallstones are undoubtedly associated with obesity. Both gall- stones and appendicitis are more common in omnivores than vegetarians, and there are some rather indirect data suggesting an association with diets high in sugars and deficient in dietary fibre. The addition of bran to the diet can make bile less saturated, and experimentally induced gallstones in animals tend to be reduced if fibre-rich foods rich are given. Historical studies provide interesting information; appendicitis rates were compared in two matched groups of South African whites, the privileged group living in uni- versity halls of residence and the other living in

establishments for the indigent, where the diets contained more fibre. Annual rates were 7.8/1000 and 1.8/1000, respectively. Of course, factors other than diets might explain this, but the rates were similar to those found in an almost identical study in Bristol (7.6/1000 in a fee- paying boarding school and 0.8/1000 in an orphanage). The case for dietary change Nutrition research often generates results that may be translated by researchers, self-styled ‘experts’, or the media into potentially confusing and conflicting messages. It is therefore important for governments who develop food and nutrition policies, for doctors and others involved in health and nutrition education, and for con- sumers to have authoritative recommendations that represent con- sensus opinions of nutrition scientists. Dietary or nutrient reference values define intakes of nutrients which are required for growth and maintenance of health and considered to reduce the risk of chronic diseases. These recommendations are intended for policy makers and health professionals who recommend diets for populations, special groups of people within populations, and individuals. Such reference values are unhelpful to the population at large. For the general public, dietary guidelines have been developed to translate reference values into practical advice. Dietary fat Reference values for macronutrients were initially centred around the evidence showing that alteration of dietary fat intake from that typical of most Western countries would be expected to reduce population and individual risk of coronary heart disease. Restriction of saturated fatty acids and trans-unsaturated fatty acids to no more than 10% total energy has been a consistent feature of all sets of national and inter- national reference values. Some have suggested an even lower propor- tion of total energy from these fatty acids for populations, groups, or individuals considered to be at increased risk of coronary heart dis- ease. Until relatively recently recommendations regarding dietary fat included a restriction on total fat intake, typically to less than 30%

 total energy. However, increasing evidence relating to the benefits of replacing saturated with unsaturated fatty acids has (other than trans-unsaturated fatty acids) resulted some countries including the Nordic group of countries suggesting an appreciably higher accept- able upper limit, around 40% total energy (Table 11.5.6). While an increase in unsaturated fatty acids (with a cis- configuration) is associated with a reduced risk of coronary heart disease, the potential contribution of total fat to the energy density of the diet appears to be the continuing justification for the World Health Organisation’s recommendation for continuing the recom- mendation to restrict total fat. There is evidence that high fat diets may promote and perpetuate excessive weight gain and obesity in individuals and populations but the extent to which this should be reflected in a restriction of total fat intake especially as it relates to populations rather than individuals remains a matter of opinion. The lower limit of fat intake in some recommendations is based on the minimum requirement considered to ensure an adequate in- take of fat-soluble vitamins, and the fact that there is no evidence of untoward effects in some Asian and African populations who traditionally consume low-fat diets. Dietary carbohydrate About 50 g of carbohydrate daily is required to avoid ketosis, but many populations maintain an adequate nutritional status when carbohydrate provides up to 80% total energy. Most Western societies are unaccustomed to a high carbohydrate intake and are reluctant to accept substantial increases. WHO and most countries recommend a relatively wide range of acceptable intakes, recog- nizing that source of carbohydrate is likely to be more important than the proportion of total energy provided by carbohydrate (Table 11.5.6). A high intake of free sugars (principally sucrose and, in the United States, high-fructose corn syrup) increases the risk of obesity by increasing the energy density of the diet, or simply by increasing total energy intake (and energy imbalance) when sugary drinks are consumed in excess. Sugars are also associated

