V

DISEASE-ORIENTATED PREVENTIVE NUTRITION

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Introduction

There is a growing array of health disorders in which nutrition is recognized as being important in the pathogenesis. These are detailed in the accompanying table which refers to: (1) *The relative importance of nutrition* among other aetiological or pathogenic factors; (2) The particular *role of energy balance* (principally to do with energy intake in food on the one hand, and energy expenditure on the other, ie basal energy expenditure, reflecting lean mass; physical activity; and the thermogenic response to food); (3) *Foods of importance* (both adversely and protectively); (4) *Nutrients of importance* (both adversely and protectively); (5) *Non-nutrients of importance* (both adversely and protectively). These last two areas are further developed in Section VI of this book.

In general it can be said that the achievement of a higher plane of energy balance for maintenance of energy stores (principally fat) at preferred levels is conducive to better health (see Section IV). There are probably several reasons for this – eating more avoids precariously low nutrient and non-nutrient intakes, and there are the benefits of exercise on eating behaviour[1], mood[2,3] and physical fitness[4,5]. We discuss below some of the conditions highlighted in Table 1.

Obesity

The problem of obesity is increasing in both developed and developing countries for complex reasons, and its prevention is no less complex. For many, its development may be more to do with inadequate physical activity than with increased energy intake. Fat distribution is an important determinant of the overall risks of obesity[6-8].

Non-food factors include the role cigarette smoking plays in predisposition to the development of abdominal fatness[9].

Genetic factors are important, both in themselves and in relation to determining food intake, insofar as total body fatness is concerned[10] and also for the distribution of fatness[11].

Excessive intake of fatty foods, particularly saturated fat, seems to be of crucial importance in the development of obesity[12-14]. All ingested fat, however, may not be the same insofar as propensity to positive energy balance is concerned. The early work by Atwater (see Lusk[15]) on the energy values of macronutrients did not make a

Table 1. Disease-related preventive nutrition.

Disorder (References)	Importance (0–5) of nutritional factors[a]	Role of energy balance	Foods of importance	Nutrients of importance	Non-nutrients of importance
Obesity 9, 11–14, 18, 21, 27	4	*Protective* Achieve at higher plane	*Adverse* Fatty Alcohol Carbohydrate drinks *Protective* Plant-derived ?Fish Water as beverage	*Adverse* Saturated fat *Protective* Unsaturated fat n-3 FA Fibre	*Adverse* Cigarette smoking *Protective* Possibly oestrogenic components for fat distribution
Macrovascular/ Atherosclerotic Disease 7, 23–28, 35, 36	4 Nutritional factors may operate more through hypertension than lipids[b]				
Coronary		*Adverse* Positive balance *Protective* Achieve at higher plane	*Adverse* Fatty *Protective* Variety Plant-derived Fish ?Modest alcohol		Components of fruits, vegetables & cereals[c]
Peripheral	*Adverse* Positive balance	*Protective* Achieve at higher plane	*Protective* Variety Fish		
Cerebrovascular	Positive balance	Achieve at higher plane	*Adverse* Alcohol *Protective* Plant-derived		
Diabetes and its complications 31, 35, 36, 42					

Insulin-dependent	2	*Adverse* Negative during onset *Protective* Achieve	*Adverse* Fatty (saturated) *Protective* Plant-derived	*Adverse* Saturated fat *Protective* n-3 fatty acids Fibre	*Adverse* Food preservatives *Protective* Physico-chemistry of food lectins
Non-insulin-dependent	4	*Adverse* Positive balance	*Adverse* Fatty (saturated) Fish *Protective* Variety Plant-derived	*Protective* Physico-chemistry of food lectins	
Osteoporosis 21, 45, 48. 139	4 May be more important during adolescence & young adult life, up until peak bone mass, than later[d]	*Adverse* Negative balance (wasting) *Protective* Positive balance (obesity)	*Adverse* Sodium-containing foods of low nutrient density ?Excessive protein Glycaemic *Protective* Foods of high nutrient density Calcium-containing Alcohol	*Adverse* High protein intake	*Adverse* Caffeine Antacids Fibre *Protective* ?Plant food oestrogenic activity or with factors like Boron which increase endogenous oestrogen
Peptic ulcer 54–56, 58–60	2–3 Under revision	*Adverse* ? *Protective* ?	*Adverse* Containing n-3 and/or n-6 fatty acids *Protective* Affecting the *Helicobactor pylori* status	*Adverse* ? *Protective* Linoleic acid Arachidonic acid Eicosapentanoic acid	
Cancer					
Colorectal 61–64	4[e]	*Adverse* ? *Protective* ?	*Adverse* Salted/smoked *Protective* Fresh fruit/vegetables	*Protective* Vitamin C	*Adverse* Nitrites Salt
Gastric 65, 67, 68, 70, 131	4	*Adverse* ? *Protective* ?	*Adverse* Salted/smoked *Protective* Fresh fruit/vegetables	*Protective* Vitamin C	*Adverse* Nitrites Salt

