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OXFORD HANDBOOK OF NUTRITION AND DIETETICS

EDITED BY
Joan Webster-Gandy | Angela Madden | Michelle Holdsworth

Covers nutritional issues through the entire lifecycle, from preconception to old age

Addresses the important and growing problem of obesity

Includes the nutritional science which underpins the application of nutrition and dietetics

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Oxford Handbook of Nutrition and Dietetics

Second edition

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Foreword

Both health professionals and the general public now realize that good nutrition is essential for good health. Indeed, nutrition is the health topic on which the lay public receives the most advice from popular books and magazines, but often this advice is unsound. It is therefore essential that health-care workers have readily available reliable information about all aspects of nutrition. This includes nutritional science, public health nutrition, and therapeutic nutrition.

This handbook provides, in concise format, the information about nutrition needed by those training to be dietitians (RD), nutritionists (RNutr), public health nutritionists (RPHNutr), or doctors or nurses either in all settings. It will continue to be a valuable resource after graduation, since the scope of modern nutrition is so large that a specialist in one field (say, public health nutrition) cannot hope to have instantly accessible all the necessary information about therapeutic diets, or nutritional sciences, and *vice versa*.

The three authors of this Handbook are all registered dietitians, each of whom has a solid research record, as well as extensive experience of the nutritional problems that dietitians, hospital doctors, general practitioners, and specialist nurses will encounter. I am confident that readers will be thankful to have this book in their pocket to guide them to the correct immediate response to a nutritional problem, even if later they have to consult a senior dietitian or textbook for more detailed advice.

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Preface

When we were approached to write this handbook the original idea was to write a book for general practice. However, we all remember being student dietitians and all created our own handbook of useful information that we carried around with us and were totally lost without. On reflection of what text books are now available in nutrition or dietetics, it became clear that, although there are now concise pocket books written for dietitians working predominantly in a clinical setting, there was a need for a user friendly handbook of nutrition and dietetics for a wider audience that included doctors, nurses, nutritionists, and other health care professionals. The available textbooks are, by necessity, large tomes or series that are unlikely to adorn the shelves of many doctors or nurses whether in primary or secondary care.

As a result, we have tried to present nutritional science, therapeutics, and community public health nutrition in a concise and integrated manner. While writing the text we have tried to identify what information would be useful to different professionals in a variety of settings. For example a doctor or nurse may want information on obesity and will find a ready reckoner for the calculation of body mass index (BMI), information on associated problems and treatment options. Dietitians working in the community or public health will have this information, but will find the sections on the measurements of obesity or nutrition interventions more informative. How well we have achieved this is for the reader to decide.

Nutrition is fascinating for many reasons, one of which is the fact that it is a very dynamic discipline. We have tried very hard to be contemporary, but there will inevitably be changes in basic science, practice and policy as the discipline continues to evolve. Major developments and changes will be posted on the relevant page of the OUP web site. For us it has been a very enjoyable, if at times rather demanding, process and we hope that this book is useful to all health care professionals.

J.W-G.
A.M.M.
M.H.

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Symbols and abbreviations

AAD	antibiotic-associated diarrhoea
↑	increase
↓	decrease
→	leads to
❗	caution
∴	therefore
🖱	website
♀	female
♂	male
💣	controversial topic
1°	primary
2°	secondary
5FU	5-fluorouracil
AA	amino acid
abv	alcohol by volume
ACE	angiotensin-converting enzyme
AcP	acute pancreatitis
AD	Alzheimer's disease
ADeH	alcohol dehydrogenase
ADH	antidiuretic hormone
ADHD	attention deficit hyperactivity disorder
ADP	air-displacement plethysmography
AfN	Association for Nutrition
AI	adequate intake
AIDS	acquired immune deficiency syndrome
AKI	acute kidney injury
ALA	alpha-linolenic acid
ALDH	aldehyde dehydrogenase
AN	anorexia nervosa
AP	assistant practitioners
Arg	arginine
ART	antiretroviral therapy
ARV	antiretroviral
ASA24	automated self-administered 24-h recall
ASD	autism spectrum disorders
Assoc. Nutr.	associate nutritionist

ATP	adenosine triphosphate
BAPEN	British Association for Parenteral and Enteral Nutrition
BCS	behaviour change strategies
BDA	British Dietetic Association
BED	binge eating disorder
BFI	baby friendly initiative
BHA	butylated hydroxyanisole
BHF	Better Hospital Food
BHT	butylated hydroxytoluene
BIA	bioelectrical impedance analysis
BMA	British Medical Association
BMI	body mass index
BMR	basal metabolic rate
BMT	bone marrow transplantation
BN	bulimia nervosa
BNF	British National Formulary
BPD	bilio-pancreatic diversion
BPD-DS	bilio-pancreatic diversion with duodenal switch
BSA	body surface area burn
BV	body volume
BWt	body weight
CBT	cognitive behavioural therapy
CC	critical care
CD	Crohn's disease
CF	cystic fibrosis
CHART	continuous hyperfractionated accelerated radiotherapy
CHD	coronary heart disease
CHO	carbohydrates
CI	Consumer International
CKD	chronic kidney disease
Cl	chlorine
CLA	conjugated linoleic acid
CMAM	community-based management of acute malnutrition
CNS	central nervous system
CoD	coeliac disease
COMA	Committee on Medical Aspects of Food Policy
CP	chronic pancreatitis
CQC	Care and Quality Commission
CRP	C-reactive protein
CRRT	continuous renal replacement therapy

CT	computed tomography
CVA	cerebrovascular accident
CVD	cardiovascular disease
CWT	Caroline Walker Trust
DASH	dietary approaches to stop hypertension
DBP	diastolic blood pressure
DEFRA	Department for Environment, Food and Rural Affairs
DES	dietary energy supply
DfE	Department for Education
DFS	Defence Food Services
DH	Department of Health
DHA	docosahexaenoic acid
DHp	dermatitis herpetiformis
DIT	dietary induced thermogenesis
DM	diabetes mellitus
DNA	deoxyribonucleic acid
DOM	Dietitians in Obesity Management
DRV	dietary reference value
DS	duodenal switch
DSW	dietetic support worker
DXA	dual-energy X-ray absorptiometry
EAR	estimated average requirements
EB	epidermolysis bullosa
ECF	extracellular fluid
EE	energy expenditure
EFA	essential fatty acids
EFAD	European Federation of the Associations of Dietitians
EFS	Expenditure and Food Survey
EFSA	European Food Safety Authority
EGRA	erythrocyte glutathione reductase activity
EMA	endomysial antibodies
EPA	eicosapentaenoic acid
EPAFF	Expert Panel on Armed Forces Feeding
ER	emergency regimen
ERF	established renal failure
EU	European Union
EUFIC	European Food Information Council
FAD	flavin adenine dinucleotide
FAO	Food & Agriculture Organization (UN)

FBDG	food-based dietary guidelines
FBS	food balance sheets
FFM	fat free mass
FFQ	food frequency questionnaire
FFST	fat-free soft tissue
FFW	food for work
FHS	food hypersensitivity
FIRSSt	Food Intake Recording Software System
FIVE	familial isolated vitamin E
FIVR	food intake visual and voice recognizer
FM	fat mass
FMN	flavin mononucleotide
FOS	fructo-oligosaccharides
FPIES	food-induced proctitis and entercolitis
FSA	Food Standards Agency
FSP	Food in Schools Programme
GAM	global acute malnutrition
GDA	guideline daily amounts
GDM	gestational diabetes
GF	gluten-free
GFD	general food distribution
GFR	glomerular filtration rate
GI	gastrointestinal, also glycaemic index
GL	glycaemic load
Gln	glutamine
GM	genetically modified
GMO	genetically modified organisms
GOR	gastro-oesophageal reflux
GORD	gastro-oesophageal reflux disease
GOS	galacto-oligosaccharides
GTF	glucose tolerance factor
GTN	glyceryl trinitrate
GVHD	graft vs. host disease
HD	haemodialysis
HDL	high density lipoproteins
HFE	high fat or energy
HFSS	high fat, sugar or salt
HIV	human immune virus
HNR	Human Nutrition Research
HPC	Health Professions Council

HR	heart rate
HT	hypertension
Ht	height
IA	insulin analogues
IBD	irritable bowel disease
ICCID	International Council for Control of Iodine Deficiency
ICF	intracellular fluid
IDA	iron deficiency anaemia
IDDM	insulin dependent diabetes mellitus
IDL	intermediate density lipoproteins
IDPN	intradialytic parenteral nutrition
IF	intestinal failure
IFE	infant feeding in emergencies
IGD	Institute of Grocery Distribution
IgE	immunoglobulin E
IMD	inherited metabolic diseases
IMF	International Monetary Fund
INR	international normalized ratio
INS	International Numbering System
IOM	Institute of Medicine
IOTF	International Obesity Task Force
ISAK	International Society for the Advancement of Kinanthropometry
IVNAA	in vivo neutron activation analysis
J	joule
kcal	kilocalories
KD	ketogenic diet
kJ	kilojoules
LBW	low birth weight
LCP	long chain fatty acids
LCT	long chain triglycerides
LDL	low-density lipoprotein
LFT	liver function test
LIDNS	Low Income Diet and Nutrition Survey
LGIT	low glycaemic index treatment
LP(a)	lipoprotein (a)
LRNI	lower reference nutrient intake
MAC	midarm circumference
MAD	modified Atkins diet
MAM	moderate acute malnutrition

MAMC	midarm muscle circumference
MAOI	monoamine oxidase inhibitors
MAS	milk alkali syndrome
MBD	mineral bone disease
MCH	mean cell haemoglobin
MCT	medium chain triglycerides
MCV	mean corpuscular volume
MDG	millennium development goals
MDT	multidisciplinary team
MEOS	microsomal ethanol-oxidizing system
MHRA	Medicines and Healthcare products Regulatory Agency
MI	motivational interviewing
MIMS	Monthly Index of Medical Specialties
MJ	megajoules
MND	motor neurone disease
MoD	Ministry of Defence
MPFR	mobile phone food record
MRC	Medical Research Council
MRI	magnetic resonance imaging
MS	multiple sclerosis
MTCT	mother-to-child transmission
MUAC	mid-upper arm circumference
MUFA	monounsaturated fatty acids
MUST	malnutrition universal screening tool
Na	sodium
NAD	nicotinamide adenine dinucleotide
NADP	nicotinamide adenine dinucleotide phosphate
NAFLD	non-alcoholic fatty liver
NASH	non-alcoholic steatohepatitis
NatCen	National Centre for Social Research
NATO	North Atlantic Treaty Organization
NCCTSL	non-carious cervical tooth surface loss
NCD	nutrition-related chronic diseases
NCHS	National Center for Health Statistics
NDNS	National Diet and Nutrition Survey
NE	niacin equivalent
NFS	National Food Survey
NG	nasogastric
NGA	non-governmental agency

NHANES	National Health and Nutrition Examination Surveys
NHS	National Health service
NICE	National Institute for Health and Clinical Excellence
NIDDM	non-insulin dependent diabetes mellitus
NIE	nutrition in emergencies
NIRI	near infrared interactance
NJ	nasojejunal
NMES	non-milk extrinsic sugars
NMN	N`-methylnicotinamide
NS	nephrotic syndrome
NS-SEC	National Statistics Socio-economic Classification
NSF	National Service Frameworks
NSP	non-starch polysaccharides
NTD	neural tube defects
OA	osteoarthritis
OCD	obsessive compulsive disorder
OFC	occipito-frontal head circumference
Ofsted	Office for Standards in Education, Children's Services and Skills
ONS	Office for National Statistics
ORP	operational ration packs
PA	physical activity
PABA	para-aminobenzoic acid
PAD	peripheral arterial disease
PAL	physical activity level
PAR	physical activity ratios
PAYD	Pay as You Dine
PCB	polychlorinated biphenyl
PCHR	Personal Child Health Record
PCOS	polycystic ovary syndrome
PCR	protein catabolic rate
PCSG	Primary Care Society for Gastroenterology
PD	peritoneal dialysis
PDA	personal digital assistant
PDis	Parkinson's disease
PEG	percutaneous endoscopic gastrostomy
PEJ	percutaneous endoscopic jejunostomy
PERT	pancreatic enzyme replacement therapy

PFS	Pollen Food Syndrome
PHCT	primary health care teams
Phe	phenylalanine
PICC	peripherally inserted central catheter
PKU	phenylketonuria
PMTCT	prevention of mother to child transmission
PN	parenteral nutrition
PNI	protective nutrient intake
PRG	percutaneous radiological gastrostomy
PSE	portal systemic encephalopathy
PUFA	polyunsaturated fatty acids
PWS	Prader–Willi syndrome
QUID	quantitative ingredient declaration
R. Nutr.	registered nutritionist
R. PHNutr.	registered public health nutritionist
RD	registered dietitian
RDA	recommended dietary allowance
RDis	Refsum's disease
RDS	rapidly digestible starch
RfS	refeeding syndrome
RIG	radiologically inserted gastrostomy
RMR	resting metabolic rate
RNA	ribonucleic acid
RNI	reference nutrient intake
RQ	respiratory quotient
RQIA	Regulation and Quality Improvement Authority
RS	resistant starch
RUTF	ready to use therapeutic food
SACN	Scientific Advisory Committee on Nutrition
SAM	severe acute malnutrition
SAP	severe acute pancreatitis
SBP	systolic blood pressure
SBS	short bowel syndrome
SCF	Scientific Committee for Food
SCI	spinal cord injury
SD	standard deviation
SDC	Sustainable Development Commission
SDS	slowly digestible starch
SEMS	self-expanding metal stent
SENr	Sport and Exercise Nutrition Register

SFA	saturated fatty acids
SFT	School Food Trust
SGA	subjective global assessment
SLE	systemic lupus erythematosus
SPT	skin prick test
TBK	total body potassium
TBW	total body water
TEE	total energy expenditure
TG	trigylceride/triacylglyceride
TOBEC	total body electrical conductivity
TPN	total parenteral nutrition
TPP	thiamine pyrophosphate
TSF	triceps skin-fold
TSH	thyroid-stimulating hormone
tTGA	IgA tissue transglutaminase
TVP	textured vegetable protein
UC	ulcerative colitis
UF	ultrafiltration
UL	upper limit
UNU	United Nation University
US	ultrasound
UWW	under-water weight
VAD	vitamin A deficiency
VLCD	very low calorie diets
VLDL	very low-density lipoproteins
WHO	World Health Organization
WRVS	Womens Royal Voluntary Services
Wt	weight

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Chapter 1

Introduction to nutrition

Definitions and titles

Nutrition

'Nutrition is the branch of science that studies the process by which living organisms take in and use food for the maintenance of life, growth, reproduction, the functioning of organs and tissues, and the production of energy.'[1]

Public health nutrition

Usually described as 'the promotion of good health through nutrition and the primary prevention of nutrition-related illness in the population'. Emphasis is on maintaining the wellness of the population through applying public health principles to influence food and nutrition systems. No internationally agreed definition.

Dietitian (dietician)

The titles dietitian and dietician are protected by law in the UK; anyone using these titles must be registered with the Health Professions Council (HPC). Anyone using these titles without registration is liable to prosecution and may be prosecuted. Registered dietitians are also able to use the post-nominal letters RD (formerly SRD in the UK). The European Federation of the Associations of Dietitians (EFAD) has defined the role of the dietitian as follows.

- A dietitian is a person with a qualification in nutrition and dietetics recognized by national authorities. The dietitian applies the science of nutrition to the feeding and education of groups of people and individuals in health and disease.
- The scope of dietetic practice is such that dietitians may work in a variety of settings and have a variety of work functions.

European academic and practitioner standards for dietetics can be found on the EFAD web site (www.efad.org).

Many dietitians work in the National Health Service (NHS) and may specialize in specific areas, e.g. oncology, renal disease. They are employed in all sectors of healthcare and are a key part of the health-care team. Dietitians also work outside the NHS in areas such as industry, sport, education, journalism, and research.

Health Professions Council

More information about Health Professions Council (HPC) is available at www.hpc-uk.org.

British Dietetic Association

The British Dietetic Association (BDA) is the professional body representing dietitians and was established in 1936 in order to:

- advance the science and practice of dietetics and associated subjects;

[1] Bender, A.E. and Bender, D.A. (1995). *Oxford dictionary of food and nutrition*. Oxford University Press, Oxford.

- promote training and education in the science and practice of dietetics and associated subjects;
- regulate the relations between dietitians and their employer through the BDA trade union.

Specialist groups within the BDA cover areas of specialist interest, e.g. Paediatric Group, Dietitians in Obesity Management (DOM) UK. Full membership is available to RDs; other membership categories are available for dietetic assistants, students, and affiliates. The BDA is responsible for the curriculum framework for the education and training of dietitians. More information about the BDA is available at www.bda.uk.org. The BDA is one of the 30 member associations, representing dietitians in 24 European countries, of the European Federation of the Associations of Dietitians (EFAD) (www.efad.org). It is also one of about 40 national dietetic associations who are members of the International Confederation of Dietetic Associations (www.internationaldietetics.org).

Dietetic support workers and assistant practitioners[2]

Dietetic support workers (DSW) and assistant practitioners (AP) work under the direct supervision of a RD. Their roles may include administration and dietetic tasks as delegated by the RD. In a hospital setting these may include assisting patients requiring special diets to choose from the hospital menu, and collecting and recording information regarding the patient's food consumption and weight. In primary care they may include providing dietary consultation, under the direction of the dietitian, and liaising with the RD regarding the patient's progress. Within a community setting they may include assisting the dietitian to assess the food and health needs of local residents, and enabling people to eat a healthier diet to prevent disease, offering guidance in relation to food selection and preparation, planning menus, standardizing recipes, and testing new products. Individual tasks undertaken by DSWs, and even more so by APs, may be exactly the same as the level 5 dietitian with the difference being in the detail of the task/activity the level of autonomy. Unlike the dietitian a DSW or AP would have established and predetermined protocols for which referrals they are able to accept, and for which conditions, and at what point they would need to hand over to a dietitian. There would be pre agreed treatment options and a DSW or AP would not have the autonomy to move away from these options without first agreeing it with a dietitian. Again in project work, e.g. healthy eating session or diet sheet/resource development, it would be expected that the dietitian would oversee the project once delegated, and then sign off the information/project plan at the end. The level of both experience and formal education achieved will lead the difference between a DSW and an AP, and the complexity of the work expected of them. National Vocational Qualification level 3 courses are available in allied health professional support (dietetics).

[2] Dietetic Support Worker and Assistant Practitioner Roles BDA 2010 Available at: www.bda.uk.com.

Nutritionist

The title 'nutritionist' has no legal standing and no educational requirements are necessary for a person to be called 'nutritionist'. The Association for Nutrition is endeavoring to regulate the field of nutrition and protection of the title 'nutritionist'.

The Nutrition Society

The Nutrition Society (www.nutritionsociety.org.uk) was established in 1941 'to advance the scientific study of nutrition and its application to the maintenance of human and animal health'. The society covers 4 key areas:

- promotion of professional study;
- promotion of high standards in professional practice;
- promotion of professional careers;
- public protection through voluntary professional registration.

In 2010 the responsibility for the UK Voluntary Register of Nutritionists was transferred to the Association for Nutrition.

The Association for Nutrition

The Association for Nutrition (AfN) is a professional body for the regulation and registration of nutritionists (including public health nutritionists and animal nutritionists). Registrants must demonstrate high ethical and quality standards, founded on evidence-based science. The AfN sets proficiency and competency criteria, promotes continuing professional development and safe conduct, and will accredit university undergraduate and postgraduate nutrition courses. The association awards the titles associate nutritionist (Assoc. Nutr.), registered nutritionist (R. Nutr.) and registered public health nutritionist (R. PHNutr.) however this is currently under review. Further details can be obtained at www.associationfornutrition.org

Registered public health nutritionist

Registered public health nutritionists work in health improvement on a population level to promote health, wellbeing and reduce inequalities. Based in a range of sectors, e.g. Primary Care, health improvement, government departments, non-governmental organizations, food retailer or manufacturer.

Registered Sport and Exercise Nutritionist

The Sport and Exercise Nutrition Register (SENr) (www.senr.org.uk) is a voluntary register designed to accredit suitably qualified and experienced individuals who have the competency to work autonomously as a Sport and Exercise Nutritionist with performance orientated athletes, as well as those participating in physical activity, sport and exercise for health. The register is administered by BDA on behalf of the SENr Board.

Components of the diet

Diet

Diet is what a person habitually eats and drinks, so everyone is always on a diet. One of the most important and difficult tasks in nutritional medicine is to estimate accurately the habitual nutritional intake and diet of the patient. These difficulties arise because a person's diet may vary widely from day to day, food processing may greatly affect the nutrient content of foods s/he eats, and hardly anyone with a nutritional problem can accurately recall what s/he has eaten.

Dietary value

Dietary value is assessed by the measured energy and nutrient content of a particular diet and often in reference to dietary reference values (see 📖 Chapter 2 'Dietary reference values', p. 20) or recommendations. Foods and diets also have many other kinds of value including political, economic, social, and cultural values (see 📖 Chapter 14 'Influences on children's food choices', p. 293). In most societies where people live above starvation level effort is put into diversifying meals and the overall diet, e.g.:

- use of food in rituals, e.g. birthday and wedding cakes, also fasting (Ramadan and Lent);
- use of food to express values and social relationships, e.g. sharing food, preparing special foods as expression of love, etc.;
- prestige foods, e.g. champagne and caviar as symbols of wealth and privilege.

Components of the diet

Diets are composed of nutrients: macronutrients (protein, fats, carbohydrates, and alcohol) and the micronutrients (vitamins, minerals, and trace elements). Food also contains many non-nutritional, but biologically active substances. These include toxins and contaminants, such as alkaloids and aflatoxins, which are detrimental to health, as well as constituents, such as phytochemicals, that may be health-promoting. As consumers we do not eat nutrients, but meals and foods. These are the components of diet that are most meaningful to the public and usually the basis of food choice.