with dental caries and in large amounts may enhance the metabolic derangements in people with insulin resistance. Foods with a high intake of free sugars are frequently nutrient poor (i.e. contain rela- tively few essential nutrients), so limiting such foods has no adverse nutritional consequences. Limiting free sugar intake of individuals to below 10% total energy is widely recommended and WHO has suggested that further reductions may be associated with additional benefits. On the other hand, intrinsic sugars (i.e. those incorpor- ated into the natural structure of foods, like fruits), milk sugars, and starches are not restricted and generally provide the balance of dietary energy not provided by protein, fat, and free sugars. Similar advice has been offered by the Specialist Advisory Committee on Nutrition (SACN) in the United Kingdom in their 2015 report (Carbohydrates and Health). They suggest that population average intakes of total carbohydrate should be around 50% total energy and free sugars around 5% total energy. There has been much discussion regarding the most appropriate carbohydrate-containing foods. Intact fruit and vegetables and minimally processed cereals and grains tend to be rich sources of dietary fibre, essential micronutrients, and some essential fatty acids. The SACN Report suggests that population average intake of fibre should be about 30 g/day, an appreciable increase from current levels. Cooked dried beans, chickpeas, and some whole-grain products are rich sources of dietary fibre and resistant starch, which having largely avoided digestion in the small intestine enter the colon in a largely undigested state. Resistant starch, oligosaccharides, and some com- ponents of dietary fibre (e.g. gum, pectins, mucilages) undergo fer- mentation that leads to the production of fatty acids, which provide a fuel source (via conversion to glucose in the liver) and may also re- duce the risk of colon cancer because of their antiproliferative effects. Other components of dietary fibre remain largely intact and act as stool-bulkers (e.g. cellulose and hemicellulose). Thus, a wide variety of fruits and vegetables, whole grains, and minimally processed cer- eals are particularly appropriate sources of carbohydrate. Some fruits, white rice and hot cooked potato are largely digested in the small intestine and provide an immediate or fairly rapid source of energy, depending upon the speed of digestion. Free or added sugars in jams and manufactured foods (e.g. confectionery prod- ucts) or added by the consumer to food and beverages are also rapid sources of energy, but increase energy density and promote obesity, so that foods rich in them should be restricted by most people. The glycaemic response following the ingestion of a specified amount of carbohydrate in a food, expressed as a percentage of the glycaemic response following a similar amount of glucose (glycaemic index), has been suggested as a useful approach to identifying the most ap- propriate carbohydrate-containing food choices (i.e. those with a low glycaemic index are particularly appropriate). While this is of some value in comparing different varieties of similar foods (e.g. different Table 11.5.6 Nutrient intake goals as recommended by WHO and FAO and in the United Kingdom (unless otherwise stated, the goals are expressed as percentage total energy) WHO/FAOa (2003) Nordicb (2012) Total fat 15–30% 25–40% Saturated fatty acids (SFA) <10% <10% Cis polyunsaturated fatty acids 6–10% 5–10% n-6 PUFA 5–8% c n-3 PUFA 1–2%

> 1%d Cis monounsaturated fatty acids By differencee 10–20% <1% As low as possible Dietary cholesterol (mg/day) <300 mg/day c Total carbohydrate 50–75%g 45–60% Free sugarsf <10% <10% Dietary fibre (NSP) 25 g/dayg 25–35 g/day Protein 10–15% 10–20% Sodium chloride <5 g/day c Potassium c Fruit and vegetables 400 g/day 400 g (5 portions) a Source: WHO (2003). Diet, Nutrition and the Prevention of Chronic Diseases. Report of a joint WHO/FAO Expert

> Consultation, Technical Series Report 916, World Health Organization, Geneva. b Nordic Council of Ministers. Nordic Nutrition Recommendations 2012. Main conclusions of the NNR. Online . Copenhagen: 2013. c No specific recommendation. d Mainly from oily fish. e Total fat (SFA & PFA & TFA). f All monosaccharides and disaccharides added to foods by manufacturer, cook, or consumer, plus sugars naturally present in honey, syrups, and fruit juice. g WHO/FAO Scientific Update on CHO (Nishida et al., 2007).