Breast 51, 71–73, 75, 79, 140, 141	3–4	*Adverse* Positive balance	*Adverse* Fatty Alcohol *Protective* Soya High P/S ratio	*Protective* β-carotene Fibre	*Adverse* Methyl-xanthines *Protective* Probably phyto-oestrogens
Bronchogenic 66, 79–81	3	*Adverse* ? *Protective* ?	*Protective* Green leafy & yellow vegetables	*Protective* β-carotene	*Adverse* ? *Protective* ?
Pancreatic 82, 83	4[f]	*Adverse* ? *Protective* ?	*Adverse* ?Coffee	*Adverse* ?Caffeine ?Trypsin inhibitors *Protective* ?	
Endometrium 85, 88–90	2–3	*Adverse* Positive balance (obesity) *Protective* ?	*Adverse* ?	*Adverse* ? *Protective* ?	
Ovary	2	*Adverse* Positive balance (obesity)	*Adverse* Coffee	*Adverse* ?Saturated fat	
Prostrate 66, 91, 92	4	*Adverse* Positive balance (obesity)	*Protective* Green leafy & yellow vegetables	*Adverse* Saturated fat ?β-carotene	?phyto-oestrogens
Skin					
Squamous cell carcinoma	?	*Adverse* ? *Protective* ?	*Adverse* ? *Protective* Vit A and/or carotenoid containing	*Adverse* ? *Protective* ?	
Inflammatory Disease:					

Psoriasis 97, 98	1–2	*Adverse* ? *Protective* ?	*Adverse* ? *Protective* Containing n-3 fatty acids	*Adverse* ? *Protective* ?	
Rheumatoid arthritis	1–2	*Protective* fasting	*Protective* Containing n-3 fatty acid Vegetarian diet		
Osteoarthritis	3	Positive balance (obesity)			
Other Metabolic Disease:					
Gout 103, 104	3[g]	*Adverse* Positive balance (obesity)	*Adverse* Alcohol High-purine-containing	*Adverse* ? *Protective* Water	
Expression of menopause 21	1–2	*Adverse* ? *Protective* ?	*Protective* ?Plant-derived	*Protective* Oestrogenic activity	
Hyperlipidaemia	3–5[h]	*Adverse* Positive balance (obesity)	*Adverse* Saturated fat especially 14:0 Alcohol Coffee *Protective* Plant-derived Fish	*Protective* Soy protein Soluable Fibre	*Protective* Allicin Anthocyamin Sopoanins etc
Hypertension 119–124, 126	4	*Adverse* Positive balance (obesity)	*Adverse* Alcohol Sodium-containing *Protective* Vegetarian orientation ?Fish but probably interactive with Na/K ratio		

Micronutrient deficiency 21, 129, 130, 132			
Vitamin	5	*Protective* Higher plane of energy nutrition	*Protective* Variety Nutrient-dense
Element (major & minor)	5	*Protective* Higher plane of energy nutrition	*Adverse* Grown in poor soils *Protective* Variety Nutrient-dense
Micronutrient Excess:			
Food supply 128, 132	5		*Adverse* Foods grown in regions with excess in soils (eg Se) Restricted variety (eg dependence on marine liver)
Self-medication	5 A matter of choice		
Immunodeficiency			
HIV expression 29, 53	Presently unknown but likely		

[a] The role of nutritional factors amongst other pathogenic factors of obese expression is rated from 0 (Norole) to 5 – (extremely important)
[b] Smoking and glycaemia are very important
[c] eg, ajone, llicin, salicylates, saponins, athocyanins
[d] Smoking is an adverse, and physical a favourable factor
[e] Genetic disposition clearly important
[f] At present under debate
[g] Genetic predisposition important
[h] Dependent on genetic basis

distinction between one type of fat and another according to chain length or degree of unsaturation. There is some recent work which suggests that polyunsaturated fat may be less likely to contribute to the development of obesity[16]. Experimental evidence suggests that fish-derived fat of the Ω-3 kind may be less likely to induce obesity[17,18]. The energy density of the food is probably also important, but this is related to the fat content and also to consumption of alcohol, which has the next highest energy value of the macronutrients after fat. Components of food which reduce energy density must also be important, including water and dietary fibre, although it should be noted that those components of dietary fibre which undergo fermentation in the large bowel do have an energy value, probably of the order of 2.0kcal (10kJ) per gram[19].

The pattern of eating in obesity has also been much discussed. It may be that snacking, provided it is not on fatty foods, may be relatively protective[20].

In the light of evidence that food has compounds with oestrogenic activity[21], that there are gender differences in the distribution of fat[6-8], and that sex hormone profiles are determinants of fat distribution[22], it seems likely that an intake of oestrogenic foods could be protective against abdominal fatness.

Macrovascular disease (atherosclerotic vascular disease)

Several studies suggest that a higher plane of energy balance is protective against coronary heart disease[7,23-26].

Nutritional factors can operate to increase the risk of macrovascular disease by various pathways and therefore preventive measures might take advantage of any one or more of these pathways. These include hyperlipidaemia[27], hypertension[28], increased platelet aggregation[29], impaired glucose tolerance and frank diabetes[30,31], cardiac membrane composition and stability[32] and cardiac substrate utilization[33]. Obesity, and in particular abdominal obesity, may well operate through insulin resistance, hyperlipidaemia and hypertension[34].