Food groups

Foods vary in their energy and nutrient content. Food groups are a classification of foods on the basis of the nutrient profile (see 📖 Chapter 2 'The Eatwell Plate', p. 27 and Table 1.1). Commonly used food groups are:

- high protein foods, e.g. meat, fish, eggs, dairy products, pulses/legumes;
- carbohydrate-rich foods, e.g. cereals, roots, and tubers;
- dairy foods;
- fruit and vegetables;
- foods rich in fat or oil.

Table 1.1 Nutrient profile of the main food groups

Food group	Fat	Carbo-hydrate	Protein	Fat-soluble vitamins	Water-soluble vitamins	Minerals
Cereals		+++	++		++ (Bs) but variable	+
Roots and tubers		+++	+ but variable		++ (C) but variable	
Legumes/ pulses	Variable	+++	++		++ (Bs)	+
Meat, fish, eggs	+		+++	++	+ (Bs)	+
Dairy products	+		++	++	+ (C)	++
Fruits		+			+++ (C)	
Vegetables			++		+++ (C, folate)	++
Sugar		+++				
Fats and oils	+++			+++ but variable		

+, This food group is a source of the nutrient(s) in most human diets; ++, this food group is an important source of the nutrient(s) in most human diets; +++, this food group is a major source of the nutrient(s) in most human diets.

Food groups are widely used in the formulation of dietary guidelines and for nutrition education messages of various kinds, such as eat five portions of fruit and vegetables a day (a UK health message). While useful, such classifications are also somewhat arbitrary; some foods can be placed in more than one food group.

Staple foods

Traditionally a staple food is one that forms the basis of the diet in terms of both quantity and frequency of consumption, and that provides the highest proportion of energy. In developed countries it is not always easy to specify one particular food as the staple. Staple foods vary with geographic region, but in global terms the most important staple foods are the following.

- *Cereals:* globally cereals supply approximately 51% of the world's dietary energy supply (DES) with rice, maize, and wheat the most

important, although other cereals, such as millet and sorghum, are also important in some regions. Cereals are a good source of carbohydrate, but also contain reasonable amounts of protein and, depending on variety and processing, some micronutrients, e.g. Fe and some B vitamins.

- *Roots and tubers, and particularly cassava or manioc:* in sub-Saharan Africa they supply 22% DES, with this figure rising to over 70% in individual countries, such as the Democratic Republic of Congo. Other important roots are potatoes, yams, sweet potatoes, and taro. They are high in carbohydrate, but low in fat, protein, and, with some important exceptions, such as sweet potatoes, micronutrients.

Other less common staple foods are sago eaten in parts of Malaysia and Indonesia, and plantain and bananas in many tropical countries (sub-Saharan Africa, Asia, Caribbean, and South America). The importance of staple foods has declined in industrialized countries (e.g. in industrialized countries cereals only supply 26% DES), but they remain important in many low-income countries. In Nepal, 77% DES from cereals (predominantly rice), while in the USA only 23% DES derives from cereals (as a mixture of rice, wheat, and maize).

Meals

Most foods are eaten as part of meals. Meals may differ in the following ways.

- The combination of foods eaten, e.g. the traditional British meal of 'meat and two veg'.
- How they are processed, prepared, and cooked. This can have an impact on the nutritional value of food, e.g. steaming, rather than boiling vegetables reduces the loss of water-soluble vitamins.
- The order in which particular items or dishes are consumed. In most European countries a formal meal is a three-course sequence pattern of starter, main course, and pudding or dessert, whereas in Chinese banquets many dishes tend to be served at once.
- How food is eaten. With hands or implements, from separate dishes or a common bowl. This is largely a matter of social etiquette, but can be important in child feeding, e.g. if small children are fed from a common pot, rather than given an individual serving.
- Who eats with whom and the allocation of food within the household. In some societies men and women eat separately, and there is also an unequal division of food between the sexes, including children.

These meal patterns may impact upon the dietary intake of individuals within a household.

Snacks

Snacks are foods that are not eaten as part of meals. The place of snacks in peoples' diets and their contribution to overall dietary intake are variable.

Food composition tables

The food composition tables used in the UK are those of McCance and Widdowson. The 6th edition was published in 2002 by the Food Standards Agency in collaboration with the Royal Society of Chemistry (🖱 www.food.gov.uk).[1] The UK Nutrient Database is maintained by the FSA. Food tables may be country-specific to account for country-specific food laws, e.g. fortification. An EU funded project (EuroFIR,[2] 🖱 www.eurofir.net) has made some progress towards the development of a comprehensive, validated databank providing a single, authoritative source of food composition data for Europe.

Food composition tables list the energy, macronutrient, and selected micronutrient content of selected foods. Mean values are derived from representative samples of each type of food and expressed in standard units of 100 g per food type. Values are usually expressed in terms of the edible portion of the food, although 'as purchased' values may be given. The contents are arranged by food groups: cereals and cereal products, dairy products, eggs, meat and meat products, etc. Foods are given an individual code. Supplements are available for specific foods, e.g. fish, fats, and oils.

Food composition tables are used to analyse the foods and diets of individuals and groups; the values obtained are often then compared with dietary reference values (DRVs). Other uses of food composition tables include:

- the planning and assessment of food supplies, e.g. during famines or war;
- designing institutional and therapeutic diets, e.g. in schools or hospitals;
- prescription of diets in clinical practice;
- modifying diets to ↑ or ↓ particular nutrients;
- health promotion and teaching;
- nutrition labelling;
- food regulations and consumer protection;
- research on relationships between diet and disease.

Food composition tables are compiled by laboratory analyses of selected samples of foods and cooked recipe dishes. They may also be compiled from published results in the literature.

❗ Tables usually include an introduction explaining how they are compiled; it is important to read this section.

Calculation of energy values

The gross energies of foods are measured using a ballistic bomb calorimeter, but the values used in the tables are the energy available for the body to metabolize—metabolizable energy. Metabolizable energy accounts for faecal and urinary losses. The difference between gross energy and metabolizable energy is about 5%. The direct measurement of metabolizable energy required human trials. Energy conversion factors, e.g. Atwater

[1] Food Standards Agency (2002). *McCance and Widdowson's the Composition of Food,* 6th summary edn. Royal Society of Chemistry, Cambridge.

factors are used (see Table 1.2). These factors are derived from elaborate human studies.

Table 1.2 Energy conversion factors

Nutrient	kcal/g	kJ/g	Comments
Protein	4	17	
Fat	9	37	Original Atwater factor was 8.9 kcal, ∴ the lower kJ figure is preferable
Carbohydrate	3.75	16	Value is for available carbohydrate expressed as monosaccharides. If carbohydrate is expressed directly or by difference 4 kcal/g is used
Sugar alcohols	2.4	10	Mean value used in food labeling
Ethyl alcohol	7	29	
Glycerol	4.31	18	Assumes complete metabolism

Calculation of protein content

Most tables give protein and amino acid analyses. Protein content is usually derived from nitrogen content. It is assumed that on average protein is 16% nitrogen. Therefore, the nitrogen content is multiplied by 6.25 (100/16) to derive protein content, but there are limitations:

- the nitrogen content of food proteins varies;
- the nitrogen content varies with amino acid composition;
- other food constituents contain nitrogen, e.g. purines, urea, pyrimidines, and dipeptides.

Calculation of fat content

Most tables give total fat and fatty acid analyses. Before determining the fat content of foods it is necessary to extract the fat with alcohol, which can be done by a variety of methods, e.g. Soxhlet extraction. Each method of extraction will vary in the extent to which different fats are extracted so introducing a possible error.

Calculation of carbohydrate content

Some tables report carbohydrate content by difference, i.e. carbohydrate = 100 – amounts of protein, water, fat, and ash. This assumes that all carbohydrates have equal digestibility, which is not correct. Other tables summarize measured values of total available carbohydrate (the sum of sugars and starches); this is usually reliable, but the ↑ use of glucose and high fructose syrups may → overestimation of sucrose.

Dietary fibre is determined by one of two methods (Englyst and Southgate) and values from the methods should not be mixed. In the UK the Englyst method is used most widely and the 'fibre' content is described as 'non-starch polysaccharides' as this best describes biologically useful fibre.

Calculation of micronutrient content

There are many methods for measuring micronutrients and these have variable accuracy. Some micronutrients have a variety of forms that are biologically active, e.g. folate. No single assay gives total free folate activity in foods.

Limitations of food tables

Real variation in energy and nutrient content All foods vary in energy and nutrient content because of many factors—variety or strain, sex and age of animals, agricultural processes, environmental factors, e.g. soil and climate, conditions and duration of storage, processing, and preparation. There is less variation in macronutrients than micronutrients with the exception of fat. The cut of meat will → a variation in fat content as will personal preference of the consumer.

Variation in water content Water content is one of the most significant sources of variation in nutrients. Dry cereal grains have relatively little water, but their content is variable and the amount of water absorbed in preparation is variable, e.g. cooked rice has water content of between 65 and 80%.

Sampling errors The sample analysed must be representative of the average composition of particular foods. This needs to take into account seasonal or regional variations. This is particularly true of processed foods, where the recipe and process is variable. Different recipes will add another layer of inaccuracy. Recipes are often given in food composition tables or the recipe used should be calculated from raw ingredients.

Inappropriate methods The choice of analytical method is important and should be reported. Some methods used for the determination of a nutrient may not be interchangeable, e.g. fibre.

Laboratory errors Laboratories and/or processes are standardized, but errors may still occur.

Use of conversion factors Conversion factors may introduce errors as described before.

Bioavailability This is not an error, but it is important to consider the bioavailability of specific nutrients.

Errors in coding and calculation Calculation of the nutrient content of foods requires precise information on the amounts of food eaten. Often average portion sizes are used, which will introduce errors. Average portion sizes will vary with age and between countries, cultures, etc. Average portion sizes have been published for some countries. Errors may also occur in the coding of foods and calculation of nutrient content.

Studies have compared values obtained directly and by using food tables, and found that energy and protein values varied by 10–15% and that values for micronutrients varied by up to 50%. Provided the limitations of the use of food tables are understood they are invaluable tools for nutritionists and dietitians.

Food composition analysis programmes are now available that make the calculations less arduous, e.g. CompEat, Dietplan 6.

Digestion

Food is broken down by mechanical and chemical mechanisms in the gastrointestinal (GI) tract before nutrients can be absorbed into the body. The GI tract is a continuous tube from the mouth to the anus and is approximately 7 m in length (Fig. 1.1). Food is transported through the lumen of the tract as it is digested.

The mouth and oesophagus

Food is chewed by teeth and mixed with saliva, which is produced by salivary glands (parotid, submaxillary, and sublingual glands). Saliva contains the enzyme amylase, which starts the digestion of starch. The food is mixed with saliva, fluid, and mucus to form a bolus that is pushed into the pharynx by the tongue. The pharyngeal muscle contracts to swallow the bolus of food. The bolus is moved down the oesophagus into the stomach by peristalsis.

The stomach

The cardiac sphincter is found at the junction of the oesophagus and stomach and contracts to prevent food leaving the stomach and re-entering the oesophagus. The stomach is a muscular organ that further breaks down the bolus by mechanical, chemical, and enzymatic actions. Parietal glands in the stomach wall secrete hydrochloric acid, which helps break down the food, denatures protein, and converts the inactive pepsinogen into active pepsin. Chief cells in the stomach secrete pepsinogen. Pepsin begins the breakdown of proteins. Renin and gastric lipase break down milk protein and fat, respectively. Goblet cells secrete mucin, which protects the stomach from hydrochloric acid. The food is converted into chyme in the stomach, which then passes into the small intestine.

The small intestine

The pyloric sphincter is a circular muscle at the junction of the stomach and small intestine that controls the release of chyme into the small intestine. The small intestine consists of the duodenum, jejunum, and the ileum. Chyme is transported along the small intestine by slow muscular contractions known as peristalsis. It can take up to 5 h to complete the movement through the small intestine; this slow transition aids absorption. The surface area of the small intestine is large to facilitate digestion and absorption. Villi and microvilli are finger-like projections lining the lumen. Enzymes lactase, maltase, and sucrase are secreted by the microvilli and complete carbohydrate digestion into monosaccharides. The villi have thin walls through which nutrients are absorbed into capillaries (carbohydrates and proteins) and lacteals (fat absorption, Fig. 1.2). The lacteals connect with the lymphatic system. Proteins are further broken down in the small intestine into amino acids, which can be absorbed through the villi wall.

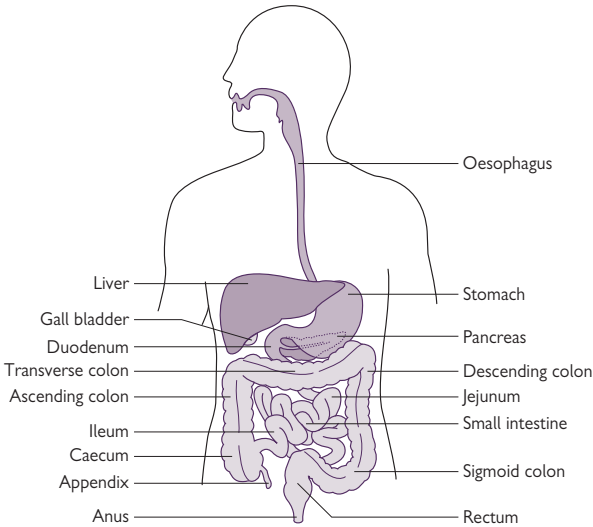

Fig. 1.1 The gastrointestinal tract.

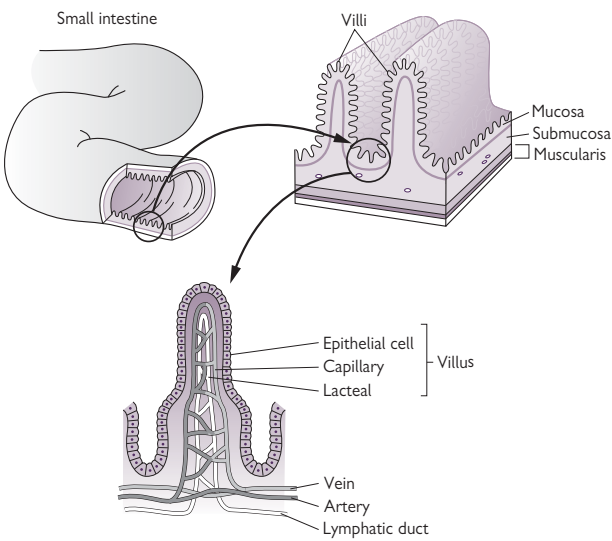

Fig. 1.2 Villi of the small intestine.

The pancreas secretes a mixture of enzymes that continue digestion; trypsinogen and carboxypeptidase break down proteins and polypeptides into amino acids, and lipase breaks down fats into fatty acids. Bile is secreted by the liver and stored and concentrated by the gall bladder. Bile dilutes and buffers the chime, and emulsifies fat so enabling lipase to break it down. Water-soluble nutrients (amino acids, monosaccharides, and water-soluble micronutrients), and short and medium chain fatty acids are taken to the liver via the portal vein. Fat-soluble nutrients are transported in the lymphatic system and enter the blood system at the left subclavian vein.

The large intestine

The remaining chyme passes into the large intestine through the ileocaecal sphincter, a circular muscle that separates the small and large intestines. The large intestine consists of the caecum (and appendix), colon (ascending, transverse, descending, and sigmoid), rectum, and anus. <10% digestion occurs in the large intestine. Water is reabsorbed to conserve water and to form faeces. Some vitamins including vitamin K and biotin are absorbed in the large intestine. Faeces consist of undigested food, particularly insoluble fibre, and are expelled from the rectum through the anus by powerful contractions. The anal sphincter controls defaecation.

Fat digestion and absorption

Most dietary fat is in the form of triacylglycerides (triglycerides) and is digested by pancreatic lipase into non-esterified fatty acids and monacylglycerides. Phospholipid digestion yields lysophosphoglyceride and a fatty acid. Cholesterol is hydrolysed before absorption. Triacylglyceride digestion is very efficient with 95% of fat being digested and absorbed; only 40% cholesterol is absorbed. The products of fat digestion pass into 'mixed micelles': large molecular aggregates of monoacylglycerides, large fatty acids, bile salts, and phospholipids. Cholesterol, carotenoids, tocopherols, and some undigested trigacylglycerides are taken into the hydrophobic core of the micelles (Fig. 1.3).

Lipid absorption occurs mainly in the jejunum. The digestion products pass from the micelles into the enterocyte's membrane by passive diffusion. A fatty acid binding protein binds to fatty acids and they are rapidly re-esterified to monoaclyglycerides. Cholesterol is re-esterified by acyl-CoA:cholesterol acyltransferase or by the reversal of cholesterol esterase. Cholesterol esterase is induced by high levels of dietary cholesterol. Fats are packaged into chylomicron taken into lacteals; they circulate via the lymphatic system and are mainly removed in adipose tissue by lipoprotein lipase. The chylomicrons are not completely consumed by the enzyme, but are degraded to smaller particles, remnants that are removed by the liver. Short and medium chain fatty acids are directly absorbed into the portal vein. Lipids are synthesized in the liver and those delivered by chylomicron remnants are packaged into very low density lipoproteins (VLDL) and secreted into the blood.

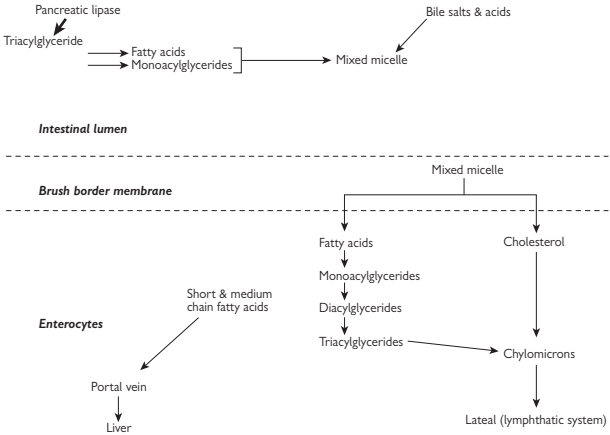

Fig. 1.3 Fat digestion and absorption.

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Chapter 2

Dietary reference values and food-based dietary guidelines

Dietary reference values

Dietary reference values (DRVs) are established within a population as a measure of nutritional adequacy. The first DRVs were established in the late 19th century and international values were established by the League of Nations in 1936–1938 to prevent deficiencies in population groups. Many countries have their own values and international values have been published by FAO/WHO/UNU. DRVs for food, energy, and nutrients for the UK (report of the Panel on DRVs of the Committee on Medical Aspects of Food Policy (COMA) were last revised in 1991.[1] See OUP website for revisions since publication of this handbook (🖱 www.oup.com). Updates may be available from the Department of Health (🖱 www.dh.gov.uk) or the Food Standards Agency (🖱 www.food.gov.uk). UK DRVs are given for energy and nutrients in 🕮 Chapter 5, 'Macronutrients and energy balance: introduction' and 🕮 Chapter 6, 'Micronutrients,' and summarized in Appendix 6.

DRVs are based on the assumption that the individual requirements for a nutrient within a population or group are normally distributed and that 95% of the population will have requirements within 2 standard deviations (SD) of the mean as shown in Fig. 2.1. They assume that individuals are healthy, and also consider gender, age, growth, and physiological status, i.e. pregnancy and lactation.

Limitations of DRVs

While DRVs can be useful, they can be misused and the inherent problems associated with making recommendations for the whole population should be appreciated.

- A standard distribution of nutrient requirements is assumed; the distribution may not be normal or insufficient data may be available to establish normality.
- Good data are required for the panel to evaluate requirements; these data may be derived from balance studies, tissue levels, pool size, etc., amount required to prevent symptoms of deficiency, or a measure of function of the nutrient (see Fig. 2.2). Such data are not always available and at times the panel has decided that the data are insufficient to set requirements and has ∴ recommended a 'safe level' of intake.

Factors affecting dietary requirements

- Metabolic requirement including:
  - age, gender, body size;
  - lifestyle (smoking, obesity, physical activity, etc.);
  - disease, e.g. fever, catabolism;
  - trauma;
  - growth.
- Bioavailability including:
  - altered absorption, e.g. milk Ca is better absorbed than non-milk Ca;
  - reduced utilization;

[1] Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom.* HMSO, London.

- ↑ losses, e.g. diarrhoea, burns, renal disease;
- environment, e.g. heating of nutrients;
- drugs, e.g. diuretics;
- dietary concentration;
- dietary interactions;
- drug–nutrient interactions.

Uses of DRVs

- Dietary assessment of individuals, although it must be remembered that DRVs are based on populations and groups not individuals. Other factors may need to be considered.
- Dietary assessment of groups or populations; it is important that the population is comparable with that for which the recommendations are derived.
- Prescription of diets and provision of supplies, e.g. school meals.
- Food labelling.
- Food formulation.

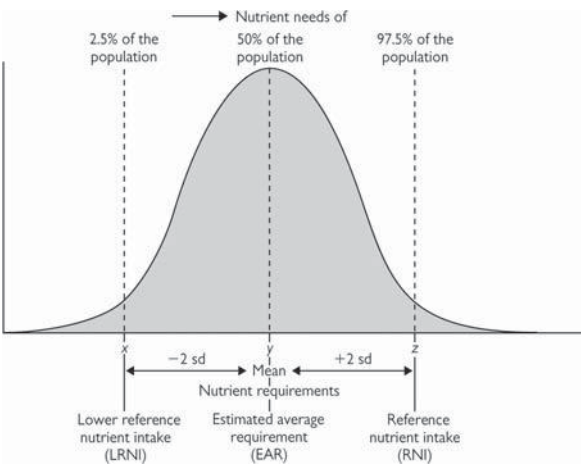

Fig. 2.1 Derivation and definition of dietary reference values in the UK. (Modified with permission from Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. Her Majesty's Stationary Office, London. Reprinted with permission.)

Definitions

⚠ The definitions will vary according to the country or organization responsible for the recommendation.