SECTION 11 Nutrition 1900 types of bread) there are several limitations, including that some low glycaemic index foods, especially those containing fat and sugars, may be very energy dense despite having a low glycaemic index (e.g. ice cream) and that there is considerable inter-and intraindividual variation in glycaemic response to foods. Synthetic forms of dietary fibre or fibre extracted from plant material favourably influence cardiometabolic risk factors but there is as yet no corroborative epi- demiological evidence that they have an effect on clinical outcomes comparable with those convincingly demonstrated for dietary fibre naturally found in foods. Dietary protein Dietary protein, the most fundamental macronutrient, has been largely taken for granted in affluent societies. The mixture of foods in the diets of Britain and similar countries provide dietary protein that contributes around 15% dietary energy across genders, occupa- tions, and socioeconomic levels, against nutrient reference amounts of 10% of energy. Protein-containing foods are also good sources of critical nutrients iron, calcium, and zinc. There is only one main subdivision of dietary proteins: animal source and plant source pro- teins. The constituent amino acid patterns are somewhat nearer to human requirements in meats and dairy products. Vegetarians (who consume dairy products) should have no difficulty to achieving ad- equate intakes. The diet of vegans whose protein intake is derived solely from plant sources requires more careful planning, but they have compensatory health advantages of less obesity and eating less saturated fat. Protein intake can be critical in old people. In Australia and New Zealand, the 2005 recommended dietary intake moves up at age 70 plus from 46 to 57 g per day in women and from 64 to 81 g/d in men. In old people dietary protein appears to be used less efficiently. There is increased splanchnic extraction and declining anabolic response to ingested protein. To help prevent sarcopenia, loss of muscle with ageing, the PROT-AGE international committee of geriatricians rec- ommend protein intake should be 1.0–1.2 g/kg (i.e. 65–78 g/d for a 65 kg woman or man). The usual daily pattern of our protein intake is to eat the bulk of it at one meal (dinner) of the day (with meat, fish, or beans). Most modern breakfasts provide some cereal, coffee, and a little milk, so much less than a third of the daily protein intake. It may be better for muscle maintenance to add a protein source at breakfast. Overall diet The appreciation that a wider range of macronutrients than was pre- viously recommended is appropriate for reducing the risk of many chronic diseases enables the translation of nutrition recommenda- tions into many acceptable dietary patterns which are diverse as those traditional to Mediterranean countries, where intake of unsaturated vegetable oils is relatively high, and to some Asian countries where diets relatively high in carbohydrate are consumed. Although a var- iety of carbohydrate intakes is acceptable, a very low carbohydrate high-fat diet is not included among the acceptable dietary patterns. The suggestion by some nutritionists and other health profes- sionals that populations and individuals at high risk of obesity and type 2 diabetes (or those who have already the disease) should be on very low carbohydrate diets (less than 20% total energy) is based principally on deleterious effects

on lipids and measure of carbo- hydrate metabolism observed when diets high in rapidly digested and absorbed carbohydrates are compared with lower carbohydrate intakes. Such untoward effects do not occur when diets are rich in vegetables, fruit, and minimally processed cereals. It has also been claimed that low carbohydrate diets, which tend to be relatively high in fat and protein, are associated with greater weight loss than Mediterranean-type diets or diets with a higher pro- portion of calories from carbohydrate. This is only true in short term studies. Macronutrient distribution is not an important determinant of long-term weight loss and diets with a higher carbohydrate con- tent may be more likely to facilitate weight maintenance. Not only is there no information about long-term clinical benefit of very low carbohydrate diets which are typically high in fat often saturated fat, there are also no data regarding their safety. Low carbohydrate diets have not been endorsed by expert committees or appropriately qualified government and nongovernment bodies. While appropriate distribution of macronutrients and good food choices might be expected to reduce cardiovascular risk, improve bowel function, and reduce the risk of certain cancers and other dis- eases of the large bowel, the importance of ensuring energy balance cannot be overstated. Obesity and its comorbidities, especially type 2 diabetes, account for a public health problem of enormous mag- nitude throughout the world. Increasing carbohydrate-containing bulking foods rich in dietary fibre at the expense of saturated fat is likely to enhance satiety. Such positive advice, along with the re- commendations to reduce frequent consumption of large portions of all energy-dense foods and sugary drinks, is likely to help reduce excessive energy intake. Increasing energy output by increasing physical activity may contribute to public health measures de- signed to stem the tide of the global obesity epidemic, but to a lesser extent than dietary measures. There is increasing recognition of the need to consider sustainability issues when making nutrition recommendations. Reference intakes for vitamins and minerals Reference nutrient intakes (adequate for most individuals) are pro- vided for vitamins and minerals by official bodies, such as the Institute of Medicine (IOM). They are set at a level of two standard deviations above the average of all individual requirements, so that requirements for the vast majority in the population are assured. Clinical vitamin deficiencies, discussed in detail in Chapter 11.2, are uncommon in affluent societies except in at-risk subgroups within populations. For example, immigrants who have migrated from sunny tropical regions to cloudy high-latitude countries may be at risk of vitamin D defi- ciency; strict vegetarians (who consume no animal or dairy prod- ucts) may become deficient in vitamin B12, and disadvantaged groups (especially the very young, pregnant, and lactating women, and older people) may have generally inadequate intakes. By contrast, inappropriate intakes of certain minerals and other nutrients are fairly common. Many groups are particularly vul- nerable to iron deficiency due to high physiological requirements (infants and toddlers, adolescents, pregnant women), high losses (menstruating women), or poor absorption (older people and those consuming foods high in inhibitors of absorption, such as fibre and tannin in tea). Vegetarians are also at increased risk of iron defi- ciency, even when total intake of iron appears to be adequate, since nonhaem iron from plant foods is less bioavailable than haem iron from animal sources. Bioavailability is enhanced by the consump- tion, at the same time, of foods rich in vitamin C. Iodine and selenium are deficient in soils in various parts of the world. Clinical selenium deficiency has only been reported from