A wide variety of food intake is associated with less macrovascular disease[35]. Intake of fish may have a beneficial effect on the arterial wall[36].

The relative importance of nutritional factors may be different in the expression, and therefore in the prevention, of macrovascular disease in different territories: coronary, peripheral and cerebrovascular, as outlined in the Table.

Diabetes and impaired glucose tolerance

In considering the prevention of diabetes, one should also consider the prevention of impaired glucose tolerance, which is more prevalent in the community and is a risk factor for macrovascular disease. Both of these problems are increasing in developed and developing countries[37,38].

The prevention of the major form of diabetes, non-insulin-dependent diabetes, appears to depend mainly on a reduction in abdominal obesity[30]. However, this must involve attention to physical activity as well as to food intake, as discussed above in relation to obesity. There may also be environmental factors which are yet to be elucidated. Genetic predisposition is also important, but so far the genetic basis has not been defined. Indeed, one of the provocative aspects of this area of preventive nutrition is that populations like the Chinese, previously thought to be somewhat resistant to the development of diabetes, are undergoing major increases in

prevalence in countries like Mauritius[38], Malaysia[39] and Australia[40]. Although there was much debate for many years about sugar intake as the basis for diabetes of the insulin-dependent and non-insulin-dependent kinds, this is now thought to be unlikely[41]. Of considerable interest and importance is the suggestion that certain preserved foods may account for substantial increases in the incidence of insulin-dependent diabetes in countries like Sweden[42].

Osteoporosis

The 'peak bone mass' in men and women is achieved by about 25–30 years, maintained relatively constantly until 35–45 years, and then lost at a constant rate of 0.3–0.5% per year. For 8–10 years in the peri-menopausal period, bone loss is accelerated to a rate of 2–5% per year in Caucasian women[43,44]. Many factors, both nutrient and non-nutrient, may contribute to the development of osteoporosis.

Reduced body fat is a strong risk factor for osteoporosis and conversely, obesity is strongly protective against bone loss. These body habitus factors appear to be mediated via peripheral oestrogen metabolism.

The degree of peak bone mass achieved is a major factor modifying the risk of the development of osteoporosis. The more bone mass is available before age-related osteoporosis develops, the less likely it is that it will decrease to a level at which fracture will occur[44,45]. An adequate intake of dietary calcium, sufficient to achieve a positive calcium balance, is probably the most important factor contributing to the level of peak bone mass developed[45]. Calcium supplementation alone in post-menopausal women probably has little effect on the rate of bone loss; even in combination with hormone replacement treatment, there is uncertainty about its value.

Dietary phosphorus may promote intestinal calcium loss, which is countered by decreased urinary excretion in normal subjects. No studies have examined the effect of high intake of phosphorus on bone loss in humans. However, chronic use of phosphate-binding antacids can cause skeletal demineralization[46].

Vitamin D deficiency results in osteomalacia. Calcitriol treatment may improve calcium absorption in osteoporotic women[47].

High dietary intake of protein, frequent in Western countries, may contribute to some of the negative calcium balance seen in perimenopausal women[48].

Dietary fibre may chelate calcium and other minerals in the intestinal tract, thus reducing absorption[49]. In cultures where dietary fibre intake is high, this process may increase the risk of osteoporosis[45].

In areas where fluoride concentration in drinking water is high, a lower prevalence of osteoporosis is found[50]. However, treatment of established osteoporosis with fluoride supplements does not appear to alter fracture rates[45].

Alcohol abusers have reduced bone mass and the risk of falling is increased so the risk of sustaining fractures is also markedly increased in this group[45].

Intake of plant-derived oestrogens may be important. Plant-derived foods such as soy-beans have oestrogenic effects that may contribute to reduction of peri-menopausal bone loss[21].

Non-nutrient factors which are important in contributing to the risk of osteoporosis include cigarette smoking, lack of physical activity and treatment with steroids, thyroid hormones and antacids[45].

Peptic ulcer disease

Peptic ulcer disease (gastric and duodenal ulcer) is not only a source of morbidity through pain but also presents the risk of upper gastrointestinal bleeding and of perforation. Until 20 years ago doctors, desperate to offer therapy, implemented dietary measures without a scientific basis. Such measures included avoidance of many foods and bland diets such as milk and white bread, later shown to be unhelpful. This unfortunate history led many authorities to dismiss the role of diet in the prevention and management of peptic ulcer disease as little more than folklore.

With the understanding of 'no acid, no ulcer', the introduction of H_2 antagonists in the 1970s allowed effective therapy against active ulceration. However, the natural course of disease remained unaltered. Relapse rates of 50–80% in the first post-treatment year were commonly reported. Bleeding and perforation, the most common complications of peptic ulcer disease, have not changed in incidence despite the advent of H_2 antagonists[51] and these complications remain common. With better understanding of the pathogenesis of peptic ulcer disease, the new dictum is 'if acid, why ulcer?'. There are cultural and geographical differences[52], as well as changes in disease prevalence in recent years[53], which underscore the importance of environmental factors; diet and bacterial infestation (*Helicobacter pylori*) are among the candidates. These factors may allow not only better control of ulcer relapse and complications, but also the possibility of primary prevention.