***UK*[1]**

Recommended Daily Amount (RDA) 'The average amount of the nutrient which should be provided per head in a group of people if needs of practically all members of the group are to be met' (Committee on Medical Aspects of Food (COMA), 1979).[2]

Recommended intakes 'The amounts sufficient, or more than sufficient, for the nutritional needs of practically all healthy persons in a population' (COMA, 1969).[3]

'Intake' emphasizes that the recommendations relate to food actually eaten.

Requirement The amount of a nutrient that needs to be consumed in order to maintain normal nutritional status.

Estimated average requirement (EAR) (point y in Fig. 2.1) The mean requirement of a nutrient for a population or group of people. On average 50% will consume more than the EAR and 50% less.

Lower reference nutrient intake (LRNI) (point x in Fig. 2.1) The level at only which approximately 2.5% of the population or group will have an adequate intake; it will not be enough for most people. An individual with this intake may be meeting their requirement, but it is highly probable that they are not.

Reference nutrient intake (RNI) (point x in Fig. 2.1) At this level intake will be adequate for 97.5% of the group or population. It is possible that an individual's intake will not meet their requirement at this level but is highly improbable. RNIs for micronutrients are given in Appendix 6 and as appropriate throughout the handbook.

Safe level Given when insufficient information is available to derive requirements. It is an average requirement plus 20% and is believed to be adequate for most people's needs. The panel judged that there was no risk of deficiency at this level and that there is no risk of undesirable effects above this level.

***FAO/WHO*[4]**

Estimated average requirement (EAR) As above.

[1] Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. Her Majesty's Stationary Office, London.
[2] Department of Health & Social Security (1979) *Recommended daily amounts of food energy and nutrients for groups of people in the United Kingdom*. Reports on Health and Social Subjects, 15. HMSO, London.
[3] Department of Health & Social Security (1969) *Recommended intakes of nutrients for the United Kingdom*. Reports on Public Health and Medical Subjects; 120. HMSO, London.
[4] FAO/WHO (1988) Expert Consultation on Human Vitamin and Mineral Requirements. WHO, Geneva.

Recommended nutrient intake (RNI) As RNI above.

Protective nutrient intake (PNI) An amount > RNI for some micronutrients that may be protective against a specified health or nutritional risk of public health relevance. PNI are expressed as daily value or an amount to be consumed with a meal.

Upper tolerable nutrient intake level (upper limit (UL)) **T**he maximum intake of some micronutrients that is unlikely to pose risk of adverse health effects in almost all (97.5%) apparently healthy individuals in a gender and age specific population.

USA[5]

Recommended dietary allowance (RDA) Average daily dietary intake that meets the requirements of nearly all (97–98%) healthy persons.

Adequate intake (AI) Established for a nutrient when available data are insufficient to estimate an intake that would maintain adequacy. The AI is based on observed intakes by a group of healthy persons.

Tolerable upper intake limit (UL) As above.

Estimated average requirement (EAR) As above.

[5] National Academy of Sciences, Institute of Medicine, Food and Nutrition Board (2006). Dietary Reference Intakes: the essential guide to nutrient requirements. National Academies Press, Washington.

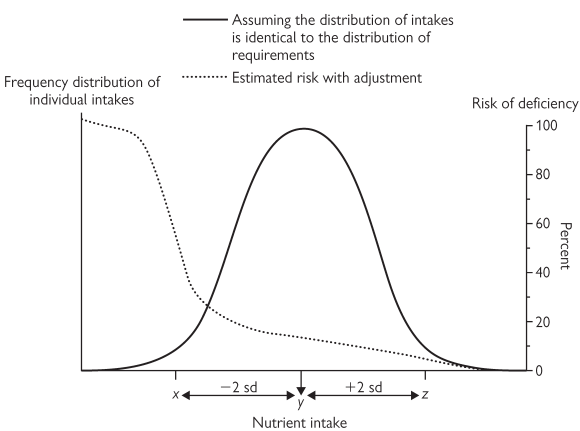

Fig. 2.2 Dietary intakes and risk of deficiency. (Modified with permission from Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. Her Majesty's Stationary Office, London. Reprinted with permission.)

Food-based dietary guidelines

Historically, dietary guidelines were nutrient-based; food-based dietary guidelines (FBDG) were developed to facilitate the teaching of 'healthy eating' and nutrition to population groups. FBDG focus on foods, rather than nutrients, and are intended for use by the general public. They are designed to be understandable to most consumers. FBDG give practical information on 'healthy eating' and intakes of nutrients to meet DRVs of macro- and micronutrients (see 🕮 Appendix 6, p. 777). FBDG are designed to be appropriate to each population group; they may be country, age, or culturally specific.

Development of FBDG

The FAO/European Food Information Council (EUFIC) report on the development of FBDG is available on the website (🔗 www.fao.org).

Key concepts

The following points should be considered in the development of FBDG.

Dietary patterns

- Total diet, rather than nutrients.
- Reflect food patterns, rather than numeric nutrient goals.
- Various dietary patterns can be compatible with health.

Practical considerations

- Food should be affordable, widely available, and accessible.
- FBDG should recognize social, environmental, and agricultural conditions affecting foods and eating patterns.
- They should be flexible such that they can be used by people with different lifestyles, ages, and physiological conditions, e.g. pregnancy.

Comprehensibility

- Should be easily understood.
- Food groups should make sense.
- Should include visual representations.
- Testing is essential before dissemination.

Cultural acceptability

- Foods and colours should be culturally appropriate.
- Should be sensitive to cultural and religious considerations.
- Avoid racial changes in current practice.
- Use appropriate dialect or language.
- Should be positive and encourage enjoyment.

Underlying assumptions

- Foods are more than nutrients—food has cultural, social, ethnic, and family messages.
- Biological functions may not be fully elucidated. Foods may be more beneficial than nutrients alone.
- Combinations of nutrients in foods can have different metabolic effects.
- Food processing and preparation influence nutritional values.

- Specific dietary patterns can be associated with reduced risk of specific diseases.
- FBDG are based on scientific knowledge and principles from science-based disciplines including:
  - nutrition;
  - food science;
  - behaviour;
  - communication;
  - agriculture.

Nutrition concepts

These concepts are generic and recommended by FAO. Country-specific concepts are developed.

❗ These concepts may be different to DRVs recommended for UK.

- Energy:
  - aims to prevent excess or deficiency;
  - promotes appropriate energy intakes by encouraging appropriate food choices;
  - physical activity is also encouraged.
- Protein:
  - *high quality protein:* 8–10% total energy;
  - *vegetable-based mixed diet:* 10–12% of total energy;
  - *elderly where energy intake low:* 12–14% total energy.
- Fat:
  - at least 15% energy from fats and oils;
  - childbearing age women at least 20% to ensure adequate essential fatty acids;
  - active non-obese <35% total energy (saturated fatty acids (SFA) <10%);
  - sedentary <30% total energy;
  - SFA <10% total fat.
- Carbohydrate:
  - main energy source >50%;
  - complex carbohydrate foods need to be cooked to be fully digestible;
  - sugar usually ↑ acceptability and energy density. Inversely related to fat intake. Moderate intakes compatible with a varied nutritious diet. No specific limit to sugar consumption but usually <10% total energy. UK government recommends that no more than 10% of total energy should come from non-milk extrinsic sugars.
- Micronutrients:
  - compounds with different metabolic activities;
  - essential for normal growth, development, and health;
  - important in preventing infectious and chronic diseases.

A summary of the recommendations for the UK is shown in Table 2.1.

Table 2.1 A summary of the dietary recommendations for the UK

Recommendation		Population group	Reason for recommendation
Fruit & veg	>5 x 80 g/day (400 g)	Adults	↓ Risk of some cancers, CVD, and other chronic conditions
Oily fish	>1 portion/week (140 g)	Adults	↓ Risk of CVD
Red and processed meat	Consider ↓ intake	All red meat consumers	↓ Cancer risk
NMES	<11% food energy[1]	All	NMES contribute to development of dental caries
Fat	<35% food energy	All	↓ Risk CVD, ↓ energy density of diet
Sat. fat	<11% food energy	All	↓ Risk CVD, ↓ energy density of diet
NSP	>18 g/day	Adults	Improve GI health
Alcohol	<3–4 units/day men <2–3 units/day women	Adults (>18 years)	↓ Risk liver disease, CVD, cancer, injury from violence or accidents
Salt	<6 g/day	Adults	↓ risk hypertension & CVD
Vitamins and minerals	DRVs	All	To prevent deficiencies and promote growth
Dietary vitamin D	DRV for young children, adults >65 years, pregnant and breastfeeding women. Others with limited sun exposure also require dietary vitamin D[2]	All	To prevent deficiency
Supplements	Vitamin D	Older adults, housebound or living in institutions, or who eat no meat or oily fish	To achieve adequate vitamin D status and ↓ risk of poor bone health

Adapted from the Nutritional Wellbeing of the British Population (2008) Scientific Advisory Committee on Nutrition.

[1] Energy consumed as food and drink excluding alcohol.

[2] Vitamin D supplements are also recommended for pregnant and lactating women.

The Eatwell Plate

'The Eatwell Plate' (Food Standards Agency, FSA) or plate model is the pictorial representation of FBDG in UK (Fig. 2.3). It is applicable to most people including minority ethnic groups, vegetarians, and people of all ages except children under 2 years. It is based on 5 food groups:

- Bread, other cereals, and potatoes.
- Fruit and vegetables.
- Meat, fish, and alternatives.
- Milk and dairy foods.
- Foods containing fat/foods containing sugar.

The FSA tips for healthy eating are as follows.

- Base your meals on starchy foods.
- Eat lots of fruit and vegetables.
- Eat more fish, include one portion of oily fish.
- Cut down on saturated fat and sugar.
- Try to eat less salt—no more than 6 g/day.
- Get active and try to be a healthy weight.
- Drink plenty of water.
- Don't skip breakfast.

A healthy diet should include:

- Meals based on starchy foods, such as bread, pasta, rice, and potatoes—including high fibre varieties where possible.
- Plenty of fruit and vegetables—at least 5 portions of a variety a day.
- Moderate amounts of milk and dairy products—choose low-fat options where possible.
- Moderate amounts of foods that are good sources of protein, such as meat, fish, eggs, beans, and lentils.
- Low amounts of foods that contain large amounts of fat or sugar.

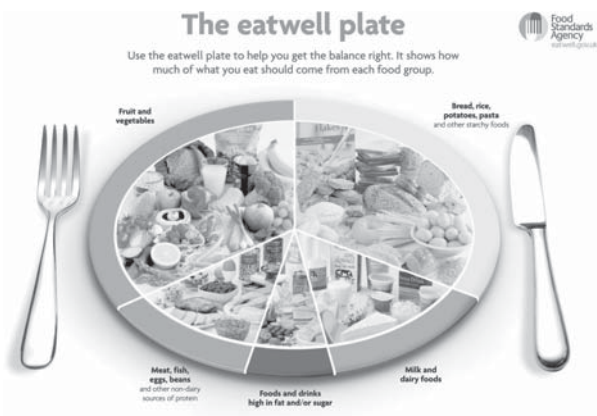

Fig. 2.3 The 'Eatwell Plate' model. (Reproduced with kind permission of the Department of Health, in association with the Welsh Assembly Government, the Scottish Government, and the Food Standards Agency in Northern Ireland.)

MyPyramid (steps to a healthier you)

MyPyramid (Fig. 2.4) is the pictorial representation of the USA FBDG (www.mypyramid.gov). It was released in April 2005 by the United States Department of Agriculture. It incorporates physical activity as an important part of good nutrition. Mypyramid is designed to be personalized and it is accompanied by a web-based personal nutrition plan. The design encompasses the following principles.

- *Activity:* the steps represent activity and the figure the importance of daily activity.
- *Moderation:* the bands represent moderation by narrowing as they reach the top. Foods with little or no fat and sugar are wider at the base and should be selected more frequently. Narrow bands represent foods with more fats and added sugars; the more active you are the more of these foods can be eaten.
- *Personalization:* this is represented by the figure on the steps, the slogan, and on the website.
- *Proportionality:* this is shown by the widths of the bands of the food groups. They are a guide as to how much a person should include in their diet. They are a general guide and not exact proportions.
- *Variety:* this is symbolized by the 6 bands that represent 5 food groups and oils. This shows that foods from each band are needed each day.
- *Gradual improvement:* this is shown by the slogan 'Steps to a healthier you'. Individuals are encouraged to take small steps each day to improve their diet and lifestyle.

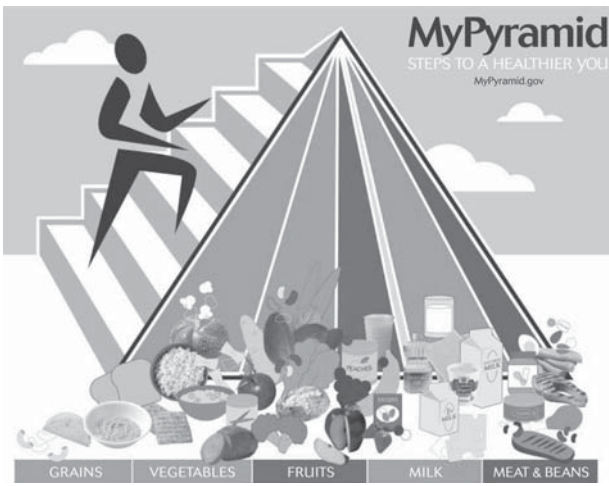

Fig. 2.4 MyPyramid food-based dietary guidance for USA (USDA Center for Nutrition Policy and Promotion).

Chapter 3

Current dietary patterns in the UK

Current dietary patterns

Information on current dietary patterns in the UK is taken from the National Diet and Nutrition Survey (NDNS). NDNS is a 4-year rolling programme commissioned by the FSA (www.food.gov.uk/science/dietarysurveys/ndnsdocuments/) and surveys people aged 1.5 years and older living in private households. Intakes from the first year of the programmes for adults aged 19–64 years (conducted between February 2008 and June 2009) and the 2000/01 survey are reported as appropriate.

❗ Previous adult NDNS surveys have slightly different methodologies ∴ be cautious when making comparisons.

Fruit and vegetable consumption

- In 2008/09 the mean fruit and vegetable consumption (including composite foods) was 4.4 portions/day.
- 35% of adults eat the recommended intake of 5 portions/day.
- In 2000/01 households receiving state benefit consumed on average 1 less portion of fruit and vegetables/day.

Vegetarianism/veganism

- 2% reported being vegetarian or vegan.
- In 2000/01 it was particularly popular in women aged 19–34 years.
- All avoided red meat, 92% avoid white meat, 29% avoided all animal products.

Supplement usage and dieting for weight loss

- In 2000/01 35% reported taking supplements with more women (40%) than men (29%) taking them. In the 1986/87 adult survey 17% women and 9% men reported taking supplements.
- 10% men reported dieting to lose weight compared with 24% women.

Macronutrient and energy intakes

See Table 3.1.

- The mean daily energy intake was below EARs for men and women.
- Average protein intakes were above the RNI.
- The mean percentage energy derived from non-milk extrinsic sugars (NMES) was above the RNI.
- The mean daily intake of non-starch polysaccharides (NSP) was below the recommended average intake of 18 g/day for men and women.
- The mean daily fat intake was <35%, the recommended intake.
- The mean energy derived from saturated fat was 12.5%, which is above the recommended level of 11%.
- The intake of trans fatty acids was below the recommended intake for men and women.

Table 3.1 Average intake of macronutrients and energy (2008/09) compared with DRVs (NDNS)†

Energy/nutrient	Men	Women
Total energy intake (MJ) (kcal)	9.48 (2255)	6.92 (1645)
% EAR	90%	86%
Protein (g)	88.4	66.3
% RNI	162%	145%
Total carbohydrate (g)	276	196
% Food energy	46.8%	45.5%
NMES (g)	73	52
% Food energy	13.0%	11.5%
DRV (% food energy)	<10%	<10%
NSP (g)	15.1	13.0
DRV (g)	18.0	18.0
Total fat (g)	81.8	61.6
% Food energy	35.5%	33.0%
DRV (% food energy)	<35.0%	<35.0%
Saturated fatty acids (g)	30.0	22.2
% Food energy	13.0%	12.0%
DRV (% food energy)	<11%	<11%
Trans fatty acids (g)	1.9	1.4
% Food energy	0.8%	0.8%
DRV (% food energy)	<2%	<2%

† Source: Bates, B., Lennox, A., and Swan, G. (2010). *The National Diet and Nutrition Survey*: Headline results from Year 1 of the Rolling Programme (2008/2009). Food Standards Agency, London.

Alcohol consumption

- Men are more likely to consume alcohol than women in each age group.
- 43% men and 30% women exceeded the recommend maximum intakes of 4 units/day for men and 3 units/day for women.
- 28% men and 15% women drank heavily; >8 units/day for men and >6 units/day for women.
- Women aged 16–24 years drank more heavily than older women.
- Men aged 50–64 years drank less heavily than other ages.

Vitamin and mineral intakes

- Mean intakes for all vitamins exceeded the RNI for men and women.
- Intakes of minerals were below the RNI for Zn in men aged 19–24 years.
- Mean intakes for all ages of women were below RNI for iron (Fe), magnesium (Mg), potassium (K), copper (Cu) and selenium (Se).
- 21% of women were below the RNI for Fe.
- 52% of women and 22% of men were below the RNI for Se.

Chapter 4

Nutrition assessment

Nutrition assessment: introduction

This chapter considers dietary assessment of populations and groups and nutrition assessment of the individual. Assessment of physical activity and energy expenditure can be found in 🕮 Chapter 5, 'Macronutrients and energy balance'.

Dietary assessment

Dietary assessment is an imprecise procedure; the imprecision can be minimized by using the appropriate technique and by an understanding of the errors implicit in the methodology. Dietary assessment is further hampered by the fact that, in assessing diet, it will change. Precision varies from very precise techniques, such as metabolic balance studies to the broad estimates of population studies. The methodology chosen must be appropriate for the nutrient/s that are being assessed and for the individual or population being assessed. The timing of the assessment is also important and must consider cultural variations, such as differences in the week (week day vs. weekend day), seasons (wet vs. dry season), and special occasions, e.g. Ramadan, Christmas.

This section gives a brief description of the methods used to assess diet; fuller descriptions and methods of assessing validity are described by Margetts and Nelson (1997)[1] and Gibson (2005)[2].

Country level assessment

Information is available on a national level on food production and agriculture; but this does not consider imports and exports. Food balance sheets (FBS, see Box 4.1), per country, are published annually by FAO (🖱 www.fao.org/es/ess/wfs.asp) and estimate a country's food supply (Table 4.1). Data are collated on domestic food production, food imports, and food taken from storage. Food *per capita* data can be derived as shown below and this can also be converted into nutrient values.

Per capita supply = total production + imports
+ adjustment for storage levels
– exports – animal use – seeds
– losses (storage, transport, and processing)

This information can be used to study the links between diet and disease and can aid the development of food-based dietary guidelines (FBDG) (see 🕮 Chapter 2, 'Dietary reference values and food-based dietary guidelines', p. 19), but the FBS only give information on availability not consumption. FBS give an estimate for the country as a whole and show no patterns of variation within the country.

[1] Margetts, B.M. and Nelson, M. (1997). *Concepts in Nutritional Epidemiology*, 2nd edn. Oxford University Press, Oxford.
[2] Gibson, RS. (2005). *Principles of Nutritional Assessment*, 2nd edn. Oxford University Press, Oxford.

Box 4.1 Food balance sheets (FAO methodology)

Strengths

- Information available in over 160 countries, since 1961.
- Data routinely collected in the countries.
- Does not entail implementation of special surveys.
- Assesses long-term trends.
- Provides global information on undernourishment.
- Provides information comparable across countries and across time.
- Is timely: estimates produced rapidly.
- Provides global information on dietary patterns.

Weaknesses

- Does not measure actual energy intake or utilization.
- Inaccuracy of country food balance data.
- In some countries estimates not consistent with socio-economic indicators.
- Underestimates actual energy needs in poor countries.
- Data on equality of food distribution not available.
- No disaggregation at subnational level.
- Does not take into account all food available for consumption, such as game or subsistence agriculture.

Household–based surveys

Examples of this type of survey are conducted on a regular basis by the Food Standards Agency (FSA) in the UK (National Diet and Nutrition Survey (NDNS) and dietary surveys that are part of the National Health and Nutrition Examination Surveys (NHANES). NHANES is conducted by the Center for Disease Control and Prevention (CDC), USA (www.cdc.gov). The limitations and strengths of each method are shown in Table 4.1.

Food account

Food account may also be known as budget household survey. The main respondent notes all food that is purchased and all food grown or received as gifts. It assumes no change in stocks. Foods taken out of the household may be recorded. Waste is assumed to be between 5 and 10%. The amount of different food groups at the household level is deducted from the price paid for each food group. The amount can be divided by the number of people living in the household according to their gender, age, or body size. Records are usually kept for 1–2 weeks, which may not reflect the full dietary cycle of the household. The degree of disaggregation of food codes affects the precision of the study, e.g. in Italy food is grouped into 46 groups, which is less precise than in the Netherlands, which uses 500 food groups for national surveys.

Inventory

This is similar to a food account with the addition of a larder inventory at the start and finish of the survey period. Over short periods the survey may be distorted as attention is drawn to larder items that would not normally be consumed.

Household record

Foods are weighed or estimated using household measures with an allowance for waste. An interviewer visits after breakfast and foods consumed at breakfast are recalled. Food for further meals is weighed or estimated. An afternoon or evening visit may be made to establish waste. This method is most useful in developing countries when most food is home produced and levels of literacy are low.

List recall

This survey is based on the recall of foods consumed in a household over a set period, usually 1 week. It does not estimate individual consumption.