 China, although the consequences of lesser degrees of selenium defi- ciency have yet to be established with certainty, especially in regions where soils are known to be deficient. Endemic iodine deficiency is widespread, especially in the Himalayas and the Andes, and clin- ical deficiency states are largely

avoided by the use of iodized salt and sanitizers containing iodine used by the dairy industry. In New Zealand, where goitre due to iodine deficiency had virtually been eliminated, mild iodine deficiency appears to be reoccurring pos- sibly as a result of reduced use of iodized salt and the introduction by the dairy industry of alternative sanitizers. Young women often have insufficient calcium to help achieve peak bone mass, and older women may have an inadequate intake to help reduce an age-related bone loss. Excessive intakes of salt (sodium chloride), to such an extent that it probably contributes to hypertension and its consequences, are common throughout the world. Targets for reduction may be more important than reference nutrient intakes for sodium. An intake of 100 mmol/day (2.3 g sodium/day, roughly equal to 6 g NaCl), a level currently exceeded in most countries, are considered to be an appro- priate maximum. Reference nutrient intakes need to be reviewed when new research becomes available. In the 1990s, a value of 200 µg/day for folate was widely recommended. It is now acknowledged that intakes of 400 µg/day can appreciably reduce the risk of neural tube defects. Most countries have altered their recommended intake to 400 µg/day. The case for increasing the recommended intakes of other nutrients be- yond established requirements in order to reduce the risk of chronic diseases is less clear-cut. Prospective cohort studies suggested that high intakes of several antioxidant nutrients can reduce cardiovas- cular disease, but these findings have not been replicated in large randomized controlled trials in which these nutrients were given as supplements. It is conceivable that these micronutrients are only protective when consumed as food constituents rather than as sup- plements such that it is not possible to specify optimal intakes in terms of reducing the risk of chronic disease. Much current interest centres around optimum dietary intakes of vitamin D, long known to be critically important in prevention and treatment of rickets and osteomalacia. Many nonbone actions of vitamin D have now been recognized and there is experimental and epidemiological evidence that vitamin D might protect against osteoporosis, some cancers, hypertension, type 1 diabetes, multiple sclerosis, and tuberculosis. Further research including clinical trials are needed before firmly establishing the most appropriate intakes and form in which this vitamin is most appropriately consumed.
Implementing nutrition recommendations Substantial changes in what have become traditional eating habits of many affluent societies are required in order to achieve the ad- vised changes in distribution of macronutrients and recommended intake of all essential micronutrients.
A multipronged approach is necessary if there is to be a real chance of achieving dietary change. At the policymaking and government level there needs to be a ser- ious commitment to enabling the population as a whole to make appropriate food choices. Fatty cuts of meat, high-fat products (e.g. meat pies), and convenience foods (e.g. fish and chips, burgers) are relatively inexpensive and therefore frequently eaten by those of lower socioeconomic status who have the highest rates of coronary heart disease. Policies are required which ensure that more appro- priate food choices are available at reasonable cost. This is not easy to achieve in many Western countries, where farmers may have con- siderable political influence, and subsidies may be available for some high-fat dairy products such as butter and cheese. Governments and intergovernmental agencies also have the re- sponsibility for ensuring that food labels and health claims are ac- curate, interpretable, and likely to facilitate health-promoting food choices, a particularly important issue given the increased con- sumption of packaged food. In countries in transition where poverty (undernutrition) and affluence (overnutrition) coexist, achieving appropriate food choices for the population as a whole presents an even greater challenge. Dietary guidelines are necessary to provide clear directions to individuals and families who wish to aim for a healthy diet pat- tern. These guidelines vary slightly from country to country, but some are almost universal (see Box 11.5.2). Others are less consistent (see Box 11.5.3). The public also need education re- garding