Diet and peptic ulcer disease

As the methods of recording and interpretation of dietary information have advanced in recent years, so too has the understanding of the influence of diet on many diseases. There are numerous mechanisms by which diet may influence peptic ulcer disease: buffering capacity, provision of protective factors, enhancement of mucus protection, effects on bile outflow, alteration of the prostaglandin-leukotriene balance and altering the pathogenicity of *H. pylori*.

One of the most promising aspect concerns the protective effect of essential fatty acids. Hollander and Tarnawski[54] found a 200% increase in linoleic acid ingestion by the United States' population over the past 50 years which coincided with a five-fold decrease in ulcer-related mortality and surgical intervention. Adipose tissue levels of linoleic acid have also been shown to be significantly lower in patients with peptic ulcer disease and non-ulcer dyspepsia[55]. Laboratory studies have shown that the administration of linoleic acid, arachidonic acid and eicosapentaenoic acid (active principles in fish oil) to rats lessens the severity of substance-induced gastric lesions[56]. These data suggest that increased consumption of polyunsaturated fatty acids and fish oils may be beneficial not only in the primary prevention of rheumatoid arthritis and cardiovascular diseases, but also in peptic ulcer disease.

Belicobacter pylori *and peptic ulcer disease*

The possibility of prevention in peptic ulcer disease has been strengthened by the discovery of *H. pylori*. Therapeutic trials (eg bismuth compounds and antibiotics *versus* H_2 antagonists) have shown a dramatic reduction in ulcer relapse rates[57] with eradication of *H. pylori*. The interaction between diet and the pathogenicity of *H. pylori* is not known and may prove to be an important link with ulcer pathogenesis.

Already, there is evidence to suggest an association between food intake pattern and *H. pylori* prevalence[58,59]: both the Chinese and the Japanese share similar food intake patterns and have a higher *H. pylori* prevalence than is found in Europids[60].

With further research into the influence of diet on *H. pylori* and ulcer diathesis, progress in the management of peptic ulcer disease and alteration of its natural course may be expected.

Cancer and diet

Colorectal cancer

The link between dietary factors and colorectal cancer has been investigated since 1933, when one of the earliest studies by Stocks and Karn was published. In this epidemiological study, vegetables, breads and milk were negatively correlated with colorectal cancer[61]. Since then, the dietary risks for colorectal cancer have been investigated in numerous studies. Much of the work remains inconsistent and awaits better definition.

Fat intake and risk of colonic cancer was examined in a recent review paper by Rogers and Longnecker[61]. They concluded that it had no consistent role in either promotion or inhibition of carcinogenesis.

Similarly, the role of dietary fibre at present remains controversial. For example, when populations from countries which have a low intake of fibre are examined, there is a poor correlation with the incidence of colonic cancer[62].

Some workers have found an inverse relationship between the intake of β-carotene and colorectal cancer[63], while others have not found a relationship[64].

Gastric cancer

The incidence of gastric cancer is high in Japan and parts of Asia, whereas in Europe and North America, the incidence is low. In Japan, there has been a recent decline in incidence[65]. The geographical differences and changing prevalence appear to be related to several dietary factors.

A positive association between gastric cancer and the consumption of salted and smoked fish and pickled vegetables has been shown in several case-control studies[66-68]. These foods contain both salt and high levels of nitrates and nitrites. Further investigation suggests an association between gastric cancer and nitrite consumption[68]. One hypothesis is that a high salt intake may cause chronic gastric irritation, thus facilitating carcinogenic transformation in the mucosa when exposed to nitrites or nitrosamines which are formed in the reduction of nitrates to nitrites[69].

A protective effect of fresh fruit and vegetables has been seen in several studies[65,68]. This is consistent with the ability of vitamin C, present in high levels in fresh produce, to inhibit the formation of carcinogenic nitrosamine compounds[70].

Breast cancer

There is evidence for dietary effects on breast cancer risk, although the precise nature of these effects is still unclear.

Obesity itself carries an increased risk for breast cancer in post-menopausal women[71], though results of studies of the association between fat consumption and breast cancer have been contradictory. A recent[71,72] prospective study of women in

Finland aged 29–69 years identified a positive relationship between *low* total energy intake and increased risk for breast cancer, which was a stronger association than that for dietary fat intake[73].

Consumption of alcohol[74] and foods containing methylxanthines[75] appears to confer an increased risk.

Bronchogenic cancer

Lung cancer is the leading cause of death from cancer among men in most developed countries[76]. Cigarette smoking is the most important causal factor[77], with occupational exposures constituting a synergistic risk factor in males[78].

Studies of dietary factors have controlled for cigarette smoking, but there has been little research examining possible interactions between tobacco and dietary factors. Dietary vitamin A has been inversely associated with lung cancer[79,80]. An inverse association between lung cancer and dietary intake of green/yellow vegetables has also been reported[66]. β-Carotene rather than retinol appears to be the important component[81].