Table 4.1 Strengths and limitations of household survey data*

Method	Strengths	Limitations
Food account	1. Cheap: data collected by government and readily available for analysis	i. Home foods only unless family members collect data.
	2. Representative: national sample	ii. Incomplete; may not include sweets, alcohol, soft drinks
	3. Possible subgroup analysis—by region, income, etc.	iii. No individual data
	4. Provides information on food consumption patterns	iv. Based on food composition tables see 'Food composition tables', 🕮 Chapter 1 (p. 10)
		v. No knowledge of change in food stocks
		vi. Bias of over purchasing especially in elderly or low income households
Inventory	1–3 plus:	i–iv plus:
	5. Considers changes in larder stocks	v. Larder inventory may distort usual purchasing patterns
	6. Measures actual home food consumption	
House-hold record	3, 5, and 6 plus:	i, iii, and iv plus:
	7. Used in societies with low levels of literacy	vi. Observer presence may distort normal patterns
	8. Can be modified to measure individual consumption	vii. Seasonal variation in food availability may limit comparisons between groups
List-recall	1–3, and 6 plus:	i–iv plus:
	9. Based on single interview	viii. Memory errors
	10. Measures food use	ix. Observer presence may bias responses
	11. Retrospective; reflects actual patterns	

* Adapted from Garrow, J.S., James, W.P.T., and Ralph, A. (1999) *Human Nutrition and Dietetics*., Table 17.1, p. 135. With permission from Elsevier.

Individual assessment

The assessment of an individual's diet is susceptible to many possible errors; these include under or over reporting by subjects, recall difficulties, measurement errors, coding and calculation errors. Estimation of portion sizes may also introduce inaccuracies. Assessment may be prospective or retrospective; the strengths and limitations of each method are summarized in Tables 4.2 and 4.3, and the steps in choosing a method are given in Fig. 4.1.

Prospective methods

Duplicate diet

Subjects weigh and record their food at the time of consumption and a duplicate of the diet is weighed and stored for direct chemical analysis; this method does not require the use of food composition tables. This method is usually used in metabolic units so the onus of weighing, etc., is not on the subject. Aliquot sampling and equivalent composite are both duplicate diet methods. In aliquot sampling a sample, usually 10%, is taken, rather than an exact duplicate. This is less wasteful, but introduces possible sampling errors. In equivalent composite assessment an investigator prepares a duplicate diet from the list of ingredients used by the subjects which is analysed chemically.

Weighed inventory

This method is widely used: subjects weigh and record all food prepared and waste. The major advantage of this method is that it does not rely on assumptions of portion size. Food composition tables are used to estimate nutrient intake from the records.

Household measures

This method is similar to the weighed inventory except that food portions are estimated. Photographs or household measures, e.g. spoons and cups, may be used to aid portion size estimation. This method requires less effort by the subject than the weighed inventory, but is more prone to error.

Retrospective methods

24-h recall

A trained interviewer guides the subject through their food intake over the previous 24 h. It is a quick method of dietary assessment, but cannot be used to classify a subject's usual intake as it is not necessarily representative of the subject's normal eating pattern.

Diet history

A diet history is an extension of a 24-h recall and gives more detailed information about the usual diet; it can typically take 2 h. The reliability of the results is very dependent on the skills of the interviewer.

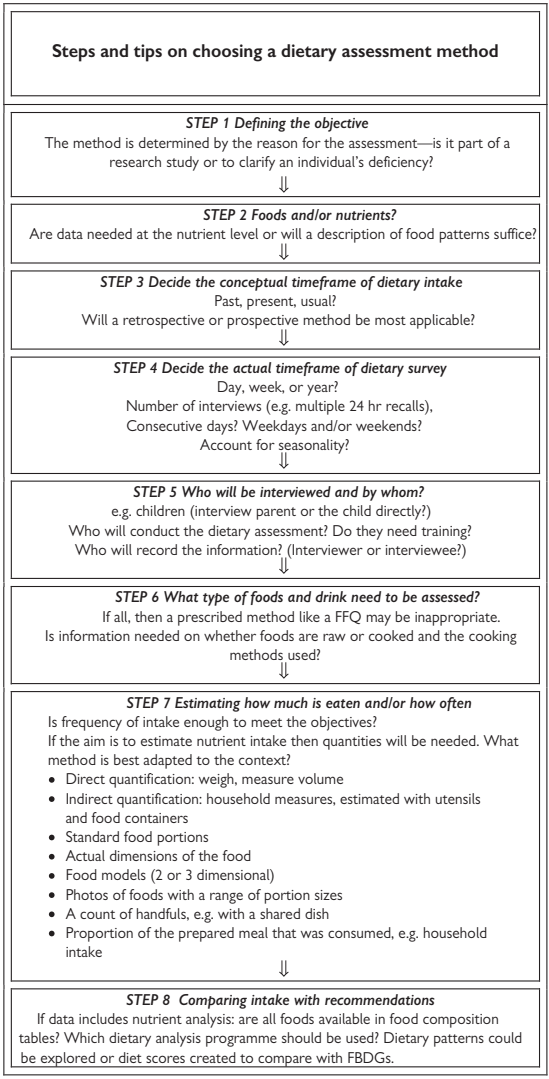

Fig. 4.1 Steps and tips on choosing a dietary assessment method.

Table 4.2 Strengths and limitations of prospective measurements of individual food consumption*

Measurement	Strengths	Limitations
General features	Current diet	Labour–intensive
	Direct observation of what is eaten	Requires numeracy and literacy skills
	Duration may be varied to meet requirements of estimates of food consumption or nutrient intake	Subjects need to be well motivated
		Usual consumption may change due to: • inconvenience of recording; • choice of foods that are easy to record; • beliefs of which foods are healthy or unhealthy
		Overweight subjects tend to under-report
		Coding and data entry errors are common
Duplicate diet	Direct analysis of nutrients (not dependent on food composition tables)	Very expensive
	Required for metabolic balance studies	Intense supervision required
		May not be usual diet
Weighed inventory	Widely used ∴ able to compare studies	Food composition tables are used
	Precision of portion sizes	
Household measures	No scales needed	Loss of precision compared with weighed inventory

* Reproduced from Garrow, J.S., James, W.P.T., and Ralph, A. (1999) *Human Nutrition and Dietetics*., table 17.1, p135. With permission from Elsevier.

Table 4.3 Strengths and limitations of retrospective measurements of individual food consumption*

Measurement	Strengths	Limitations
General features	Inexpensive	Biases caused by: • errors in memory, conceptualization of food portion sizes, perception; • presence of observer
	Quick	Daily variation not usually assessed
	Lower respondent burden	Dependent on regular eating habits
	Can assess typical or past diet	Food composition tables used to estimate nutrients
Diet history	Assesses usual diet	Over-reporting of foods believed to be healthy
24-h recall	Very quick	Prone to underestimate consumption due to omissions
	Can be repeated to gain measure of daily variation and improve precision	Single observation provides poor measure of individual intake
Food frequency questionnaires	Suitable for large-scale surveys	Requires validation in relation to reference measure
	Can be posted	Literacy and numeracy skills required if self-completed
	Short versions (or screeners) can focus on specific foods, e.g. fruit and vegetables	

* Adapted from Table 17.2, p.137 of Garrow, J.S., James, W.P.T., and Ralph, A. (2000). *Human Nutrition and Dietetics*, 10th edn. Churchill Livingstone, Edinburgh.

Food frequency questionnaire

Printed questionnaires are used and subjects (or interviewer) tick the category that approximates to their usual consumption of a list of foods, i.e. never eaten, eaten once a month, eaten once a fortnight, number of times eaten per week. This is quantified and intake is estimated. Food frequency questionnaire (FFQ) can be conducted via post. The number of foods can vary; sometimes only a few are used when assessing a food group or nutrient, e.g. fruit. These are sometimes called screeners. FFQs are often used in large surveys. It is necessary to validate FFQs against a more precise method such as weighed food intake. There are 3 main types of FFQ:

- *qualitative*—no portion size;
- *semi-quantitative*—standard portion size is used;
- *quantitative*—subjects are asked to record data on portion size.

Computer-based dietary assessment methods

Many computer-based or Internet based methods to assess dietary intake have been developed; usually based on FFQs and 24-h recalls e.g.

- Food Intake Recording Software System (FIRSSt).
- EPIC-SOFT (which is the widely used in Europe) (http://epic.iarc.fr/).
- Automated self-administered 24-h recall (ASA24) http://riskfactor.cancer.gov/tools/instruments/asa24/.

They aim to reduce the burden of data collection, data coding, and data management and to standardizing data collection.

New approaches using technologies to assess dietary intakes

New methods are currently being developed and tested to improve the accuracy of estimation of dietary intake using technologies such as mobile phones with a camera, Personal Digital Assistant (PDA) and microphones, e.g.

- Food intake visual and voice recognizer (FIVR).
- Mobile Phone Food Record project (MPFR).

Physical assessment

Observation of an individual may offer a gross assessment of nutritional status (see Chapter 25, 'Nutrition screening', p. 502).

- *Physical appearance*: e.g. pallor, emaciation, and hair changes may be indicative of long-term energy deficit; loose dentures and loose clothing may indicate recent weight loss; xanthoma or corneal arcus in some types of hyperlipidaemia; nail and teeth changes that occur in bulimia nervosa.
- *Oedema*: may be present following protein depletion.
- *Pressure sores or poor wound healing:* may be the result of immune response abnormalities or under nutrition.
- *Breathlessness:* may be the result of anaemia.
- *Mobility:* may be reduced following ↓ in muscle mass due to immobilization, which may present difficulties in food purchasing and preparation.
- *Mood*: e.g. apathy and depression, may be present in patients with eating disorders and other causes of under nutrition.

Biochemical and haematological assessment

Various parameters of nutritional status can be measured by analysis of serum, plasma, whole blood, urine, and faeces. Some measures are dynamic and reflect very recent changes and do not reflect long-term nutritional status. See sections on specific nutrients in 📖 Chapter 5, 'Macronutrients and energy balance' (p. 57), 📖 Chapter 6, 'Micronutrients' (p. 93), and 📖 Chapter 25, 'Nutrition screening' (p. 502).

- *Vitamin and mineral status:* may be assessed by circulating levels although deficiency of some micronutrients must be prolonged before blood levels are affected. For other micronutrients the body is very finely balanced and a dietary deficiency is balanced by ↑ mobilization from tissues, e.g. phosphate, or ↓ excretion.
- *Protein status*: may be reflected by serum proteins such as albumin, although levels do not truly reflect changes in protein status; levels are affected by other factors, e.g. infection, CRP levels. Serum transferrin and rapid turnover proteins, e.g. thyroxine, are reasonable markers of protein status, but are also affected by metabolic stress and may not be very specific.

Body composition

Body composition can be used to establish nutritional status especially when measuring adiposity. Current methods are based on a limited number of cadavers analyses; complete chemical analysis of whole cadavers is perhaps the only true 'gold standard' in body composition. Only a few cadavers have been analysed, and they varied in ethnicity, age, gender, and cause of death. While unsystematic cadaver selection may confound the validity of data obtained, the enormity and difficulty of the task involved, and significant ethical considerations, make these remarkable analyses unlikely to be repeated. Therefore, existing assumptions are used as reference for most modern techniques in body composition, ensuring a degree of consistency, at least, even if absolute accuracy cannot be wholly guaranteed. Modern body composition analysis may be perceived at five interactive levels: atomic, molecular, cellular, tissue/organ, and whole body.

Theoretical models

Theoretical models are used to derive reference data for the development of indirect methods, e.g. anthropometry. The body is divided into compartments; the classic 2 compartment model divides the body into fat mass (FM) and fat-free mass (FFM). FM consists of all extractable lipids and the remainder is FFM. Cadaver analysis was used to derive properties of FM and FFM.

- Density of FM = 0.901 g/ml.
- Density of FFM = 1.10 g/ml.
- Densities of FM and FFM are constant within and between individuals.
- FFM is assumed to be 73.8% water, 19.4% protein, and approx 7% mineral.

Other models require a combination of techniques to isolate the specified component of FFM.

- The 3-compartment model estimates FM + total body water (TBW) + 'dry' FFM (protein and mineral).
- The 4-compartment model is FM + TBW + protein + mineral.

The method used to derive each compartment will vary with more sophisticated methods being needed to differentiate between compartments. Table 4.4 summarizes the methods available. Multi-component models are expensive, time-consuming and difficult to use in field conditions; so alternative techniques may be more appropriate.

Direct methods

Until the development of *in vivo* neutron activation analysis (IVNAA) the only direct method of body composition analysis was cadaver analysis. In IVNAA the body is exposed to neutron irradiation. Of particular nutritional interest is that the measurement of nitrogen by IVNAA is used to determine total body protein. IVNAA is expensive and, as the body is irradiated, there are ethical issues, especially for use in children which limits its usefulness.

Table 4.4 Summary of methods for the determination of body composition*

Method	Accuracy	Cost	Radiation	Time	Convenience for subject
Cadaver analysis	+++	–		–	
IVNAA	+++	–	–	++	++
Densitometry	++	+		++	+/–
Dilution	++	+/–	(–)	+	++
TBK	++	–		++	++
DEXA	+++	+/–	–	++	++
CT scanning	++	–	–	++	++
MRI scanning	++	–		++	+
Anthropometry	+	+++		++	+
Infrared interactance	+	++		++	++
BIA	+	+		+++	+++
TOBEC	+	–		++	++
Urinary metabolites	+	+		–	–

+++, Excellent; ++, very good; +, good; +/– reasonable; – bad;

IVNAA, *In vivo* neutron activation analysis; TBK, total body potassium; DEXA, dual energy X–ray absorptiometry; CT, computer-assisted tomography; MRI, magnetic resonance imaging; BIA, bioelectrical impedance analysis; TOBEC, total body electrical conductivity.

* Reproduced from Gibney, M.J., Lanhan-New, S.A., Cassidy, A., and Vorster, H.H. (2009) *Introduction to Human Nutrition*, 2nd edn. Permission requested from Wiley Blackwell.

Independent methods

Each individual method can be used in multi-component models or used individually, provided they are applied appropriately and that the limitations are recognized.

Hydro-densitometry or under-water weighing

Body weight (BWt) is measured in air and during a procedure involving water displacement with the subject submerged, and with a correction for lung volume, to provide under-water weight (UWW). Body volume (BV) is calculated as BWt – UWW, and density (d) = BWt/BV, from which body fat and FFM may be obtained, as above.

Air-displacement plethysmography (ADP)

BV can also be determined using air displacement plethysmography (ADP), consisting of a chamber in which the subject sits comfortably and breathes normally. ADP measures chamber volume with and without the subject present by generating and measuring small pressure changes. BV is assessed by the difference in pressure changes with corrections for air in the lungs and adjacent to skin. To derive percentage body fat body density is substituted into appropriate equations, e.g. Siri.

Siri formula: % Body fat = (495/body density) – 450.

As above, BV and weight obtained during both these techniques are also integral to the 3- and 4-component models if required.

Isotope dilution techniques

Total body water (TBW) may be measured using isotope dilution techniques in which labelled water is administered. Deuterium (^{2}H) dilution is the technique of choice as it is a safe stable isotope, occurs naturally in the water. Oxygen-18 (^{18}O) is also a stable isotope and potentially more accurate for TBW estimation, however, ^{18}O-labelled water is more expensive and less readily available than ^{2}H-labelled water. In health, hydration of FFM is relatively constant, usually between about 72 and 74%, depending on factors, such as age.

❗ Caution is necessary in patients with abnormal hydration, e.g. liver and kidney diseases.

Dual-energy X-ray absorptiometry (DXA)

X-rays at two distinct energies are used to differentiate bone, fat and fat-free soft tissue (FFST), and so may be considered a specific three-component model. Therefore, dual-energy X-ray absorptiometry (DXA) can provide estimates of FM and FFM which quite accurate and precise, at least in health. DXA may be less accurate or precise in extremes of body dimensions (e.g. obesity and anorexia) and disproportionate changes in body chemistry (e.g. oedema), and may not assess changes accurately (e.g. in weight loss).

Although DXA directs ionizing radiation, the exposure is considered relatively low, depending on particular manufacturer and model, making it relatively safe. However, DXA should not be used in pregnant women and there may be ethical issues in children.

Alternative methods

There are a number of methods that can be utilized to gather important body composition information in health and disease. However, caution is advised to ensure that estimates provided are appropriate to requirements, otherwise substantial errors are possible. Many indirect techniques depend on assumptions that may be uncertain, leading to such errors.

Bioelectrical impedance analysis

An electrical current flows predominantly through tissues containing water and ions, but not through fat, which is an insulator therefore, body resistance or impedance (Z) was originally used as an index of TBW. However, because of complex differential electrical properties of tissues, and lack of uniformity of body shape and dimensions, an essentially empirical approach was adopted. Whole body impedance was regressed against reference measures of TBW, which was then extended to FFM and fat, producing equations claimed to estimate or 'predict' body composition.

In practice, bioelectrical impedance analysis (BIA), which is now in widespread use, applies measures of impedance between hand and foot, usually, but foot to foot or hand to hand equipment is also widely available.

Impedance is commonly adjusted for an index of height (often Ht^2/Z) and then a derived equation to predict body composition. Often with other anthropometric measures incorporated, ∴ there are a very large number of different BIA prediction equations available.

❗ Caution is needed when using or interpreting BIA, as prediction equations are often originated, and applied inappropriately and without sufficient understanding.

Although useful for large scale epidemiology and field studies, due to portability and low cost, BIA may be relatively ineffective in different population groups and certain disease states because it may be insensitive to underlying variability between individuals (e.g. males vs. females, adults vs. children) or fundamental changes caused by disease.

Total body potassium counting

Total body potassium (TBK) can be used to provide estimates of body composition, as TBK is present only in FFM with 98% in cells and its concentration assumed to be relatively constant, but slightly different in males and females. A known proportion (0.012%) of natural potassium occurs as a radioactive isotope, ^{40}K, the radioactive decay of which produces γ-emissions detectable using whole body counters. Therefore, it is possible to estimate TBK and then to derive FFM from it in a non-invasive process that may take only 20–30 min, depending on the type of counter. Whole body counters are rarely available, are expensive and affected by variations in body shape and dimensions, and environmental contamination.

Imaging techniques: computed tomography, magnetic resonance imaging and ultrasound

Assessments of segmental and whole body tissues by computed tomography (CT) and magnetic resonance imaging (MRI) are very accurate, and extremely valuable measures in their own right, and may complement body composition estimates from other techniques. Estimates of gross body composition (e.g. body fat) are also possible, but these generally require the use of assumptions. MRI and CT are extremely expensive and CT exposes the subject to relatively high doses of radiation, which has major ethical considerations.

Ultrasound (US) is a safe technique for complementing body composition methodology by providing relative dimensions of body tissues, e.g. adipose tissue and muscle, particularly tissue depths. Recent developments enable three-dimensional (3-D) assessments of tissues and organs.

Urinary metabolites

Excreted metabolic end products can be utilized to provide estimates of FFM. 24-h collections of urinary nitrogen (for protein turnover), creatinine (the constant spontaneous degradation product of creatine, found only in muscle) and *N*-methyl-histidine (N-MH; specific end product of muscle protein degradation) have all been used as indices of FFM. As urinary excretion is variable and physical losses of urine are known to occur, resulting in major source of error, completeness of 24-h urine collection may be confirmed by use of orally administered para-aminobenzoic acid (PABA). Creatine and N-MH measurement require the subject to be essentially on a meat-free diet. Nitrogen balance is representative of FFM, but also requires determination of faecal nitrogen. This technique is rarely used outside metabolic units.

Anthropometry and derived prediction equations

Body mass index (BMI) can be calculated from weight (Wt) and height (Ht); $BMI = Wt/Ht^2$. BMI is in itself precise and informative particularly at the group level, but Wt and Ht, or BMI are not considered accurate or precise enough to be used independently to predict or estimate body composition, especially for individuals.

Skin-fold thicknesses obtained at various sites are used as indices of subcutaneous adipose tissue and can also be integrated to give predictions of body fat, but these estimates are not considered accurate or precise enough for estimating body composition. (See 🕮 this Chapter, 'Anthropometry', p. 50.)

Surface imaging

Photonic scanning provides 3-D imaging of the body surfaces, which is useful in terms of body shape and dimensions, and informative in health and disease, but is not considered accurate or precise enough to estimate or predict body composition.

Miscellaneous techniques

Total body electrical conductivity (TOBEC), in which a conductive coil surrounds a subject, uses induced electrical properties of the body to assess body composition, although now it is rarely used in humans.

Near infrared interactance (NIRI) has been applied to body composition based on the differential response of a reflected infra-red beam to fat and FFM, but there are serious questions about its validity because of the uncertainty of assumptions needed to interpret subcutaneous adipose tissue characteristics in terms of whole body composition.

Subcutaneous adipose tissue topography (Lipometer) works in similar way as NIRI, but uses optical light, and also produces uncertain body composition estimates for similar reasons.

Anthropometry

Anthropometry simply means the measurement of man and involves the measurement of height, weight, skin-fold thicknesses, circumferences and various lengths and breadths of the body. These techniques require relatively cheap equipment and are ∴ widely used in clinical practice. The techniques for some anthropometric measurements are shown in Tables 4.5 and 4.6.

Children

See 🕮 Chapter 13, 'Growth reference charts' (p. 238), 🕮 Appendix 2 (p. 764).

WHO growth charts

The WHO produced new growth charts in 2006 for 0–5 years, representing growth standards of healthy breastfed children in optimal conditions using data collected in the WHO Multicentre Growth Reference Study. These were extended in 2007 for children and adolescents aged 5–19 years (℘ www.who.int/childgrowth/en/). The WHO Reference 2007 is a reconstruction of the 1977 National Center for Health Statistics (NCHS)/WHO reference. It uses the original NCHS data set supplemented with data from WHO child growth standards sample for <5 years.