food groups and the nutrients they contain, the inter- pretation of food labels, the meaning of health claims, and the methods of food preparation. The increased use of convenience and packaged food has meant that many people no longer possess basic cooking skills. They also need (and usually want) to know the merits and demerits of obtaining certain essential micronu- trients by taking supplements or fortified food products rather than conventional foods. Doctors are frequently asked to give nutritional advice but may lack the necessary expertise. Dietitians, nutritionists, and appropriately trained practice nurses play an invaluable role in providing the public with practical advice to facilitate changes from the typical Western diet, as well as providing instruction re- garding therapeutic diets for those with diseases requiring spe- cific diet therapy. The enormous potential for dietary change to reduce the effects of a wide range of diseases should encourage physicians to approach the nutritional management of their pa- tients with enthusiasm. Box 11.5.2 Dietary guidelines for which there is almost complete agreement 1 Eat a nutritionally adequate diet composed of a variety of foods. 2 Eat less of foods rich in saturated fat and use mono- and polyunsatur- ated fats instead. 3 Adjust energy balance for body weight control: energy-dense foods high in fat and sugars should be restricted and exercise increased to avoid excess weight gain. 4 Eat more of a variety of vegetables, fruits, and whole grains. 5 Reduce intake of salt and foods rich in salt. 6 Drink alcohol in moderation, if you do drink. Box 11.5.3 Additional dietary guidelines in some countries 1 Recommendation regarding sugar and sugary foods may vary from ‘no increase’ to ‘decrease’. 2 Drink plenty of fluids each day. 3 Make sure you get enough calcium or milk. 4 Eat foods containing iron. 5 Drink fluoridated water. 6 Preserve the nutritive value of food (by good food preparation). 7 Eat three good meals a day.

 FURTHER READING Ascherio A, et al. (1996). Dietary fat and risk of coronary heart dis- ease in men: cohort follow up study in the United States. BMJ, 313, 84–90. Bauer J, et al. (2013). Evidence-based recommendations for optimal dietary protein intake in older people: a position paper from the PROT-AGE Study Group. J Am Med Dir Assoc, 14, 542–59. Bernstein AM, et al. (2010). Major dietary protein sources and risk of coronary heart disease in women. Circulation, 122, 876–83. FAO/WHO Scientific Update on Carbohydrates in Human Nutrition (2007). Eur J Clin Nutr, 61(Suppl 1), S132–S137. Food and Nutrition Board, Institute of Medicine (1998/2011). Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, (1998); and Dietary Reference Intakes for Calcium and Vitamin D (2011). National Academies Press, Washington DC. https://www.nap.edu. Lean M, et al. (2018). Primary care-led weight management for remis- sion of type 2 diabetes (DiRECT): an open-label, cluster-randomised trial. Lancet, 391, 541–51. Liu S, et al. (1999). Whole-grain consumption and risk of coronary heart disease: results from the Nurses’ Health Study. Am J Clin Nutr, 70, 412–19. Mann J, Truswell AS (eds) (2017). Essentials of human nutrition, 5th edition. Oxford University Press, Oxford. Mann J, et al. (2014). Low carbohydrate diets: going against the grain. Lancet, 384, 1479–80. Mann J, et al. (2015). Diverging global trends in heart disease and diabetes: implications for dietary guidelines. Lancet Diabetes Endocrinol, 3, 584–5. Moynihan PJ, Kelly SA (2014). Effect on caries of restricting sugars intake: systematic review to inform WHO guidelines. J Dent Res, 93, 8–18. Nordic Council of Ministers (2012). Nordic Nutrition Recommendations 2012. Main conclusions of the NNR, Copenhagen. https://norden. diva-portal.org/smash/get/diva2:704251/FULLTEXT01.pdf Reynolds, A, Mann, J, Cummings, J, Winter, N, Mete, E, Te Morenga, L (2019). Carbohydrate quality and human health: A series of system- atic reviews and meta-analyses. Lancet. Advance online publication. doi: 10.1016/S0140-6736(18)31809-9. Rimm EB, et al. (1996). Vegetable, fruit, and cereal fiber intake and risk of coronary heart disease among men. JAMA, 275, 447–51. Schwab U, et al. (2014). Effect of the amount and type of dietary fat on cardiometabolic risk factors and risk of developing

type 2 diabetes, cardiovascular diseases, and cancer: a systematic review. Food Nutr Res, 58. doi: 10.3402/fnr.v58.25145. eCollection 2014. Scientific Advisory Committee on Nutrition (SACN) (2015). Carbohydrates and health. The Stationery Office Limited, London. Te Morenga L, Mallard S, Mann J (2013). Dietary sugars and body weight: systematic review and meta-analyses of randomised con- trolled trials and cohort studies. BMJ, 346, e7492. Truswell AS (2010). Cholesterol and beyond: the research on diet and coronary heart disease 1900–2000. Springer, Dordrecht, Heidelberg, London. U.S Department of Agriculture (2015). Scientific Report of the 2015 Dietary Guidelines Advisory Committee. United States Department of Agriculture, Washington, DC. https://health.gov/dietaryguidelines/ 2015-scientific-report/ WHO (2003). Diet, nutrition and the prevention of chronic diseases. WHO Technical Report Series 916. World Health Organization, Geneva.

Revision #1
Created 2026-01-22 16:37:23 UTC by Omar Ayman
Updated 2026-01-22 16:37:23 UTC by Omar Ayman