Pancreas cancer

There is some controversial work which suggests that excessive coffee consumption, as a source of caffeine, in conjunction with cigarette smoking may increase the risk of cancer of the pancreas[82,83]. Another suggestion is that trypsin inhibitors in food may cause protracted overstimulation of exocrine pancreatic function and therefore predispose the pancreas to neoplastic change[83]. Given the real increase in incidence of pancreatic cancer which is apparently taking palce, at least in developed countries where it is now the fifth leading cause of premature death from cancer[84], the issue of prevention of this disorder by nutritional means is assuming greater importance.

Endometrial and ovarian cancer

A well established risk for endometrial cancer is the use of high dose unopposed oestrogen therapy. Diabetes mellitus[85], obesity[85] and hypertension, with their nutritional implications, have been associated with endometrial cancer. Upper body fat localization (android obesity) is an additional risk factor[86]. No possible direct dietary risk factors have been identified to date.

Development of ovarian cancer is inversely related to oral contraceptive use[87]. There are conflicting studies on the relationship of coffee drinking[88,89] and saturated fat intake[89,90] as dietary risk factors for ovarian cancer.

Prostate cancer

Dietary fat, β-carotene and cadmium have been related to prostate cancer.

Epidemiological studies show positive correlations between prostate cancer mortality and *per capita* fat intake[91]. This correlation is also found in studies of dietary fat intake and prostatic cancer[92].

β-carotene may be a risk factor for prostate cancer, unlike other types of cancer, especially in males over the age of 70 years[92]. Hirayama, however, showed that green and yellow vegetables were protective in a 10 year prospective study of Japanese men[66].

The effect of dietary cadmium is not well established, with conflicting reports in the literature.

Skin cancer

There is no definite association between diet and pigmented skin malignancies.

Vitamins A and E and selenium have received some interest. Lower mean carotene intakes were found in a small study of patients with melanoma[93]. In another study, no differences in vitamin A, vitamin E or selenium levels were found between melanoma patients and controls[94].

Data linking dietary polyunsaturated fat intake and melanoma were inconclusive[95]. One study reported a small increase in risk associated with alcohol intake[96].

Inflammatory disease

Psoriasis

There is evidence that psoriasis is associated with some local changes in lipid metabolism. Activity of both phospholipase A_2 and phospholipase C is increased in affected skin[97]. In addition, studies of plasma essential fatty acids in patients with psoriasis show reduced levels of linoleic acid and arachidonic acid[98].

Supplementation with fish oils has been studied on the basis that the patogenesis of psoriasis is related to the overproduction of pro-inflammatory metabolites from arachidonic acid. Despite reduction in the levels of eicosanoids, the therapeutic results have generally been poor[97].

Rheumatoid arthritis

Fasting is an effective short-term treatment for rheumatoid arthritis, usually followed by relapse on the re-introduction of food[99]. A vegetarian diet has been shown in a recent controlled study to have beneficial and sustained effects on established rheumatoid arthritis[100].

Dietary fish oil supplementation in addition to conventional therapy for rheumatoid arthritis has been shown in controlled trials to have a definite anti-inflammatory effect[101,102]. This effect appears to be mediated through eicosapentaenoic acid and is more potent when dietary n-6 fatty acid intake is reduced[103].

Osteoarthritis

Most would regard obesity as a predisposing factor towards osteoarthritis in weight-bearing joints, such as hips and knees. However, good studies on this are few and far between. In those who develop premature osteoarthritis weight-bearing is likely to have been less of a problem. However, like virtually all disease processes, it will have a multi-factorial basis and, so far as nutritional factors are important at all, they are likely to vary in their importance in different individuals.

Metabolic disease

Gout

While the mainstay of treatment of acute gout is pharmacological, dietary measures

aimed at the reducing serum urate may be helpful both in the acute attack and in prevention of further episodes.

Such measures include energy restriction and body weight reduction (since urate production appears to vary directly with body weight[104]), restriction of purine-rich foods (which are metabolised to urate)[103], avoidance of alcohol and avoidance of fasting (since metabolic acidosis may trigger acute attacks[104a]) and increasing renal clearance of urates by increasing water intake.

Menopause

Determinants of expression of the menopause are largely unknown, Descriptions of oestrogenic activity in some plant-derived foods (*phyto-oestrogens*) and their effects in postmenopausal women[21] suggest a role for nutrient factors in the menopause. In some food cultures, up to 50% of the energy intake is derived from foods which contain oestrogenic compounds[21].

Lipid disorders

Obesity is an important risk factor for hyperlipidaemia, influencing triglyceride levels predominantly via increased VLDL synthesis[105].

Dietary saturated fatty acid and cholesterol intake are major determinants of serum lipid levels. Saturated fats raise the serum cholesterol and low density lipoprotein and polyunsaturated and monounsaturated fats cause them to decrease[106,107]. Dietary cholesterol has a less potent influence on serum lipid levels[108].

In epidemiological studies, fish intake is protective for coronary heart disease. In addition, intake of fish with a high ω-3 fatty acid content has a beneficial effect on serum lipids[109,110].

Substitution of soya bean protein for animal protein in the diet results in a fall in serum cholesterol and LDL levels[111]. The mechanism of this effect is unclear, but may explain why in some Asian populations, where soya bean is the main source of protein, lipid levels are lower than would be expected from calculations based solely on the fat and cholesterol content of the diet.