UK–WHO 0–4 years charts

New UK–WHO Growth Charts for children from birth to 4 years of age were introduced in 2009 (England) and in 2010 (Scotland). The new charts combine UK90 and WHO data, and replace previous UK90 charts for ≤4 years. New features include an adult height predictor, a BMI conversion chart, and guidance on gestational age correction. The UK90 charts should still be used for all children aged >4 years. They are included in the UK Personal Child Health Record (PCHR) or 'red book' issued to each newborn (see 🕮 Appendix 2, p. 764). See ℘ www.rcpch.ac.uk/Research/UK-WHO-Growth-Charts. BMI percentiles should be used to identify overweight and obesity, and the new chart is recommended for routine clinical diagnosis of growth faltering. Overweight is classified as ≥91st centile and obesity ≥98th centile of the UK-WHO charts. Epidemiological studies use an internationally acceptable definition to classify prevalence of child overweight and obesity[3] or the WHO estimates of overweight from WHO growth charts for BMI-for-age.

Z-score

Anthropometric measurements can be expressed as *Z*-scores. A *Z*-score is the standard deviation (SD) score; the deviation of the value for an individual from the median value of the reference population divided by the SD for the reference population:

$$Z\text{-score} = \frac{\text{(observed value} - \text{median reference value)}}{\text{SD reference population}}$$

[3] Cole, T.J., Bellizzi, M.C., Flegal, K.M., and Dietz, W.H. (2000). Establishing a standard definition for child overweight and obesity worldwide: international survey, *Br. Med. J.* **320**, 1240–3.

Table 4.5 Standardized anthropometric measurements: circumferences

Site	Anatomical reference	Measurement
Waist	Narrowest part of torso	Apply tape snugly around waist. Take measure at end of natural expiration
Hip (buttocks)	Maximum posterior extension of buttocks	Apply tape snugly around buttocks
MAC (biceps)	Midpoint between acromion process of scapula and olecranon process of ulna	Arms hanging freely with palms facing thighs

Adapted from Heyward, V.H. and Stolarczyk, L.M. (1996). *Applied Body Composition Assessment*, Table 2.1 (pp. 28–9) and S.1 (pp. 71–4). (Copyright 1996, Human Kinetics).

Table 4.6 Standardized anthropometric measurements: skin-fold measurements*

Site	Direction of fold	Anatomical reference	Measurement
Subscapular	Diagonal	Inferior angle of subscapular	Fold is natural cleavage line just inferior to interior angle of scapula with caliper applied 1 cm below
Supra-iliac	Oblique	Iliac crest	Fold is grasped behind to mid-axillary line and above iliac crest
Triceps	Vertical	As circumference above	Midpoint is measured and fold is 1 cm above line on posterior aspect of arm
Biceps	Vertical	Biceps brachii	Fold is lifted over the belly of biceps at line marked for triceps; caliper is applied 1 cm below fingers

* Adapted from Heyward, V.H., and Stolarczyk, L.M. (1996). *Applied Body Composition Assessment*, Table 2.1 (pp. 28–9) and S.1 (pp. 71–4). (Copyright 1996, Human Kinetics). Permission requested from Dr Timothy Lohman.

Adults

Weight

Body weight is a crude measure of body composition; scales require regular calibration and servicing, and weight may vary between scales. Monitoring of weight over a period can be a useful indicator of nutritional status.

Height

- A stadiometer is used or the subject is measured against a wall. The floor should be uncarpeted.
- The subject should be barefoot and their weight evenly distributed between both feet.
- Arms should hang loosely.
- Heels should be together, touching the vertical board or stadiometer. Head, scapula, and buttocks should be touching the vertical board or wall.
- Hold the head erect with eyes focused straight ahead.
- Subject should inhale.
- The rod is lowered to the most superior point, compressing hair.

Surrogate measures of height

- Recall height may be used in bed-bound patients. This tends to overestimate height by 7 mm, but this does not affect BMI categorization.
- Ulna length can be measured by bending the left arm across the chest with the palm facing inwards and the fingers pointing to the shoulder. The measurement is taken between the central and post prominent parts of the styloid process and the tip of the olecranon (equations for the predicted height are shown in Box 4.2).
- Knee height is measured in sitting subjects. The knee and ankle are bent to 90° and the observer's hand is placed flat on the thigh. The tape measure is held between the fingers and the height measured to the floor, on the lateral plane of the leg, in the same plane as the lateral malleolus (prediction equations are given in Box 4.2).
- Demispan can be measured in patients sitting in a chair or supine. The right arm is raised until it is horizontal with the wrist in natural flexion and rotation. The tape is placed between the middle and ring finger and runs smoothly along the arm. The measurement is taken from the tip of the finger to the centre of the sternal notch (prediction equations are shown in Box 4.2).

Body mass index

BMI reflects body fat stores and is calculated as: BMI = Wt (kg)/Ht $(m)^2$.

BMI is correlated to the risk of obesity and underweight-associated morbidity. Overweight subjects have an ↑ risk of associated health problems, this risk ↑ with ↑ BMI. The WHO cut-offs for the definition of overweight and obesity are given in Table 4.7 (see 🕮 Chapter 21, p. 412).

BMI is a useful clinical and epidemiological tool. Appendix 2 gives BMI calculator. It should be used with caution in the elderly and in muscular subjects. Cut-off values for Asian populations are likely to be lower but this is still being debated by WHO and the International Obesity Task Force (℔ www.iotf.org).

Box 4.2 Prediction equations

Equations for the prediction of height from ulna length

- Men (<65 years) Predicted height (cm) = 79.2 + 3.60 ulna length (cm)
- Men (≥65 years) Predicted height (cm) = 86.3 + 3.15 ulna length (cm)
- Women (<65 years) Predicted height (cm) = 95.6 + 2.77 ulna length (cm)
- Women (≥65 years) Predicted height (cm) = 80.4 + 3.25 ulna length (cm)

❗ Equations only validated for ulna length measured on the left side.

Equations for the prediction of height from knee height

- Men (18–60 years) Predicted height (cm) = 71.85 + (1.88 x knee ht (cm))
- Men (60–90 years) Predicted height (cm) = 59.01 + (2.08 x knee ht (cm))
- Women (18–60 years) Predicted height (cm) = 67.85 + (1.87 x knee ht (cm))
- Women (60–90 years) Predicted height (cm) = 62.25 + (1.91 x knee ht (cm))

❗ Equations only validated for knee height measured on the left side.

Equations for the prediction of height from demispan

- Men (16–54 years) Predicted height (cm) = 68 + (1.3 x demispan (cm))
- Men (>55 years) Predicted height (cm) = 71 + (1.2 x demispan (cm))
- Women (16–54 years) Predicted height (cm) = 62 + (1.3 x demispan (cm))
- Women (>55 years) Predicted height (cm) = 67 + (1.2 x demispan (cm))

❗ Equations only validated for demispan measured on the right side.

Table 4.7 WHO cut-offs for BMI

BMI	Weight status	Risk of co-morbidities
Below 18.5	Underweight	Low
18.5–24.9	Normal	Average
25.0–29.9	Overweight	Increased
30.0–39.9	Obese	Moderate–severe
Above 40	Very obese	Severe

Circumferences

Waist circumference and waist–hip ratio have been proposed as measures of risk of obesity–associated morbidity. The WHO cut–offs for waist circumference are shown in Table 4.8.

❗ The precise sites used will vary depending on which manual is followed. The examples in this chapter may differ from those used by the International Society for the Advancement of Kinanthropometry (ISAK) or WHO.

Skin-fold thickness measurements

Most of the body's fat is stored subcutaneously. The thicknesses of skin-folds at specific sites are measured (at least 3 measurements are needed at each site) by calipers (Figs 4.2 and 4.3) and can be used to estimate total subcutaneous fat. The most commonly used sites are subscapular, supra-iliac, biceps, and triceps. Total skin-folds from these sites can be substituted into prediction equations to give an estimate of % FM. The most commonly used equations are those derived by Durnin and Womersley (1974).[4] Equations that are appropriate for specific ages and ethnic groups are available. Skin-fold measurements are cheap and quick, but the technique requires training and skill. Ideally practitioners should attend an accredited course, e.g. ISAK (🖱 www.isakonline.com).

Arm muscle measurement

Midarm circumference (MAC) can be measured as shown in Table 4.5. It is assumed that the arm is a cylinder of muscle covered by adipose tissue and that the double thickness of the fat layer is measured by triceps skin-fold (TSF) thickness. Midarm muscle circumference (MAMC) can be calculated:

$$\text{MAMC (cm)} = \text{MAC} - (3.14) \times \text{TSF (cm)}.$$

This estimate of FM and FFM is used clinically to monitor nutritional status. Appendix 2 gives reference values.

Table 4.8 WHO waist circumference cut-offs and risk of associated metabolic complications

	Increased	Substantially increased
Men	≥94 cm	≥102 cm
Women	≥80 cm	≥88 cm

[4] Durnin, J.V.G.A, and Womersley, J. (1974). Body fat assessed from total body density and its estimation from skinfold thickness: measurements on 481 men and women aged from 16 to 72 years. *Br. J. Nutr.* **32**, 77–97.

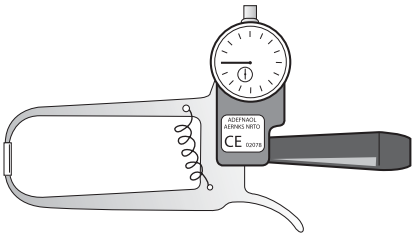

Fig. 4.2 Diagram of calipers.

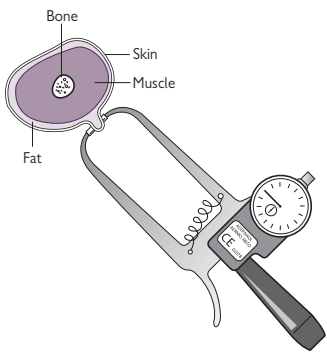

Fig. 4.3 Diagram of skin-fold measurement.

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Chapter 5

Macronutrients and energy balance

Macronutrients: introduction

The macronutrients are protein, fat, and carbohydrate, and they are required in gram amounts. They are major sources of energy as well as providing essential nutrients such as amino acids.

Protein

Protein provides approximately 10–15% of the energy in the diet. Protein is essential for numerous structural and functional purposes and is essential for growth and repair of the body. In adults approximately 16% of body weight is protein. 43% of this is muscle, 15% skin, and 16% blood. Protein is in a constant state of flux in the body with protein being synthesized and degraded continuously.

Protein flux (Q) can be described by the following equation:

$$Q = I + D + S + O$$

where I = intake, D = degradation, S = synthesis, and O = oxidation to CO_2 and urinary nitrogen.

Function

Protein has numerous functions in the body. Examples of the different functions of protein are as follows.

- *Structural:* Protein is important for the structure of the body and about half of the body's protein is in structural tissues such as skin and muscle. These structural proteins are collagen (25% of the body's protein), actin, and myosin.
- *Transport:* Proteins act as transport carriers in the blood and body fluids for many molecules and nutrients, e.g. haemoglobin, lipoproteins.
- *Hormonal:* Hormones and peptides are proteins or amino acid chains, e.g. insulin, pancreatic polypeptide.
- *Enzymes:* All enzymes are proteins. Extracellular enzymic proteins include the digestive enzymes, e.g. amylase. Intracellular enzymes are involved in metabolic pathways, e.g. glycogen synthestase.
- *Immune function:* Antibodies are protein molecules. Proteins are also involved in the acute phase response to inflammation.
- *Buffering function:* The protein albumin acts as a buffer in the maintenance of blood pH.

Structure

Proteins are macromolecules consisting of amino acid chains. Amino acids are joined to each by peptide bonds (Fig. 5.1). Amino acids form peptide chains of various lengths from two amino acids (dipeptide), 4–10 peptides (oligopeptides) and more than 10 amino acids (polypeptides). Reactive side groups of the amino acids combine to form links between amino acids in the chain and other peptide chains. The polypeptides form β pleated sheets or α helices. Polypeptides fold and cross-links form between amino acids to stabilize the folds. Proteins are formed by the combination of polypeptides. These cross-links give the peptide a distinctive function and shape (Fig. 5.2). There are approximately 20 amino acids and each has a

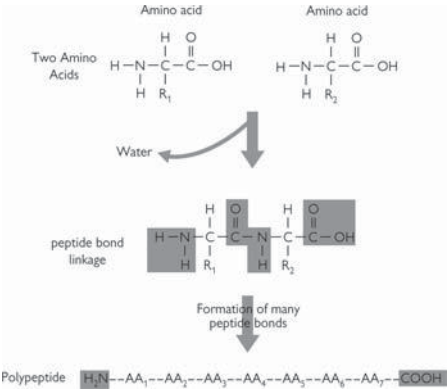


Fig. 5.1 Formation of a polypeptide.

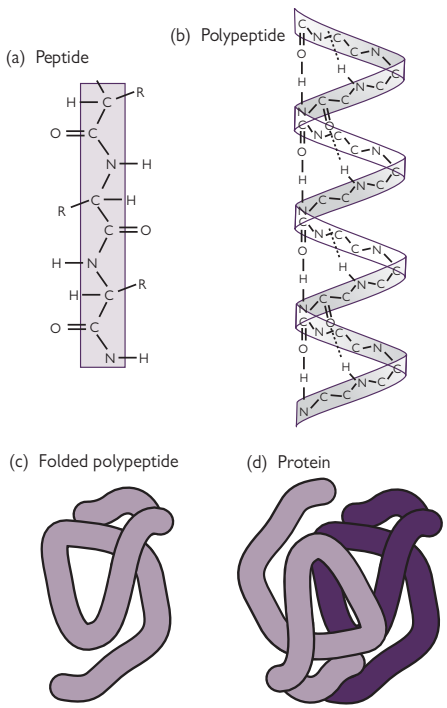


Fig. 5.2 Formation of a protein.

different side group, size, and different properties, e.g. pH, hydrophilic or hydrophobic. These properties are used in the analysis of amino acids.

Indispensable (essential) amino acids

Some amino acids can be synthesized by the body but others must be supplied by the diet. These are known as indispensable or essential amino acids; there are eight essential amino acids (Table 5.1). Some amino acids are only essential in specific circumstances. In childhood seven other amino acids are essential that are not essential in adults (arginine, histidine, cysteine, glycine, tyrosine, glutamine, proline). These amino acids are essential in children because they are required in amounts larger than can be synthesized because of high demand, immature biological pathways, or a combination. Conditionally indispensable or essential amino acids only become essential in circumstances when the requirement is ↑ e.g. glutamine.

Requirements

The amino acid content of a protein determines its biological value. Proteins that contain all the indispensable amino acids in sufficient quantities have high biological value. High biological value proteins are from animal sources, e.g. meat, eggs, milk, dairy products, and fish. If one or more indispensable amino acids are absent from a protein it will have low biological value. Generally plant proteins are of low biological value. The indispensable amino acid that is in shortest supply is known as the limiting amino acid. By combining foods with low biological value it is possible to provide all indispensable amino acids in the diet; this is important in vegan diets. For example, the limiting amino acid in wheat is lysine and in pulses it is methionine. A diet combining wheat products such as bread with pulses will provide all the indispensable amino acids, e.g. pitta bread and dhal.

Table 5.1 Classification of amino acids

Indispensible/ essential amino acids	Indispensible (conditionally essential) amino acids	Dispensible (non-essential) amino acids
Leucine (Leu)	Tyrosine (Tyr)	Glutamic acid (Glu)
Isoleucine (Ile)	Glycine (Gly)	Alanine (Ala)
Valine (Val)	Cysteine (Cys)	Aspartic acid (Asp)
Phenylalanine (Phe)	Arginine (Arg)	
Threonine (Thr)	Proline (Pro)	
Methionine (Met)	Histidine (His)	
Tryptophan (Trp)	Glutamine	
Lysine (Lys)	Serine (Ser)	
	Asparagine (Asn)	

As already stated, protein is constantly being turned over; 3–4 g proteins are turned over per kg of body weight per day. Each day 10–15 g of nitrogen are excreted in urine (6.25 g protein is equivalent to 1 g nitrogen). Small amounts are lost in faeces and skin. When nitrogen (protein) intake equals nitrogen excretion the body is said to be in nitrogen balance. Healthy adults will be in positive nitrogen balance. Nitrogen balance studies have been used to derive the recommended requirements that are shown in Table 5.2.

Deficiency

If energy intake is insufficient, protein will be degraded to produce energy; ∴ protein deficiency can occur when the diet does not provide enough protein or energy or a combination of both. Protein energy malnutrition (PEM) is a major cause for concern in developing countries (see 🕮 Chapter 20, 'Global nutrition', p. 389), but does occur in the UK amongst at risk groups. These include immunocompromised individuals (e.g. AIDS), anorexia, and cancer patients with cachexia. Mild PEM is fairly common amongst surgical or elderly hospital patients. Protein deficiency can also occur as the result of ↑ losses in renal disease, ↑ catabolism in trauma, burns or sepsis, or malabsorption. Protein deficiency results in muscle wasting, stunted growth, poor wound healing, and susceptibility to infection, oedema, and fatty liver.

Sources of dietary protein

In the typical UK diet 60% of protein intake has high biological value. High biological protein is supplied by meat and meat products, fish, eggs, and milk and dairy products (Table 5.3). Plants such as cereals and pulses supply proteins of low biological value.

Table 5.2 Recommended nutrient intake of protein for all age groups and average daily intakes of protein of adult men and women in UK*

Age	Weight (kg)	RNI (g/day)
Children (both sexes)		
0–3 months	5.9	12.5
4–6 months	7.7	12.7
7–9 months	8.8	13.7
10–12 months	9.7	14.9
1–3 years	12.5	14.5
4–6 years	17.8	19.7
7–10 years	28.3	28.3
Males		
11–14 years	43.0	42.1
15–18 years	64.5	55.2
19–50 years	74.0	55.5
50+ years	71.0	53.3
Females		
11–14 years	43.8	41.2
15–18 years	55.5	45.4
19–50 years	60.0	45.0
50+ years	62.0	46.5
Additional RNI required for females		
During pregnancy		+6.0
Lactation: 0–6 months		+11.0
Lactation: 6+ months		+8.0
Adults	**Average daily intake UK (g/d)**	
Men	88.4	
Women	66.3	

* Source for RNIs, Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. HMSO, London. Source for average daily intakes for adults, Bates, B., Lennox, A., and Swan, G. (2010). *The National Diet and Nutrition Survey: Headline results from Year 1 of the Rolling Programme (2008/2009)*. Food Standards Agency, London.

Table 5.3 Contribution of food sources to protein intake*

Food group	% Daily intake
Meat and meat products	38
Chicken, turkey and dishes	12
Cereals and cereal products	22
Bread	11
Milk and milk products	16

* Bates, B., Lennox, A., and Swan, G. (2010). The National Diet and Nutrition Survey: Headline results from Year 1 of the Rolling Programme (2008/2009). Food Standards Agency, London.

Fats

Fats are often referred to as lipids. Lipids are described by chemists as substances that are poorly soluble or insoluble in water but are soluble in organic solvents. Fat is the term most often used when discussing foods and lipids metabolism. Over 95% of dietary fats are triglycerides (triacylglycerols); other types of fat include cholesterol, phospholipids, sterols, and carotenoids.

Function

The functions of fat in the diet are:

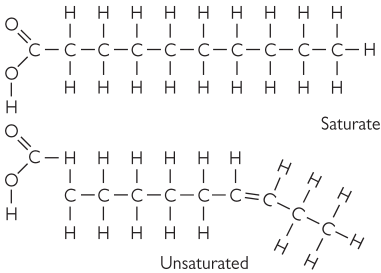
- Energy source—fat provides 37 kJ (9 kcal) per gram.
- Fat provides essential fatty acids.
- Fat is a carrier for fat soluble vitamins A, D, E, and K.
- ↑ Palatability by improving taste perception and appearance of food.
- Some fats are important constituents of cell membranes and can be converted to biologically active compounds, such as steroid hormones, interleukins, thromboxanes, and prostaglandins.
- Cholesterol is converted to bile acids, which are important in digestion.

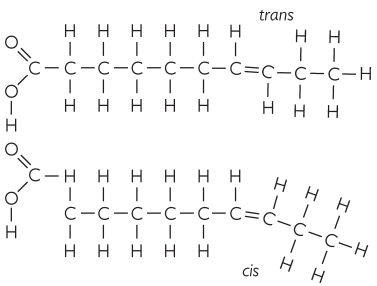
Fatty acids

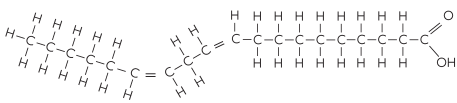
Fats consist of fatty acids that have carbon chains containing up to 22 carbon molecules in the chain. The type of fatty acid attached to the glycerol molecule determines its physical properties, nutritional function, and physiological function. Hydrogen is added to fatty acids to make them more solid when manufacturing some food products such as vegetable spreads; this process is known as hydrogenation.

Fatty acids are carbon molecules with a methyl group at one end and a carboxyl acid at the other (Fig. 5.3). They can have chains of 4–22 carbon molecules although most have 16–18. Hydrogen atoms are attached to the carbon chain; the number of hydrogen atoms determines the degree of saturation (with hydrogen atoms) of the fatty acid. A fatty acid with hydrogen atoms on every arm is 'saturated'. Unsaturated fatty acids contain double carbon bonds where there is no hydrogen (Fig. 5.3). If there is only one double bond the fatty acid is monounsaturated. When more than one double bond is present the fatty acid will be polyunsaturated.

Fatty acids have a common name, e.g. linoleic acid, a systematic name, and a notational name. The systematic name reflects the number of carbon atoms, and the number of double bonds, so that linoleic acid becomes octadecadienoic acid. This represents 18 carbons (octadeca-) and two double bonds (di-). The notational name for linoleic acid is 18:2 *n*6 or 18:2 ω 6; again this represents 18 carbon atoms and two double is now also represented. The position is relative to the methyl (or omega) end of the carbon chain. Linoleic acid has its first double bond between the sixth and seventh carbons. Common names, systematic names, and notational names are shown in Table 5.4.







Linoleic acid, a polyunsaturated fatty acid.
Both double bonds are cis

Fig. 5.3 Structure of fatty acids.

Saturated fatty acids

Saturated fatty acids (SFA) contain carbon atoms linked by single bonds and hydrogen on all available arms; they have a relatively high melting point and tend to be solid at room temperature. SFA are obtained from animal storage fats and their products, e.g. meat fat, lard, milk, butter, cheese, and cream. Fats from plant origin tend to be unsaturated with the exception of coconut oil and palm oil. Some manufactured margarines and spreads contain significant amounts of SFA. Plasma low-density lipoprotein (LDL) cholesterol, and ∴ plasma cholesterol, tends to be raised by SFA. High intakes of SFA are associated with atherogenesis and cardiovascular disease.

Monounsaturated fatty acids

Monounsaturated fatty acids (MUFA) contain only one double bond and are usually liquid (oil) at room temperature. Olive oil and rapeseed oil are the most concentrated dietary sources of MUFA. MUFA are present in many foods including meat fat and lard. Dietary MUFA does not raise plasma cholesterol and lowers LDL lipoprotein without a detrimental effect on high density lipoproteins (HDL).

Polyunsaturated fatty acids

Polyunsaturated fatty acids (PUFA) contain two or more double bonds and are liquid at room temperature. They are easily oxidized in foods and in the body. PUFA are involved in the metabolism of cholesterol, are components of phospholipids in cell membranes, and are precursors of biologically active compounds such as prostaglandins, interleukins, and thromboxanes. Therefore they have a vital role in the immune response, blood clotting, and inflammation. PUFA are derived from the essential fatty acids linoleic acid (*n*6 or ω6) and α-linoleic acid (*n*3 or ω3) and are ∴ divided into omega 3 (ω3) or omega 6 (ω6) groups of PUFA. Essential fatty acids (EFA), eicosapentaenoic acid (EPA), and docosahexaenoic acid (DHA) are important in neural development of the fetus and infant. PUFA occur as *cis* or *trans* forms depending on the way the hydrogen atoms are arranged. In *cis* formation the hydrogen atoms are bonded to either end of the double bond on the same side. And in the *trans* form the hydrogen atoms are on opposite side (Fig. 5.3). Most naturally occurring fats are in the *cis* form.

Omega (ω) 3 PUFA

ω3 PUFA (and parent essential fatty acid α -linoleic acid) are found in fish and fish oils and their health benefits are being more fully explored. The health benefits of ↑ consumption of oily fish include improved cardiovascular risk factors. The Western diet contains a high ratio of ω6:ω3 PUFAs; a lower ratio (4:1) is recommended. Research studies have shown benefits in cognitive function but epidemiological studies are required.

Table 5.4 Nomenclature of fatty acids

Common name	Notational name	Systematic name
Saturated fatty acids		
Butyric	4:0	Tetranoic
Caproic	6:0	Hexanoic
Caprylic	8:0	Octanoic
Capric	10:0	Decanoic
Lauric	12:0	Dodecanoic
Myristic	14:0	Tetradecanoic
Palmitic	16:0	Heaxdecanoic
Stearic	18:0	Octadecanoic
Arachidic	20:0	Eicosanoic
Behenic	22:0	Docosanic
Monounsaturated fatty acids		
Palmitoleic	16:1*n*7	9 *cis*-hexadecenoic
Oleic	18:1*n*9	9 *cis*-octadecenoic
Elaidic	18:1*n*9	9 *trans*-octadecenoic
Eicosenoic	20:1*n*9	11 *cis*-eicosaenoic
Erucic	22:1*n*9	13 *cis*-docosaenoic
Polyunsaturated fatty acids		
Linoleic	19:2*n*6	9,12 *cis, cis*-octadecadienoic
Alpha-linolenic	18:3*n*3	9,12,15 all *cis*-octadecatrienoic
Gamma-linolenic	18:3*n*6	5 *trans*, 9 *cis*, 12 *cis*-octadecatrienoic
Arachidonic	20:4*n*6	5, 8, 11, 14 *cis*-eicosatetraenoic
EPA	20:5*n*3	Eicosapentaenoic
DHA	22:6*n*3	Docosahexaenoic

Trans fatty acids

Trans fatty acids are rare in naturally occurring fats. Some is made in the rumen of cows and sheep and ∴ found in lamb, beef, milk, and cheese. The most significant source of *trans* fatty acids in the diet is obtained through the hydrogenation of PUFA to produce more solid forms of vegetable oils for spreads, margarines, and some food products. *Trans* fatty acids have been associated with adverse effects on lipoprotein status by elevating LDL and depressing HDL although further research is required. It is recommended that intake should not exceed 2% of total energy intake.

Essential fatty acids

Linoleic and α-linoleic acids are essential fatty acids. Other longer chain fatty acids such as arachidonic, EPA, and DHA are physiologically important but can be synthesized to a limited extent from linoleic and α-linoleic acid. These longer chain fatty acids are not essential fatty acids but their intake may become critical in fatty acid deficiency. EFA are most commonly found in plant and fish oils. Deficiency of linoleic acid has been demonstrated in children although a deficiency of α-linoleic acid has not been seen in healthy people. This has → debate about the essentiality of α-linoleic acid. Deficiency is characterized by a scaly dermatitis. The recommended intake of linoleic acid is at least 11% of total energy and 0.2% for α-linoleic acid.

Sterols

Sterols are relatively simple molecules; the most common sterol is the wax-like cholesterol. Cholesterol and cholesterol ester (cholesterol to which a fatty acid is attached) are only found in animal foods. Phytosterols are found in plant foods. Cholesterol has structural roles in lipoproteins and membranes and is a precursor for bile acids, steroid hormones, and vitamin D. Dietary cholesterol has little influence on plasma levels as most circulating cholesterol is endogenous. Reduction of intake of saturated fat results in lower plasma cholesterol levels.

Lipid transport

Fat digestion and absorption are covered in 📖 Chapter 1, 'Digestion'. Lipids are not soluble in water and ∴ complex with apolipoproteins to form water-miscible compounds. Approximately 2% of total plasma lipids are free fatty acids and are transported as compounds of albumin. The remainder of lipids is carried in the blood as lipoproteins. Lipoproteins are identified by the apoliprotein that is present (apo A, apo B, apo C, apo D, and apo E). There are five classes of lipoproteins, which vary in density:

- Chylomicrons.
- Very low density lipoproteins (VLDL).
- Low density lipoproteins (LDL).
- High-density lipoproteins (HDL).
- Lipoprotein (a) (LP(a)).

High and low levels of the lipoproteins have adverse effects on health. High levels of LDL are associated with ↑ health problems and LDL is colloquially known as 'bad cholesterol'. HDL is colloquially known as 'good cholesterol' (see Table 5.7 for dietary sources).

Table 5.5 Average intake for adults compared with DRVs for fat for adults (as a percentage of daily food energy intake) in the UK[*] [†]

	Average intake (% daily food energy)	DRV (% food energy intake)	
	♂	♀	
Total fat	35.5	34.7	35
Saturated fat	13.0	12.6	11
Monosaturated fatty acids	12.8	12.3	13
Polyunsaturated fatty acids	6.3	6.4	6.5
Trans fatty acids	0.8	0.8	<2
Cholesterol[‡]	304	213	<245 mg

[*] Source for average daily intakes for adults, Bates, B., Lennox, A., and Swan, G. (2010). *The National Diet and Nutrition Survey: Headline results from Year 1 of the Rolling Programme (2008/2009)*. Food Standards Agency, London

[†] Source: Henderson, L., Gregory, J., and Irving, K. (2003), *The National Diet and Nutrition Survey; adults aged 19 to 64 years*. Vol. 2, *Energy protein, carbohydrate, fat and alcohol intakes*. HMSO, London.

[‡] Cholesterol intake and DRV are expressed in mg/day.

Table 5.6 Sources of total fat, saturated and trans fatty acids in the diet of adults in the UK (NDNS)[*]

Food	Total fat (%)	Saturated fatty acids (%)	Trans fatty acids (%)
Meat, meat products, and meat dishes	26	26	25
Cereal and cereal products	18	18	19
Milk and milk products	13	22	22
Vegetables, potatoes	11	7	8
Fat spreads	9	9	9

[*] Source for average daily intakes for adults, Bates, B., Lennox, A., and Swan, G. (2010). *The National Diet and Nutrition Survey: Headline results from Year 1 of the Rolling Programme (2008/2009)*. Food Standards Agency, London.

Chylomicrons

Chylomicrons mainly consist of triglycerides as they transport dietary lipids. Plasma levels rise after eating and are negligible in the fasting state. Chylomicrons leave the enterocytes of the small intestine and enter the lymphatic system before transferring to blood vessels. The triglycerides are hydrolysed by lipoprotein lipase so releasing fatty acids that are used for energy or stored in adipose tissue. The life cycle of a chylomicron is 15–20 minutes and the liver clears the remnant from the blood. Fat-soluble vitamins reach the liver as part of the remnant.

Very low-density lipoproteins

VLDL are synthesized in the liver and are large particles that are rich in triglycerides. They deliver fatty acids to adipose tissue, muscles, and heart where lipoprotein lipase facilitates their release from triglycerides. The enzyme in the heart has a high affinity for triglyceride and, when triglyceride concentrations are low, they are preferentially released into heart tissue. Following release of triglycerides the remaining remnants are intermediate density lipoproteins (IDL), which are the precursors of low density lipoproteins (see Table 5.8).

Low-density lipoproteins

LDL contain mainly cholesterol and cholesterol ester as they are the end product of VLDL metabolism. They carry approximately 70% of plasma cholesterol and are taken up by the liver and other tissues by LDL receptors.

High-density lipoproteins

The liver and intestine synthesize and secrete HDL. HDL is involved in the reverse transport of cholesterol from tissues to the liver or transfers it to other lipoproteins.

Lipoprotein (a)

This is a complex of LDL with apolipoproteins (a).

Table 5.7 Dietary sources of cholesterol

Cholesterol content	Food
High	Liver, offal
	Eggs, mayonnaise
	Shellfish
	Fish roe
Medium	Meat fat
	Full fat milk and dairy produce,
	e.g. cream, cheese, butter
	Meat and fish products
	Manufactured meat products, e.g. pies
Low	Skinless poultry
	Skimmed milk and dairy products,
	e.g. cottage cheese, low fat yoghurt
Cholesterol free	Fruit (including avocados and olives) and vegetables
	Vegetable oils
	Cereals, pasta
	Rice
	Egg white
	Sugar

Table 5.8 Functions of plasma lipoproteins

Lipoprotein	Function
Chylomicrons	Transport dietary lipids to peripheral tissues and liver
VLDL	Transports lipids from liver to peripheral tissues
LDL	Transports cholesterol to peripheral tissues and liver
HDL	Removes cholesterol from peripheral tissues to the liver
Albumin	Transports free fatty acids from adipose tissue to peripheral tissues

Carbohydrate

Carbohydrates are the most significant source of energy in the diet (see 📖 this Chapter 'Energy balance', p. 80). In developing countries up to 85% of energy in the diet is provided by carbohydrate; this figure is as low as 40% in some developed countries. The relationship between dietary carbohydrates and fat is usually reciprocal. Diets rich in fat will have low levels of carbohydrates and vice versa.

Structure and classification

The empirical formula for carbohydrates is $C_x(H_2O)_y$; glucose is the simplest carbohydrate ($C_6H_{12}O_6$ or $C_6(H_2O)_6$) (Fig. 5.4). Simple carbohydrates (monosaccharides) can combine to form disaccharides, e.g. sucrose ($C_{12}H_{22}O_{11}$) from two disaccharides, oligosaccharides, e.g. raffi-nose which is formed from 3–11 monosaccharides, or polysaccharides, which form from 12 or more saccharides, e.g. starches.

❗ It is important to recognize that the physical effects (food matrix) of a carbohydrate may influence its nutritional properties.

Carbohydrates that can be digested and absorbed in the small intestines and → ↑ in blood glucose levels are referred to as glycaemic carbohydrates (Table 5.9). Plant polysaccharides that cannot be digested (non-glycaemic) are referred to as fibre or non-starch polysaccharides. Sugar alcohols are also classified as carbohydrates although their empirical formula is slightly different.

Sugars (mono-and disaccharides)

Monosaccharides include glucose, fructose, and galactose. The monsaccharide free glucose is found in small amounts in fruit and vegetables but is not abundant in natural foods. It is made from starch and used commercially. Fructose is found in honey, fruit, and vegetables and is manufactured from fructose-rich corn syrup for the food industry. Sucrose is the commonest disaccharide and is extracted from sugar beet or sugar cane. Table sugar is 99% sucrose and the major dietary source of disaccharides. Sucrose is hydrolysed into glucose and fructose. Lactose is found in milk and milk products. It is hydrolysed to glucose and galactose. Maltose is present in malted wheat and barley. Malt extract is used in brewing and in malted products.

Oligosaccharides

Raffinose, stachyose, and verbascose are oligosaccharides that are made of galactose, glucose, and fructose. They are found in legumes and seeds. Humans do not have the enzyme needed to digest them but they may be fermented in the colon. Fructo-oligosaccharides and inulin have been shown to stimulate growth of the potentially beneficial bifidobacteria in the colon.

Sugar alcohols

Sorbitol, inositol, and mannitol are sugar alcohols that are only partially absorbed and ∴ provide less energy than the corresponding sugars. Therefore they have been used as sugar substitutes. Small amounts occur naturally but significant amounts in the diet come only from manufactured foods. Large amounts can cause osmotic diarrhoea.

Table 5.9 Classification of carbohydrates in the diet (FAO/WHO 1998)*

Glycaemic	**Non-glycaemic**
Monosaccharides	**Oligosaccharides**
Glucose	Raffinose, stachyose, verbascose
Fructose	Human milk oligosaccharides
Galactose	Fructo-oligosaccharides
	Inulin
Disaccharides	
Sucrose	
Lactose	
Maltose	
Trehalose	
Polysaccharides	**Non-starch polysaccharides**
Starch—amylopectin, amylose, modified food starches	Cellulose (insoluble)
	Hemicellulose (soluble and insoluble forms)
	β-glucans (mainly soluble)
	Fructans, e.g. inulin (not assayed by current methods)
	Gums (soluble)
	Mucilages (soluble)
	Algal polysaccharides (soluble)
Sugar alcohols†	
Sorbitol	
Xylitol	
Mannitol	

*WHO/FAO (1998). *Carbohydrates in human nutrition*, FAO food and nutrition paper no.66. FAO, Rome.

†Sugar alcohols are only partially absorbed.

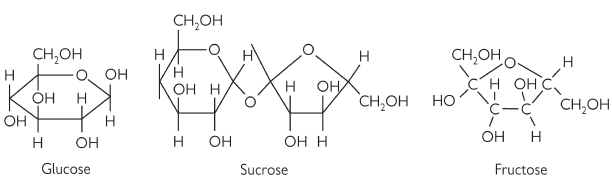


Fig. 5.4 Carbohydrate molecules.

Starch

Starch is the main storage polysaccharide in plant cells and is found in large quantities in cereal grains, potatoes, and plantains. Starch is the largest source of carbohydrate in the diet. Starch consists of two glucose polysaccharides: amylose and amylopectin. The linkages between the glucose molecules are degraded by the action of α-amylase. Many factors affect the rate at which the linkages are degraded so that some starches are readily digested while others pass undigested into the colon. This has resulted in the classification of starches (Table 5.10) into rapidly digestible starch (RDS), slowly digestible starch (SDS), and resistant starch (RS). Both RDS and SDS are digested in the small intestine while RS passes undigested into the colon where it is available for fermentation.

Non-starch polysaccharide: fibre

In the UK, non-starch polysaccharide (NSP) is the term used in preference to fibre. Dietary fibre is defined as NSP that does not include lignin or resistant starch; the terms are ambiguous. Fibre can be classified as soluble (in water at pH 7.0) or insoluble and it is this classification that categorizes the function of these polysaccharides. Insoluble fibre consists mainly of cellulose and some hemicelluloses. Insoluble fibre binds to water in the colon and swells. This stimulates peristalsis so ↑ transit time in the colon thereby reducing the risk of constipation and possibly reducing the risk of colon cancer. Soluble fibre blunts the response of blood glucose to ingestion. The reabsorption of bile acids is slowed by soluble fibre so ↑ cholesterol losses in faeces and reducing blood cholesterol levels. Table 5.11 lists sources of soluble and insoluble fibre in the diet.

Intrinsic sugars

These are sugars that are present in intact cells, e.g. fructose in whole fruit and sugars in milk, i.e. lactose and galactose.

Non-milk extrinsic sugars

Sugars that are in a free or readily absorbable state, e.g. added sugars (usually sucrose), or released from disrupted cells, e.g. fructose in fruit puree or juice. Non-milk extrinsic sugars (NMES) contribute to the development of dental caries.

Recommended intakes

Sugar and starch

SACN recommend that the intake of intrinsic or milk sugars should not be limited in adults. They recommended that infant formulas should contain approximately 40% energy from sugars; this is similar to the sugar content of breast milk. It is recommended that the average intake of NMES should not exceed 60 g/day or 11% daily energy.

Starches, intrinsic sugars, and milk sugars should provide the balance of dietary energy not provided by alcohol, protein fat, and NMES, which is on average 37% in UK (Tables 5.12–5.14).

NSP It is recommended that the adult diet contain 18 g NSP/day (12–24 g/day; see Table 5.15).

Table 5.10 Classification of starch

Class	Glycaemic response	Food source
Rapidly digestible starch	Large	Cooked starchy cereals, warm potatoes
Slowly digestible starch	Small	Muesli, oats, pasta, legumes
Resistant starch	None	Unripe bananas, whole grains

Table 5.11 Dietary sources of soluble and insoluble fibre in the diet

Soluble fibre	Insoluble fibre
Apples	Beans
Barley	Brown rice
Citrus fruits	Fruits with edible seeds
Guar gum	Lentils
Legumes	Maize
Oats	Oats
Pears	Pulses
Strawberries	Wheat bran
	Wholemeal breads
	Wholemeal cereals
	Wholemeal pasta
	Whole wheat flour
	Peas

Table 5.12 Daily carbohydrate and NMES intake of adults (NDNS)*

	Men	Women
Total carbohydrate (g/day)	256	198
% total energy	46.8	47.8
NMES (g/day)	72.6	51.7
% total energy	13.0	12.1
DRV (% total energy)	10.0	10.0
NSP (g/day)	15.1	13.0
DRV (g/day)	18.0	18.0

*Source for average daily intakes for adults, Bates, B., Lennox, A., and Swan, G. (2010). *The National Diet and Nutrition Survey: Headline results from Year 1 of the Rolling Programme (2008/2009)*. Food Standards Agency, London.

Table 5.13 Sources of carbohydrate in the diet (NDNS)*

Food group	% Daily intake
Cereals and cereal products	42
Bread	17
Potatoes and savoury snacks	12
Non-alcoholic beverages	8
Alcoholic beverages	4

*Source for average daily intakes for adults, Bates, B., Lennox, A., and Swan, G. (2010). The National Diet and Nutrition Survey: Headline results from Year 1 of the Rolling Programme (2008/2009). Food Standards Agency, London.

Table 5.14 Sources of NMES in the diet (NDNS)*

Food group	% Intake
Non-alcoholic beverages	25
Alcoholic beverages	13
Sugar, preserves, and confectionery	25
Table sugar, preserves & sweet spreads	15
Cereals and cereal products	20

*Source for average daily intakes for adults, Bates, B., Lennox, A., and Swan, G. (2010). *The National Diet and Nutrition Survey: Headline results from Year 1 of the Rolling Programme (2008/2009)*. Food Standards Agency, London.

Table 5.15 Sources of NSP in the diet (NDNS)*

Food group	Selected food	% intake
Cereals and cereal products		37
Breakfast cereals		6
Vegetables (excluding potatoes)		21
Potatoes and savoury snacks		14
Fruit and nuts		10

*Source for average daily intakes for adults, Bates, B., Lennox, A., and Swan, G. (2010). *The National Diet and Nutrition Survey: Headline results from Year 1 of the Rolling Programme (2008/2009)*. Food Standards Agency, London.

Glycaemic index

The glycaemic index (GI) is a method of ranking foods and carbohydrates based on their immediate effect on blood glucose levels. The FAO/WHO (1998)[1] define the GI as 'the incremental area' under the blood glucose response curve of one 50 g carbohydrate portion of a test food expressed as a percentage of response to the same amount of carbohydrate from a standard food taken by the same subject.' The standard carbohydrate is glucose that has a GI of 100. Foods with a high glycaemic index are readily absorbed and raised blood glucose quickly. Low glycaemic index foods are digested and absorbed slowly and raise blood glucose levels slowly. The GI can only be determined by *in vivo* measurement. Foods are categorized into:

- *Low GI:* 55 or less.
- *Medium GI:* 56–69.
- *High GI:* 70 or more.

Table 5.16 lists examples of GI of these categories. A list of foods that have been tested has been published by Foster-Powell *et al.* (2002)[2]; more information is available at the Glycemic Index Foundation www.glycemicindex.com. A list of commercially available products in the UK has been published by Henry *et al*, (2005)[3]. The way a food is processed, prepared, and cooked will affect the GI of the food. The overall GI of the diet is important rather than aiming to introduce a few low GI foods. The health benefits of a low GI diet include:

- Improved diabetic glucose control (see Chapter 22, p. 448);
- Improved risk factors for heart disease (see Chapter 23, p. 470);
- Weight reduction (see Chapter 21, 'Obesity', p. 411);
- There is some evidence to suggest ↓ risk of colon and breast cancers.

Glycaemic load (GL)

GL extends the concept of GI by considering the GI and the amount of a carbohydrate have on postprandial blood glucose levels.

$$(GL = \text{Carbohydrate in food portion (g)} \times GI)/100$$

Blood glucose levels rise more rapidly after a high GL meal than a low GL meal. It is recommended that a healthy diet should have a low GI and a low GL.

[1] WHO/FAO (1998). *Carbohydrates in human nutrition*, FAO food and nutrition paper no.66. FAO, Rome.

[2] Foster-Powell, K. Holt, S.H.A., and Brand-Miller, J.C. (2002). International table of glycaemic index and glycaemic load. *Am. J. Clin. Nutr.* **76**, 5–56.