Soluble fibre such as guar gum, pectin and oat gum has a hypocholesteraemic effect when given in large quantities. Insoluble fibre has little effect[112].

Alcohol consumed in moderate amounts on a regular basis leads to an appreciable increase in high density lipoproteins (HDL)[113]. In some individuals, triglycerides can be raised in response to alcohol.

Ingestion of large quantities of coffee, especially boiled coffee as prepared in Scandinavian countries, is associated with a rise in serum cholesterol[114].

Recent work has investigated the lipid lowering effects in animal models of plant derived compounds such as anthocyanins[115-117] and saponins[118].

Hypertension

The association between hypertension and obesity is well established[119]. In epidemiological studies, increased body weight is related to raised arterial blood pressure in both normotensive and hypertensive populations. The Framingham study showed that relative body weight and body weight change over time were directly related to the subsequent rate of development of hypertension[120]. Similarly,

reduction in body weight is well correlated with a fall in blood pressure in hypertensive subjects[121].

Vegetarian diets have been associated with a reduction in blood pressure. This effect appears to be greater on systolic than on diastolic pressure[122].

The role of dietary fat is less clear. Feeding of fish oil and diets low in saturated fatty acids has resulted in a reduction in blood pressure, but many studies have failed to account for other dietary components in their analysis[123].

Alcohol, even in small quantities, has a pressor effect[124]. There is a higher prevalence of hypertension when more than 60 to 80 g of ethanol per day is consumed.

Caffeine has a short term pressor effect, but there is no long term association with hypertension, even where large quantities are consumed[125].

Sodium intake has received much attention. While there is a heterogeneous response to sodium restriction in the diet and a particularly *salt-sensitive* subpopulation[126], reduction in the dosage of drug treatment has been shown following moderate sodium restriction[127].

Micronutrient deficiencies

Deficiencies and excesses of vitamins, minerals and essential fatty acids from diet or disease-related malabsorption are prime examples of nutrition-based disease.

Prevention of deficiencies depends on an adequate food supply. In developed countries this may also include maintaining a high level of physical activity in order to ensure that enough food can be eaten[128]. Further protection is afforded by a diet that is derived from a wide variety of sources. Attention to agriculture is important[129,130]. On a worldwide basis, many nutritional deficiencies (for example iodine deficiency) relate to inadequate supply of elements in local soils[131].

Micronutrient excess may be seen in geographical regions where there is an overabundance of some elements such as selenium in the soil[130].

In developed countries self-medication, eating disorders and alternative practitioner prescription are commoner causes of excessive intake of micronutrients[132].

Immune deficiency

Acquired immune deficiency syndrome (AIDS) resulting from infection with human immunodeficiency virus (HIV) is one disease example of the interaction between nutrition and the immune system.

The interaction between nutritional status and increased disease susceptibility is well described[133]. Many of the immunological abnormalities seen in HIV infection are similar to those seen in malnutrition and single nutrient deficiencies[134], suggesting a possible role for nutritional status in acquisition of HIV infection itself, as well as influencing progression of the disease and susceptibility to opportunistic infection[134,135].

Assessment of nutritional status and nutritional diagnosis

Assessment of nutritional status and nutritional diagnosis are dealt with in depth in other publications[136], but overviews are provided here for convenience. Diagnosis is a skill among health care professionals for which medical practitioners are especially trained.

Food intake assessment

Food intake assessment is dealt with to some extent in Section III. The usual ways of obtaining information are (1) the use of targeted questions, (2) a history of usual food intake across the day, and (3) a food diary, usually for one week to include work and non-work days. As one proceeds to obtain such information, opportunities for prevention can be identified and explored.

Body composition assessment

Body composition assessment is often poorly understood by the public and by health care professionals alike, due to the limitations of weight measurement, and even of weight-height assessments. A suitable approach is as follows[8,137]. Body composition measurements quantify body fat and fat-free compartments. More sophisticated techniques can further quantify subcategories of the fat-free compartment, ie protein content, skeletal mass, and total body water. In addition to the measurement of body fat content, the distribution of fat can be defined by some of these techniques.

Techniques may be classified as *portable*, suitable for field or office practice or *non-portable*, suitable for institutional use (see Table 2 and Appendix 1).

Table 2. Techniques for measurement of body composition.

	Technique	
	Portable	Non-portable
Fat mass	Anthropometry	Whole-body densitometry
	Bioelectrical impedance	DEXA
		CT, MRI scan
Fat-free mass	Anthropometry (arm muscle circumference)	IVNAA nitrogen
	Bioelectrical impedance	Total body K
	DEXA	
	D_2O dilution)	D_2O dilution

Fat mass

Portable techniques

ANTHROPOMETRY

Body mass index (BMI) is calculated from the measurement of weight/height^2. BMI has a good correlation with body fatness and correlates well with morbidity and mortality in the obese individual.

Trunk circumferences define fat distribution. A waist:hip ratio >0.95 (males) and >0.85 (females) is consistent with abdominal obesity.