[3] Henry, C.J.K., Lightowler, H.J. Strik, C.M., Renton, H. and Hails, S. (2005). Glycaemic index and glycaemic load values of commercially available products in the UK. *Brit. J. Nutr.* **94**, 922–930.

Table 5.16 Examples of low, medium, and high GI foods

Low GI	Medium GI	High GI
Apples, oranges, pears, peaches	Honey	Glucose
Beans and lentils	Jam	White and wholemeal bread
Pasta (all types made from durum wheat)	Shredded Wheat	Brown rice, cooked
Sweet potato, peeled and boiled	Weetabix	White rice, cooked
Sweet corn	Ice cream	Cornflakes
Porridge	New potatoes, peeled and boiled white basmati rice, cooked	Baked potato
Custard	Pitta bread	Mashed potato
Noodles	Couscous	
All Bran, Special K, Sultana Bran		

Energy balance

In order to maintain body weight, energy intake must equal energy expenditure. If energy expenditure exceeds energy intake body weight will be lost. Weight loss is achieved by ↑ energy expenditure or ↓ energy intake. To gain weight the equation is reversed.

The SI unit of energy is the joule (J); the joule is a small amount of energy. Energy in food is usually expressed as kilojoules (kJ) and energy expenditure is expressed as kJ or megajoules (MJ). In practice many people continue to express energy in kilocalories (kcal). A calorie can be defined in several ways although the most frequently used definition is:

- The energy required to raise the temperature of 1 g of water from 14.5°C to 15.5°C.

Energy expenditure can be expressed per unit of time, e.g. kJ per minute or MJ/day or in Watts (W) (see Box 5.1 for a summary of units).

Box 5.1 Units used in energy balance

- 1000 joules = 1kJ
- 1000 kJ = 1MJ
- 1 kcal = 4.184 kJ*
- 1 kJ = 0.239 kcal
- 1 W = 1 joule per second
- 0.06 W = 1 kJ per min
- 86.4 W = kJ per 24 h

*The Royal Society (London) recommended conversion factor.

Energy expenditure

Total energy expenditure (TEE) has the following components:

- basal metabolic rate (BMR), 50–75%;
- physical activity (PA), 20–40%;
- dietary induced thermogenesis (DIT), 10%.

Growth, pregnancy, lactation, injury, and fever are energy-requiring processes that will ↑ energy expenditure and → ↑ energy intake.

Basal metabolic rate

Basal metabolic rate (BMR) is the amount of energy expended by the body to maintain normal physiological functions. It remains constant throughout the day, under normal conditions, and constitutes 50–75% of TEE; it is the largest component of TEE.

BMR is affected by many factors:

- *Body weight:* BMR ↑ or ↓ with ↑ or ↓ body weight;
- *Body composition:* Fat mass is relatively metabolically inactive and expends less energy gram for gram than fat free mass (FFM). Men have a higher FFM to fat ratio than women and ∴ have a higher BMR than women of the same age and weight;

- *Age:* children have a higher BMR per kg than adults due to the energy requirement of growth. As adults age, metabolism slows and FFM ↓ ∴ ↓ BMR;
- *Gender:* men generally have a higher BMR due to differences in body weight and body composition. The BMR of a 65 kg man will be approximately 1 MJ/day higher than a weight-and age-matched woman;
- *Genetic factors:* BMR can vary by up to 10% between subjects of the same age, sex, and body weight. Recent research has shown that there are ethnic differences in BMR;
- *Physiological changes*: BMR ↑ during pregnancy and lactation;
- *Disease and trauma:* Fever, sepsis, infection, surgical and physical trauma ↑ BMR;
- *Nutritional status*: the body adapts to changes in energy intake by altering body weight and/or body composition. An individual who is consuming more energy than is required will ↑ weight and ↑ BMR so making further weight impossible unless intake ↑ further;
- *Environment*: the energy cost of maintaining body temperature is influenced by ambient temperature, wind speed, radiant temperature of the surrounding, and clothing;
- *Hormonal status:* several hormonal factors influence BMR especially thyroid function. BMR is ↑ in hyperthyroidism and ↓ in hypothyroidism. There are small cyclical changes during the menstrual cycle of some women with a rise after ovulation;
- *Pharmacological effects:* therapeutic drugs and substances such as caffeine and capsaicin can modulate BMR;
- *Psychological effects:* anxiety will ↑ energy expenditure in the short term. Longer term effects of stress and anxiety have not been established.

Measurement of BMR

- BMR must be measured under standard conditions.
- Post-absorptive state—at least 12 h after last food or drink. This should also include other stimulants such as caffeine or smoking.
- Thermoneutral environment—20–25°C; comfortably warm.
- Supine—sitting up will ↑ energy expenditure slightly.
- Awake but in a state of complete physical and mental relaxation.
- Heavy physical activity on the day before the measurement may influence the BMR and should be avoided.

In practice BMR is usually measured first thing in the morning before eating and drinking or undertaking physical activity. If any of the conditions are not met the measurement is termed resting metabolic rate (RMR). RMR is slightly higher than BMR while sleeping metabolic rate is 5–10% lower than BMR.

Measurements of energy expenditure

Energy expenditure can be measured directly (the measurement of heat production), indirectly (the measurement of O_2 consumption), or by non-calorimetric methods, e.g. heart rate monitoring. More recently, methods have been developed that are indirect measures of gaseous exchange (O_2 consumption), i.e. doubly labelled water technique.

Direct calorimetry

Direct calorimetry is the measurement of heat produced by the body. Subjects are placed in an insulated chamber and heat loss is measured over a period of at least 24 h. Direct calorimetry is difficult in practice as the chamber must be capable of detecting all heat generated within the chamber and other sources of heat must be eliminated or accounted for. Direct calorimeters are very precise instruments but are expensive and difficult to build and maintain and few are available; ∴ this method is not frequently used.

Indirect calorimetry

Indirect calorimetry is based on the principle that food is oxidized in the body to produce energy and that by measuring oxygen consumption it is possible to calculate energy expenditure. The following equation demonstrates the amount of energy produced by the oxidation of 1 → mole of glucose:

$C_6H_{12}O_6$	+	$6O_2$	→	$6CO_2$	+	$6H_2O$	+	heat
(180 g)		(6 × 22.41)		(6 × 22.31)		(6 × 18 g)		(2.78M)

The energy produced by the oxidation of 1 g glucose is ∴ 15.4 kJ (2780/180) and 1 l of oxygen is equivalent to the production of 20.7 kJ (2780/6 x 22.4). Therefore if the amount of oxygen used is known, it is possible to calculate the amount of energy or heat produced. Similar calculations can be made for protein, fat, and alcohol.

Respiratory quotient (RQ) is the ratio of oxygen used to the amount of carbon dioxide produced. From the RQ it is possible to estimate the macronutrient composition of the diet (see Table 5.17). The energy content of a mixed diet is approximately 35% fat, 50% carbohydrate, and ∴ has an RQ of 0.87. To improve the accuracy of the calculations an estimate of nitrogen excretion is used. Substitution into a formula yields energy expenditure (EE). The formulae most frequently used are those of Weir (1949)[4], or Elia and Livesey (1992)[5] (see Box 5.2).

Indirect calorimetry equipment

Various apparatus is available to measure oxygen consumption. The simplest method is the Douglas bag where expired air is collected into a strong non-permeable bag. The volume of expired air over a set period is measured using a dry gas meter and the expired gases are analysed and compared to the ambient air. From this it is possible to calculate O_2 consumption and CO_2 production rates and ∴ calculate energy expenditure. In clinical situations, a ventilated hood, canopy, or tent, e.g. Deltatrac, Gem, Sensormedics, is used which measures gaseous exchange continuously and has a processor to calculate energy expenditure. Other systems are available that can be used during exercise. Respiration chambers are used by some research units; these are small chambers in which a subject

[4] Weir, J.B. De V. (1949). New methods for calculating metabolic rate with special reference to protein metabolism. *J. Physiol.* (*Lond.*) **109**, 1–9.

[5] Elia, M. and Livesey, G. (1992). Energy expenditure and fuel selections in biological systems: the theory and practice of calculations based on indirect calorimetry and tracer methods. In *Metabolic Control of Eating, Energy Expenditure and the Bioenergetics of Obesity* (ed. A.P. Simonopoulos), pp. 68–131. Karger, Basel.

stays for several hours or days and gaseous exchange is measured continuously. These chambers are expensive to build and use but give precise measurements.

Non-calorimetric methods

- Heart rate is related to energy expenditure and this relationship has been used to estimate energy expenditure although the results are not very reliable, particularly at low activity levels.
- Accelerometers are often used to measure physical activity; they are small computer motion analysers that measure duration, frequency, and intensity of physical activity. They are used in conjunction with log books that enable the full analysis of activities.

Box 5.2 Weir, and Elia and Livesey formulae

Weir formula

$$EE\ (kJ) = 16.489\ VO_2\ (l) + 4.828\ VCO_2\ (l) - 9.079\ N\ (g)$$

If nitrogen cannot be measured protein is assumed to be 15% of the energy of the diet and the formula becomes:

$$EE\ (kJ) = 16.318\ VO_2\ (l) + 4.602\ VCO_2\ (l)$$

Elia and Livesey formula

$$EE\ (kcal/24\ h) = ((15.913\ VO_2\ (l) + 5.207\ VCO_2\ (l)) \times 1.44 - 4.464\ N\ (g)) \times 0.239$$

where $VO_2 = O_2$ consumed, $VCO_2 = CO_2$ produced, and N = urinary nitrogen excretion

Table 5.17 Energy values for oxidation of nutrients*

Nutrient	O_2 consumption (l/g)	CO_2 production (l/g)†	RQ	Energy released (kJ/g)	Energy released (kJ/l O_2)
Starch	0.829	0.832	0.994	17.49	21.10
Glucose	0.746	0.742	0.995	15.44	20.70
Fat	1.975	1.402	0.710	39.12	19.81
Protein	0.962	0.775	0.806	18.52	19.25
Alcohol	1.429	0.966	0.663	29.75	20.40

*Reproduced from Garrow, J.S., James, W.P.T., and Ralph, A. (1999). *Human Nutrition and Dietetics*, table 17.1, p. 135. With permission from Elsevier.

† CO_2 is not an ideal gas.6l mole at STP occupies 22.26 l not 22.4 l.

Doubly labelled water

Data is collected on free-living subjects over a period of 10–20 days. It does not require extensive equipment for the collection of gases and ∴ does not restrict the subject. Subjects are given an oral dose of water that has known amounts of the stable isotopes deuterium (^2H) and ^{18}O. These isotopes mix with the body's water and, as energy is used, CO_2 and H_2O are produced. As ^{18}O is in both H_2O and CO_2 it is lost more rapidly than ^2H which is only lost in H_2O. The difference between the rate of loss of ^2H and ^{18}O reflects the rate at which CO_2 is produced. From this it is possible to calculate energy expenditure. This method requires collection of body fluid, either blood, urine, or saliva, before the test period and samples at specified times during the study. It is possible to use this method in babies, hospital patients, field work, and other groups in whom it is difficult to measure energy expenditure by other methods. Specialist equipment is required for the analysis of blood and urine samples and, due to a world shortage of ^{18}O, this method is expensive.

Estimation of energy requirements

Energy requirements are estimated by using prediction equations such as the Schofield equations (1985), see Appendix 14. Table 5.18 shows the Schofield equations with additional data on men aged 60–70 years (DH 1991). Regression analysis of measured BMR against gender, age, and weight was used to generate the equations that estimate BMR. Numerous equations are available; ideally they should be population. specific. They are developed for use in healthy groups; in individuals the accuracy may be ±10–20%. If equations are extended for use in illness the accuracy may be reduced by 50%.

TEE is calculated by using a physical activity level (PAL) that has been derived from experimental studies, often using doubly labelled water; this is known as the factorial method.

For example, a sedentary male worker, aged 40 years, weight 90 kg, with an inactive lifestyle would have PAL of 1.4 (Table 5.19); ∴ his TEE would be

BMR from prediction equations (7.973 MJ) x 1.4 = 11.16 MJ.

If an activity diary has been kept it is possible to calculate TEE more accurately by partitioning time during the day spent on specific activities and using physical activity ratios (PAR; see 🕮 Appendix 6, p. 778) it is possible to calculate a directly related PAL value for the day.

TEE = BMR x [(PAR x time for activity A) +
(PAR x time for activity B) + ……..]

Table 5.18 Formulae for the estimation of BMR*

	Age (years)	BMR prediction equation (MJ/day)†
Men	10–17	0.074 (w) + 2.754
	18–29	0.063 (w) + 2.896
	30–59	0.048 (w) + 3.653
	60–74	0.0499 (w) + 2.930
	75 +	0.035 (w) + 3.434
Women	10–17	0.056 (w) + 3.434
	18–29	0.062 (w) + 2.036
	30–59	0.034 (w) + 3.538
	60–74	0.0386 (w) + 2.875
	75 +	0.041 (w) + 2.610

*Equations based on Schofield, W.N. (1985). Predicting basal metabolic rate, new standards and review of previous work. *Hum. Nutr. Clin. Nutr.* **39**C (Suppl. I), 5–41. Additional data on men aged 60–70 years from Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. HMSO, London.

†w, Weight in kg.

Table 5.19 Calculated PAL values for light, moderate, and heavy activity (occupational and non-occupational)*

Non-occupational activity level	Occupational activity level					
	Light		Moderate		Heavy	
	M	F	M	F	M	F
Sedentary	1.4	1.4	1.6	1.5	1.7	1.5
Moderately active	1.5	1.5	1.7	1.6	1.8	1.6
Very active	1.6	1.6	1.8	1.7	1.9	1.7

*Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. HMSO, London.

Energy intake

Energy is provided by the macronutrients and alcohol.

- Protein, 4 kcal (17 kJ)/g.
- Carbohydrate, 3.75 kcal (16 kJ)/g.
- Fat, 9 kcal (37 kJ)/g.
- Alcohol, 7 kcal (29 kJ)/g.

Polyols (e.g. sorbitol) and volatile fatty acids (produced by gut bacteria by fermentation of some fibre components) contribute small, negligible amounts of energy.

Energy consumption

The average daily energy intakes for adults in UK are 9.48 MJ (2255 kcal) for men and 6.92 MJ (1645 kcal) for women; these intakes are below EARs. The sources of energy are shown in Fig. 5.5.

❗ In the UK adults are not energy deficient, as demonstrated by the rising prevalence of obesity. The low percentages of EARs may be due to under-reporting and reflect the widely held belief that EARs need revision. The level of physical activity is also important.

Energy requirements

The DH recommendations are shown in Table 5.20 for babies and children to 10 years. These are given as estimated average requirements (EAR). EARs for men and women are grouped for age, weight, and activity level as shown in Table 5.21.

❗ SACN are currently re-evaluating the DRVs for energy requirements in the UK. The recommended equations for the calculation of BMR are also being re-evaluated and the equations of Henry (2005) will be recommended for use in the UK once the report is published[6].

Table 5.20 Estimated average requirements (EARs) for energy of children 0–18 years*

Age	EAR MJ/d (kcal/day)	
	Boys	Girls
0–3 months	2.28 (545)	2.16 (515)
4–6 months	2.89 (690)	2.69 (645)
7–9 months	3.44 (825)	3.20 (765)
10–12 months	3.85 (920)	3.61 (865)
1–3 years	5.15 (1230)	4.86 (1165)
4–6 years	7.16 (1715)	6.46 (1545)
7–10 years	8.24 (1970)	7.28 (1740)
11–14 years	9.27 (2220)	7.72 (1845)
15–18 years	11.51 (2775)	8.83 (2110)

* Source for EARs, Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. HMSO, London.

[6] Henry CJ. (2005). Basal metabolic rate studies in humans: measurement and development of new equations. *Public Health Nutr*, **8**(7A):1133–1152.

Table 5.21 Estimated average requirements (MJ/day) according to body weight and PAL[†]

Body weight (kg)	BMR[*] (MJ/day)	PAL				
		1.4	1.5	1.6	1.8	2.0
Males						
30	4.97	7.0	7.5	8.0	9.0	9.9
35	5.34	7.5	8.0	8.6	9.6	10.7
40	5.71	8.0	8.6	9.1	10.3	11.4
45	6.08	8.5	9.1	9.7	11.0	12.2
50	6.45	9.0	9.7	10.3	11.6	12.9
55	6.82	9.6	10.2	10.9	12.3	13.6
60	7.19	10.1	10.8	11.5	12.9	14.4
65	7.56	10.6	11.3	12.1	13.6	15.1
Females						
30	4.58	6.4	6.9	7.3	8.2	9.2
35	4.86	6.8	7.3	7.8	8.7	9.7
40	5.14	7.2	7.7	8.2	9.2	10.3
45	5.42	7.6	8.1	8.7	9.8	10.8
50	5.70	8.0	8.5	9.1	10.3	11.4
55	5.98	8.4	9.0	9.6	10.8	12.0
60	6.26	8.8	9.4	10.0	11.3	12.5

[*] BMR, Basal metabolic rate calculated as per Table 5.18.

[†] Source Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. HMSO, London.

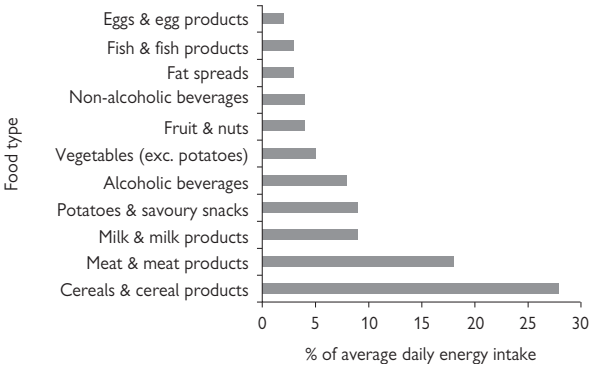


Fig. 5.5 Percentage contribution of food types to average daily total energy intake of UK adults. Source Bates, B., Lennox, A., and Swan, G. (2010). *The National Diet and Nutrition Survey: Headline results from Year 1 of the Rolling Programme (2008/2009)*. Food Standards Agency, London.

Physical activity assessment

Physical activity (PA) is the most variable component of TEE and most amenable to change. PA is a complex behaviour that includes any bodily movement produced by the contraction of skeletal muscles resulting in energy expenditure. It incorporates all daily activities and is not synonymous with exercise, which is a sub-category and tends to be structured leisure-time activity. Sedentary behaviour is independent from PA and should be considered as a separate component, but can often be measured with the same instrument as PA. The choice of measuring instrument is a balance between accuracy, reliability, detail and practical considerations. The timing of the assessment is important and must include consideration of day-to-day variability in PA patterns, (e.g. week day vs. weekend day), seasons and special occasions that could influence habitual PA. The length of measurement period is dependent on these factors and the aim of the assessment. If the aim is to assess habitual PA, a longer measurement period is required and repeated measurement periods (e.g. different times of year) should be considered (see Table 5.22).

PA assessment in children

There are additional challenges in assessing PA in children as their activity patterns are more varied and movement is more sporadic and multidimensional than in adults and they have cognitive limitations in recalling their activity. With some of the objective instruments for PA assessment, changes in body size and energy efficiencies with growth also need to be considered. Instruments can broadly be divided into subjective and objective and can be used in combination to provide complimentary measurements.

Table 5.22 Definitions in PA assessment

Measurement	Description
Intensity	Intensity of activity usually defined in terms of metabolic equivalents (MET), such as light (1.1–2.9 MET) moderate (3.0–5.9 MET) and vigorous (6.0+ MET) intensity[1]
Frequency	Frequency of time spent in specific activities or intensity levels over a set period of time
Duration	Time spent in specific activities or intensity levels including total time per day, proportion of waking hours or length of bout of activity
Patterning	Occurrence of specific activities or intensity levels over set period of time e.g. time of day or day of week
Types of activity	Specific activities of interest e.g. walking or cycling
Domains of activity	Context of activities e.g. home, work, leisure-time or mode of transport
Sedentary behaviour	Time spent in activities involving being sedentary e.g. watching television, reading or on computer

[1] METS are used when describing PA intensity multiples of an individual's resting oxygen uptake, defined as the rate of oxygen (O_2) consumption of 3.5 ml of O_2/min/kg body weight in adults.

Subjective instruments

Diaries

PA recalls

Activity recalls are analogous to 24-h diet recalls, but may cover a longer time period (1 d—1 m). Activities tend to be of moderate-vigorous intensity that are relatively easy to recall. Repeated recalls at intervals across a time period can be used to capture information on patterning or estimate habitual PA.

Questionnaires

Questionnaires are widely used and there are many questionnaires used in different populations and age groups. Questionnaires vary from a few generic questions to detailed lists of questions on different activities. To assess total PA, questionnaires should include all domains and all common activities undertaken in the population of interest, taking into account culture-specific activities. It is important that a questionnaire is designed and validated against a criterion measure for use in the population group in which PA is being assessed (see Box 5.3).

Box 5.3 Good design features of questionaires

- Good for use in large groups
- Assess patterns, frequency, duration and type of PA
- Capture context of PA
- Measures of sedentary behaviour possible
- Limited in ability to assess energy expenditure
- Difficult for individual to quantify some activities
- Subject to recall bias or social desirability in reported activities
- Limited applicability in children due to cognitive
- Can require considerable data processing

Objective instruments

Pedometers

Pedometers are motion sensors worn on the hip or waist that measure locomotor activity as steps taken, walking or running. There is a large variation in the accuracy and reliability of different pedometer models. This is part reflects the different mechanics of the models and also variations in stride length. Some models allow a setting of the individual's walking stride length to improve the estimation of the distance covered and steps taken (see Box 5.4).