Skinfold thicknesses make an assumption that subcutaneous fat measurements represent total body fat. Durnin & Womersley[138] developed a regression equation using four skinfolds (biceps, triceps, subscapular and suprailiac), gender, and age to derive body density and hence subcutaneous fat mass. Equations have been developed using multiple or single skinfold sites.

BIOELECTRICAL IMPEDANCE

Application of a constant low level alternating current to the body can be used to determine total body water and, by regression analysis from other techniques, to determine fat mass and fat-free mass. Use of this method has been validated in healthy populations, but in disease states such as renal failure, which may alter the compartmentation of total body water, the technique is less reproducible.

Non-portable techniques

WHOLE BODY DENSITOMETRY

Whole body densitometry is the gold standard for the measurement of body fat. Underwater weighing is used on the basis that the volume of an object submerged in water is equal to the volume of water that the object displaced. The mass of the object in air and in water is then converted to a total body density. Body fat mass can then be calculated using one of the equations describing the relationship between fat density and body density.

DUAL ENERGY X-RAY ABSORPTIOMETRY

Dual energy X-ray absorptiometry (DEXA) is a recent addition to the body composition analysis field. While its primary use is in measurement of bone mineral content (see below), it has the experimental capacity to measure fat mass and fat distribution.

OTHER METHODS

Other techniques such as CT scanning and magnetic resonance imaging have been used in some centres to measure body composition.

Fat-free mass

Portable techniques

ANTHROPOMETRY

Arm muscle circumference can be derived from the mid-arm circumference and the triceps skinfold, and is a good indication of body protein stores.

Arm muscle circumference = mid-upper arm circumference – (π × triceps skinfold)

BIOELECTRICAL IMPEDANCE

See comments in Fat mass section above.

Non-portable techniques

IN-VIVO NEUTRON ACTIVATION ANALYSIS (IVNAA)

While other elements have been analysed, in clinical practice this technique measures only nitrogen, from which total body protein is calculated.

Neutron activation analysis involves the delivery of a beam of neutrons to the subject. These neutrons are captured by the target atoms in the body, creating unstable isotopes: in the case of protein, the isotope formed is ^{15}nitrogen. The isotope reverts to a stable state by the emission of γ rays of a characteristic energy, which can then be detected by the use of standard γ-spectrographic analysis. This method, targeting nitrogen, allows: (1) the determination of total body nitrogen and therefore total body protein, the principal nitrogen-containing component of the body; and (2) the indirect determination of skeletal muscle mass.

TOTAL BODY POTASSIUM

The naturally occurring radioactive isotope of potassium, ^{40}K, is present in a known, constant, very low percentage of total potassium. Since, body potassium is essentially intracellular and not present in stored fat, measurement of ^{40}K allows an estimation of body cell mass.

DUAL ENERGY X-RAY ABSORPTIOMETRY

See comments in Fat mass section above.

DEUTERIUM DILUTION

As water is not present in stored triglyceride and occupies a relatively fixed fraction (72–72%) of the fat-free (or lean body) mass, estimation of the total body water can be used to derive lean body mass. Assuming a two compartment model, fat can be calculated by difference between weight and lean mass.

The technique involves the administration of a known quantity of deuterium (D_20), an equilibration period, and a sampling period. It assumes that D_20 has the same distribution volume as water and is exchanged by the body in a manner similar to water. Sampling can be from either serum or saliva and while the analytical equipment is only suited to a laboratory, the technique allows the collection of specimens in the field for later analysis.

The process of nutritional diagnosis

As in other areas of medical practice, the diagnosis of problems related to nutrition requires skills. Eliciting a history which includes assessment of food intake and the ability to define and measure physical nutritional status are discussed above and are skills that are adapted from routine medical assessments.

Further considerations for a nutritional diagnosis are as follows. The relative importance of food and nutrient intake in relation to other components of diagnosis should be considered. Factors to consider include: the person's genetic predisposition to an illness; other lifestyle aspects such as physical activity, alcohol consumption, cigarette smoking and stress; the presence of other disease processes and medication; and the possible interaction of all these factors. Nutritional assessments should thus be made in the context of overall clinical assessment.

Functional outcome should be borne in mind in formulating a diagnosis and management plan. Common situations where nutrition plays an important role require the development of personal 'clinical nutrition repertoires'. Such situations include:

- Premature macrovascular disease
- Osteoporosis
- Obesity
- Eating disorders
- Diabetes mellitus
- Use of nutrient and herbal remedies.

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APPENDIX 1

Body Composition Reference Values*

	Normal Range	
Indices of Body Composition	Female	Male
Body mass index (BMI)[a]	20–25	20–25
Waist: hip ratio	<0.85	<0.95
Relative fat mass[b]	20–30%	15–25%
Nitrogen Index (IVNAA)[c]	1.0	1.0

* based on regression analysis of normal population factors of height, arm span and gender (Stroud et al[143]).
[a] BMI <18.5 is a robust indication of chronic energy under-nutrition (see James[142])
[b] Measured by anthropometry or bioelectrical impedance
[c] Nitrogen Index $\frac{\text{Measured total body protein (IVNAA)}}{\text{Calculated total body protein}}$

APPENDIX 2

Recommended daily intakes: macronutrients

Guideline for active young individuals	Man (~70kg)	Woman ~58kg)
Energy		
Kilojoules	11 600	8 400
Kcal	2800	2 400
Protein (grams)	70	60
Carbohydrate (grams)	400	280
Fat (grams)	80	60
Cholesterol (milligrams)	300–400	300–400

Adapted from Wahlqvist ML Collins L.[44]

APPENDIX 3

Recommended daily intakes: micronutrients

GROUP	AGE (years)	WEIGHT (kg)	ENERGY kJ	ENERGY Kcal	PROTEIN (g)	VITAMIN A (μg)	VITAMIN B-1 (mg)	VITAMIN B-2 (mg)	VITAMIN B-6 (mg)	VITAMIN B-12 (μg)	VITAMIN C (mg)	VITAMIN E (mg)
Men	19-64	70	11600-8800	2800-2100	70	750	1.1	1.7	1.3-1.9	2.0	30	10.0
	65+	70	8800	2100	70	750	0.9	1.3	1.0-1.5	2.0	30	10.0
Women	19-54	58	8400-7500	2000-1800	58	750	0.8	1.2	0.9-1.4	2.0	30	7.0
	55+	58	6400	1500	58	750	0.7	1.0	0.8-1.1	2.0	30	7.0
Pregnant		70 (2nd & 3rd trimesters)	9000-8200 (2nd & 3rd trimesters)	2150-1950 (2nd & 3rd trimesters)	66 (2nd & 3rd trimesters)	750	1.0	1.5	1.0-1.5	3.0	60 (2nd & 3rd trimesters)	7.0
Lactating		58	10900-10100	2600-2400	78	1200	1.2	2.0	1.6-2.2	3.5	60	9.5
Infants	0-½											
– breast-fed		—	—	—		425	0.15	0.4	0.25	0.3	25	2.5
– formula-fed		—	—	—		425	0.25	0.4	0.25	0.3	25	4.0
	½-1	—	—	—	2-3	300	0.35	0.6	0.45	0.7	30	4.0
Children	1-3	13	5400	1300	20-39	300	0.5	0.8	0.6-0.9	1.0	30	5.0
	4-7	18-19			25-51	350	0.7	1.1	0.8-1.3	1.5	30	6.0
Boys			7200	1700		500	0.9	1.4	1.1-1.6	1.5	30	8.0
	8-11	28	9200	2200	37-66	725	1.2	1.8	1.4-2.1	2.0	40	10.5
	12-15	41	12200	2900	51-87	750	1.2	1.9	1.5-2.2	2.0	50	11.0
	16-18	61	12600	3000	67-90							
Girls	8-11	27	9200	2200	37-66	500	0.8	1.3	1.0-1.5	1.5	30	8.0
	12-15	42	10400	2500	52-75	750	1.0	1.6	1.2-1.8	2.0	40	9.0
	16-18	55	9200	2200	60-66	750	0.9	1.4	1.1-1.6	2.0	50	8.0

GROUP	AGE (years)	FOLACIN (μg)	NIACIN (mg)	CALCIUM (mg)	IODINE (μg)	IRON (mg)	MAGNESIUM (mg)	POTASSIUM (mg)	SELENIUM (μg)	SODIUM (mg)	ZINC (mg)
Men	19-64	200	18-20	800	150	7	320	1950-5460	85	920-2300	12-16
	65+	200	14-17	800	150	7	320	1950-5460	85	920-2300	12-16
Women	19-54	200	12-14	800	120	12-16	270	1950-5460	70	920-2300	12-16
	55+	200	10-12	1000	120	5-7	270	1950-5460	70	920-2300	12-16
Pregnant		400	14-16	1100 (3rd trimester)	150	22-36 (2nd & 3rd trimesters)	300	1950-5460	80	920-2300	16-21
Lactating		350	17-19	1200	200	12-16	340	2540-5460	85	920-2300	18-22
Infants	0-½										
– breast-fed		50	4	300	50	0.5	40	390-580	10	140-280	3-6
– formula-fed		50	4	500	50	3.0	40	390-580	10	140-280	3-6
	½-1	75	7	550	60	9	60	470-1370	15	320-580	4.5-6
Children	1-3	100	9-10	700	70	6-8	80	980-2730	25	320-1150	4.5-6
	4-7	100	11-13	800	90	6-8	110	1560-3900	30	460-1730	6-9
Boys	8-11	150	14-16	800	120	6-8	180	1950-5460	50	600-2300	9-14
	12-15	200	19-21	1200	150	10-13	260	1950-5460	85	920-2300	12-18
	16-18	200	20-22	1000	150	10-13	320	1950-5460	85	920-2300	12-18
Girls	8-11	150	14-16	900	120	6-8	160	1950-5460	50	600-2300	9-14
	12-15	200	17-19	1000	120	10-13	270	1950-5460	70	920-2300	12-18
	16-18	200	15-17	800	120	10-13	270	1950-5460	70	920-2300	12-18

Adapted from National Health & Medical Research Council. Recommended Dietary Intakes for Use in Australia, 1987[145]