Box 5.4 Pedometers

- A simple and inexpensive measure of walking activity
- Small and non-invasive for people to wear
- Only assesses locomotor activities, not activities of upper body, cycling or water activities
- Unable to assess patterns, frequency, duration of activity, types of PA or sedentary behaviour
- Measurement capability varies with body placement e.g. hip or waist
- Limited application in groups of children as pedometer steps influenced by body size
- Limited applicability in those with restricted mobility
- Data not stored in memory of device
- Best for ranking individuals or assessing change in locomotor activities e.g. to monitor adherence to a walking intervention
- Unable to estimate energy expenditure

Accelerometers

Accelerometers detect and record acceleration resulting from normal bodily movement (Box 5.5). Models can measure acceleration in one direction (usually vertical), two and three directions (triaxial; vertical, medio-lateral and anterior-posterior) and are commonly expressed as a movement count value. The sampling period of current models vary from recording movement every second to every 60 s. The length of the measurement period depends on the sampling period and memory capacity of the accelerometer, but generally ranges from several days to weeks. Most

models store the movement counts for downloading at the end of the measurement period into a PC via an interface. This allows the activity count data to be used to measure patterns, frequency and duration of PA; estimate time spent in different intensities of activity with the use of appropriate cut points; and measure time spent sleeping and sleep quality.

Box 5.5 Accelerometers

- Most commonly used objective instrument
- Small and non-invasive for people to wear
- Often used in children
- Limited applicability in those with restricted mobility
- Unable to assess types of PA
- Measurement capability varies with body placement e.g. hip, ankle or wrist as does not capture all activity of across the body e.g. if worn on hip, upper body activity is not captured
- Most models are currently not waterproof so limited capability to measure water-based activities
- Time is required for processing output data. Many models have software provided for these analyses
- Some variation in the accuracy and reliability of different accelerometer models
- Estimation equations for energy expenditure have been developed for different accelerometer models and populations, but these have limited accuracy in estimating energy expenditure on an individual basis.

Heart rate monitors

Heart rate (HR) provides an indirect measure of PA, as it measures the individual's physiological response to PA. Minute-by-minute HR data is recorded from a chest strap and can then be downloaded at the end of the measurement period into a pc via an interface for processing see Box 5.6).

Box 5.6 Heart monitors

- Can assess whole range of movements and activities (including water activities)
- Can measure patterns, frequency and duration of PA
- Unable to assess types of PA or sedentary behaviour
- Can estimate time spent in different intensities of activity with the use of appropriate cut points
- At low levels of PA heart rate is a less reliable measure of PA
- Can estimate energy expenditure and patterning of expenditure with an individualised calibration of the O_2/HR relationship
- HR responses reflect not only PA but also affected by hydration, prandial status, body position, ambient temp., humidity, emotion stress, smoking, caffeine intake and certain drugs e.g. beta-blockers
- Relationship between individuals' HR and PA or EE can alter with changes with body weight, body composition, physical fitness, ageing and illness
- Downloaded data requires considerable processing to estimate PA or PA EE

Combined monitors

Instruments are increasingly becoming available that combine more than one objective method to overcome some of the limitations of the individual methods (e.g. combining heart rate monitoring with accelerometry). Some of these monitors can estimate energy expenditure, but may have limited accuracy in estimating on an individual basis.

Doubly labelled water

The doubly labelled water does not give a direct measure of PA EE. However, when combined with measured or estimated BMR, reasonable estimates of PA energy expenditure can be derived by subtraction of BMR and DIT, averaged over the period of isotope sampling (commonly 7–10 d; see Box 5.7). See previous section in this 🕮 chapter for methodology (p. 81).

Box 5.7 Doubly labelled water

- Provides no measure of day-to-day PA EE
- Unable to assess patterns, frequency, duration or types of PA or sedentary behaviour
- Most applicable in healthy groups, limitations in application in some illnesses
- Can be used in children and infants
- Application constrained by expense of method and specialist processing

Chapter 6

Micronutrients

Micronutrients: introduction

The micronutrients are by definition required in small amounts. Many are essential as they cannot be made in the human body. They include vitamins, minerals and trace elements.

Vitamins: introduction

Vitamins are a group of organic compounds that have a variety of functions in the body and that are chemically different from each other. To show that a compound is a vitamin it is necessary to show a deficiency in experimental subjects and restoring the missing compound can reverse that this. The name 'vitamin' is derived from 'vital amine'; as the name suggests these essential compounds were initially thought to be amines. Vitamins can be divided into fat-soluble and water-soluble groups; vitamins A, E, D, and K are fat-soluble and may be stored in the body, the remainder being water-soluble and the body has limited or no stores.

Vitamin A (retinol) and carotenoids

Vitamin A is the term for the biologically active compound retinol and its provitamin (precursor) carotenoids. The most common provitamin A carotenoids are β-carotene, α-carotene, γ-carotene, and β-cryptoxanthin. Only 50 of approximately 600 naturally occurring carotenoids are converted into vitamin A. Carotenoids with no vitamin A activity include zeaxanthin, the pigment in sweet corn, and lycopene, the red pigment in tomatoes. The vitamin A activity of β-carotene is calculated as 6 μg being equivalent to 1 μg of retinol. Other carotenoids are considered to have less activity; 12 μg is considered to be equivalent to 1 μg of retinol.

Function

- Vitamin A is essential for the production of rhodopsin in the rods of the retina. Exposure to light results in a series of changes in the configuration of rhodopsin, which leads to the adaptation of vision in the dark.
- Growth.
- Cell differentiation.
- Embryogenesis.
- Immune response.

Measurement

Biochemical assessment of vitamin A is controversial. The measurement of retinol concentration in serum or plasma is a useful and common measure of vitamin A status. Deficiency is indicated by values below 10 μg/dl (0.3 μmol/l) and values below 20 μg/dl (0.7 μmol/l) are marginal.

Deficiency

Deficiency of vitamin A is rare in the UK, but is common in Latin America, Africa, and Asia especially amongst children.

- *Eye changes:* night blindness presents when vitamin A status is marginal and, with prolonged or severe deficiency, changes to the cornea and conjunctiva occur. These eye changes are known collectively as xerophthalmia; these changes consist of conjunctival xerosis, Bitot's spots, corneal xerosis, corneal ulceration, and corneal necrosis and scars.
- *Epithelial tissues:* skin keratinization occurs in vitamin A deficiency. Horny plugs block the sebaceous glands leading to follicular hyperkeratosis.
- *Immunity:* vitamin A deficiency results in ↑ susceptibility to infectious diseases, such as diarrhoea and respiratory infections.

A deficiency of vitamin A can contribute to nutritional deficiency anaemia.

Requirement and intake

See Tables 6.1 and 6.2, and Box 6.1. Fat is necessary for the absorption of vitamin A; as retinol is found in foods of animal origin some fat is usually consumed at the same time. Vitamin A absorption is impaired by mineral oils, neomycin, cholestyramine, and commercial fat replacers, e.g. olestra. A low vitamin A intake is associated with lower socio-economic class and low consumption of cereals, milk, eggs, and vegetables.

Toxicity

The early reports of vitamin A toxicity are from polar explorers who ate the polar bears' livers, which are particularly rich in vitamin A. Acute toxicity occurs when more than 200 mg (0.7 mmol) is consumed by adults or more than 100 mg in children. The acute symptoms of vitamin A toxicity include vomiting, abdominal pain, anorexia, blurred vision, headache, and irritability. Chronic toxicity can occur when 10 mg is consumed over periods of a month or more. Symptoms include headache, muscle and bone pain, ataxia, skin disorders, alopecia, liver toxicity, and hyperlipidaemia. Not all the chronic symptoms are reversible. Vitamin A is teratogenic and pregnancy intakes should not exceed 3 mg/d. There is no risk of toxicity from carotenoids in foods although large intakes can → yellow discoloration of the skin.

❗ Vitamin A supplements should not be taken during pregnancy (see 📖 Chapter 2, 'Dietary reference values and food-based dietary guidelines', p. 19, and 📖 Chapter 12, 'Diet before and during pregnancy', p. 217).
Liver is not recommended during pregnancy.

Table 6.1 Reference nutrient intakes (RNI) for all ages and average daily intakes for adult men and women for vitamin A provided by food (μg retinol equivalent/day)*

Age (years)	RNI
0–1	350
1–3	400
4–10	500
11–14	600
Males 15+	700
Females 15+	600
Pregnancy	+100
Lactation	+350
	Average daily intake UK
Men	1034
Women	1070

* Source for RNIs, Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. HMSO, London. Source for average daily intakes for adults, Bates, B., Lennox, A., and Swan, G. (2010). *The National Diet and Nutrition Survey: Headline results from Year 1 of the Rolling Programme (2008/2009)*. Food Standards Agency, London.

Table 6.2 Contribution of foods to vitamin A intake*

Food group	% Daily intake
Meat and meat products	28
Liver and liver products	21
Vegetables	27
Cooked carrots	12
Milk and milk products	14
Fat spreads	10

* Source Henderson, L., Irving, K., and Gregory, J. (2003). *The National Diet and Nutrition Survey: adults aged 19 to 64 years*. Vol. 3 *Vitamin and mineral intake and urinary analytes*. HMSO, London.

Box 6.1 Good food sources of vitamin A

Most concentrated sources of retinol in the diet

- Liver and liver products
- Kidney and offal
- Oily fish and fish liver oils
- Eggs

Most concentrated sources of β carotene in the diet

- Carrots
- Red peppers
- Spinach
- Broccoli
- Tomatoes

Vitamin E

Eight naturally occurring forms of vitamin E are synthesized in plants: four tocopherols (alpha, α-, beta, β-; delta, δ-; and gamma, γ-) and four tocotrienols (α, β, δ, and δ). α tocopherol has the highest biological activity and is used as the standard against which the activity of other forms is measured. Synthetic vitamin E is a mixture of isomers and has biological activities ranging from 20 to 80%.

Function

- *Antioxidant:* Vitamin E is a powerful antioxidant and protects cell membranes and lipoproteins from damage by free radicals.
- Maintenance of cell membrane integrity.
- Regulation of prostaglandin synthesis.
- DNA synthesis.

Measurement

Plasma concentration is the simplest measure and a direct indicator of status. Acceptable levels of intake are indicated by values of 5–20 μg/ml in adults and children aged 12 years and over, and 3–15 μg/ml for younger children.

Deficiency

Experimental, symptomatic vitamin E deficiency has not been induced in humans. Evidence for the essentiality of vitamin E in humans is provided by a genetically-inherited disease familial isolated vitamin E (FIVE) deficiency. Sufferers develop reduced tendon reflexes by 3–4 years of age. By early adolescence they display symptoms of the nervous system including loss of touch and pain sensation, unsteady gait, loss of co-ordination, and impaired eye movement. In conditions that → chronic or severe fat malabsorption, cystic fibrosis, cholestatic liver disease, and abetalipoproteinaemia, similar symptoms may develop (especially in children) that can be corrected by vitamin E supplementation (5–25 IU/day).

Requirement and intake

See Tables 6.3 and 6.4, and Box 6.2. Vitamin E requirements are influenced by the amount of polyunsaturated fatty acids (PUFA); it is estimated 0.4 mg α-tocopherol is required per gram dietary intake of PUFA. The average adult diet in the UK contains 7% energy from PUFA, which would mean a vitamin E requirement of 6 mg for women and 8 mg for men. Milk formulas should not be <0.3 mg α-tocopherol equivalents/100 ml reconstituted feed and not <0.4 α-tocopherol equivalents/g PUFA.

Toxicity

Vitamin E has low toxicity, but at very high doses it acts as an antagonist to vitamins A, D, and K. Symptoms of toxicity include headache, nausea, muscle weakness, double vision, and creatinuria, and gastrointestinal (GI) disturbances have been reported at intakes greater than 900 mg/kg of the diet.

Table 6.3 Average daily intakes of vitamin E (mg) for adult men and women provided by food (α-tocopherol equivalents)*

	Average daily intake UK
Men	10.6
Women	8.1

* Henderson, L., Irving, K., and Gregory, J. (2003). *The National Diet and Nutrition Survey: adults aged 19 to 64 years.* Vol. 3. *Vitamin and mineral intake and urinary analytes.* HMSO, London.

Table 6.4 Contribution of foods to vitamin E intake*

Food	% Daily intake
Fat spreads	18
PUFA reduced fat spread	8
Cereals and cereal products	17
Vegetables (excluding potatoes)	13
Potatoes and savoury snacks	13
Meat and meat products	11

* Source: Henderson, L., Irving, K., and Gregory, J. (2003). *The National Diet and Nutrition Survey: adults aged 19 to 64 years.* Vol. 3 *Vitamin and mineral intake and urinary analytes.* HMSO, London.

Box 6.2 Good food sources of vitamin E

- Wheat germ oil
- Almonds
- Sunflower seeds and oil
- Safflower oil
- Hazelnuts
- Peanuts and peanut butter
- Corn oil

Vitamin D (calciferols)

The term vitamin D refers to two molecules, ergocalciferol (D_2) and cholecalciferol (D_3). Cholecalciferol is the most effective form of vitamin D and is manufactured in the skin by the action of ultraviolet radiation on 7-dehydrocholesterol. Dietary ergocalciferol and cholecalciferol are biologically inactive and are activated to 25-hydroxyvitamin D in the liver (this has a limited amount of biological activity). Further conversion in the kidney results in the production of the more active form 1,25-dihydroxyvitamin D (calcitrol).

Function

- 1,25-dihydroxyvitamin D maintains plasma Ca by controlling Ca absorption and excretion. Vitamin D and its metabolites are also involved in bone mineralization.
- Children with vitamin D deficiency (rickets) often have impaired immune function that is corrected by the administration of vitamin D.
- It has recently been postulated that vitamin D may inhibit cell proliferation in some forms of cancer.

Measurement

Vitamin D status is assessed by the measurement of plasma 25-hydroxyvitamin D, normal values above 27.5 nmol/l. Plasma vitamin D levels vary with the seasons, being highest in the summer and lowest in winter. Plasma Ca and phosphate fall in severe deficiency and alkaline phosphatase is elevated in mild and severe deficiency states.

Deficiency

Severe deficiency results in rickets in children, which is characterized by reduced calcification of bone epiphyses. It results in skeletal deformities, bone pain, and muscle weakness. In adults deficiency results in osteomalacia, which leads to bone pain, partial (Looser's zone) fractures, and muscle weakness. People who stay indoors and are fully covered are at risk of deficiency due to lack of ultraviolet radiation from sunlight. Supplements are recommended for housebound elderly and some ethnic groups, e.g. Asian and Muslim women due to low sun exposure (see 📖 Chapter 12, 'Dietary reference values and dietary guidelines during pregnancy', p. 222). Malabsorption ↑ the risk of deficiency.

Requirement and intake

See Tables 6.5 and 6.6, and Box 6.3.

Toxicity

Excessive exposure to sunlight does not → vitamin D toxicity as excess D_3 is converted to inert products. Overdose with supplements results in hypercalcaemia, which has symptoms of thirst and anorexia and is accompanied by the risk of soft tissue calcification and urinary Ca stones.

Table 6.5 Reference nutrient intakes (RNI) for all ages and average daily intakes of vitamin D for adult men and women provided by food (μg/day)*

Age (years)	RNI
0–6 months	8.5
7–12 months	7.0
1–3	7.0
4–65	0†
65+	10
Pregnancy	+10
Lactation	+10
Average daily intake UK	
Men	3.1
Women	2.7

* Source for RNI's Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. HMSO, London. Source for average daily intakes for adults, Bates, B., Lennox, A., and Swan, G. (2010). *The National Diet and Nutrition Survey: Headline results from Year 1 of the Rolling Programme (2008/2009)*. Food Standards Agency, London.

† Certain at risk groups or individuals may require dietary vitamin D.

Table 6.6 Contribution of foods to vitamin D intake*

Food group	% Daily intake
Fish and fish dishes	25
Oily fish	24
Meat and meat products	22
Cereals and cereal products	21
Fat spreads	17
Reduced fat spreads	8

* Source: Henderson, L., Irving, K., and Gregory, J. (2003). *The National Diet and Nutrition Survey: adults aged 19 to 64 years.* Vol. 3 *Vitamin and mineral intake and urinary analytes.* HMSO, London.

Box 6.3 Good food sources of vitamin D

Cod liver oil
Oily fish (salmon, mackerel, etc.)
Milk
Margarine
Breakfast cereals
Eggs
Liver

Vitamin K

Naturally occurring vitamin K can be classified into two groups. The major form of vitamin K_1 (phylloquinine) is found in plants while the vitamin K_2 group of compounds (menaquinones) are synthesized by intestinal bacteria.

Function

- Vitamin K promotes the synthesis of γ-carboxyglutamic acid (Gla) in the liver. Gla is an essential part for prothrombin (factor II) and other coagulation factors (VII, IX, and X). Vitamin K is ∴ essential for blood coagulation.
- Other proteins contain Gla and require vitamin K for their synthesis. These include osteocalcin, a bone protein made by osteoblasts.

Measurement

Traditionally, vitamin K deficiency screening was based on coagulation assays of the levels of the active forms of coagulation proteins that require vitamin K. To entirely eliminate a diagnosis of congenital vitamin K deficiency it is necessary to conduct individual factor assays. It is now possible to assay for undercarboxylated vitamin K dependent proteins that are produced when vitamin K is in short supply or blocked by antagonists such as warfarin. A few specialist centres are now able to assay plasma and tissues levels directly by high performance liquid chromatography.

Deficiency

Vitamin K deficiency is characterized by poor blood clotting and results in low prothrombin activity. New born babies are given an injection of vitamin K at birth. Infants are born with very low stores and due to sterility of their intestines do not have bacteria producing vitamin K. Deficiency is rare in adults, but does occur in patients with obstructive jaundice as lack of bile can → poor absorption of vitamin K. The anticoagulants warfarin and dicoumarol can → a deficiency as their mode of action is to block some of the enzymes that recycle vitamin K in the liver.

Requirement and intake

See Box 6.4. Studies into vitamin K requirements are not entirely satisfactory as it is difficult to induce deficiency solely by dietary manipulation. It is suggested that the requirements are between 0.5 and 1.0 μg per kg/day. Determination of vitamin K levels in foods and unreliability of estimates of intake in the UK means that a consensus on usual intake is not available. Studies in the USA suggest that intakes vary between 30 and 100 μg/day.

Toxicity

Large intakes of naturally occurring vitamin K do not appear to be toxic. Synthetic preparation of vitamin K_3 (menadione) is used to treat intracranial and pulmonary haemorrhage in premature infants and overdosage can → liver overload and brain toxicity.

❗ Supplements containing vitamin K should not be taken when taking anticoagulant drugs, e.g. warfarin (see 📖 Chapter 38, 'Drug–nutrient interactions', p. 738).

Box 6.4 Good food sources of vitamin K

- Green leafy vegetables (spinach, broccoli, cabbage, and kale).
- Vegetable oils especially soya bean oil.
- Eggs.
- Meat.
- Dairy products.

Vitamin C (ascorbic acid)

Most animals can synthesize vitamin C from glucose or galactose; humans, primates, guinea-pigs, Indian fruit-eating bats, and some birds lack this ability and it is an essential nutrient in these species. L-ascorbic acid and L-dehydroascorbic acid are both biologically active forms of vitamin C.

Function

Vitamin C is a powerful reducing agent (antioxidant) and is essential for many oxidation-reduction reactions.

- Vitamin C is required for the synthesis of collagen, the main protein in connective tissue and ∴ essential for the maintenance of muscles, tendons, arteries, bone, and skin. It is essential for the normal functioning of enzymes involved in collagen synthesis.
- The hydroxylation of dopamine to the neurotransmitter noradrenaline requires vitamin C.
- Vitamin C is required for the production of carnitine. Low levels of carnitine are associated with fatigue and muscle weakness.
- Various peptide hormones and releasing factors require activation by a vitamin C dependent enzyme.
- Numerous other enzymes need vitamin C; these enzymes control many functions including the synthesis of bile and the metabolism of drugs and carcinogens by the liver.
- Vitamin C enhances the absorption of Fe when consumed in the same meal.

Measurement Vitamin C status is assessed by measurement in plasma and leucocytes; plasma levels are the most practical measure of status. Plasma levels <11 mmol/l show deficiency, >17 mmol/l are adequate. Leucocyte levels of >2.8 pmol/10^6 cells are adequate.

Deficiency

Vitamin C deficiency is uncommon except in populations where there is prolonged lack of fruit and vegetables. Deficiency of vitamin C is characterized by abnormalities of the connective tissue including poor wound healing, which are described by the term scurvy. Weakness, fatigue, bleeding gums (gingival), hyperkeratosis, and skin haemorrhages are symptoms of scurvy.

Requirement and intake See Tables 6.7 and 6.8, and Box 6.5) Regular smoking ↑ vitamin C turnover and it is estimated that smokers require 80 mg/day.

Toxicity High doses (1–10 g/day) of vitamin C are sometimes taken in the belief that such doses can prevent the common cold. There is no evidence to support this hypothesis although they may reduce the severity of symptoms to an extent. Sudden cessation of high dose supplements may precipitate rebound scurvy. Intakes at such high levels have been associated with diarrhoea and ↑ risk of kidney oxalate stone formation.

Table 6.7 Reference nutrient intakes (RNI) for all ages and average daily intakes for adult men and women for vitamin C provided by food (mg/day)*

Age (years)	RNI
0–1	25
1–10	30
11–14	35
Males 15+	40
Females 15+	40
Pregnancy	+10†
Lactation	+30
Average daily intake UK	
Men	96
Women	92

*Source for RNIs, Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. HMSO, London. Source for average daily intakes for adults, Bates, B., Lennox, A., and Swan, G. (2010). *The National Diet and Nutrition Survey: Headline results from Year 1 of the Rolling Programme (2008/2009)*. Food Standards Agency, London.

†Last trimester only.

Table 6.8 Contribution of foods to vitamin C intake*

Food group	% Daily intake
Drinks	27
Fruit juice	19
Soft drinks, including low calorie	8
Vegetables excluding potatoes	22
Fruit and nuts	19
Potatoes and savoury snacks	15

* Source: Henderson, L., Irving, K., and Gregory, J. (2003). *The National Diet and Nutrition Survey: adults aged 19 to 64 years*. Vol. 3 *Vitamin and mineral intake and urinary analytes*. HMSO, London.

Box 6.5 Good food sources of vitamin C

- Kiwi fruit
- Citrus fruit (oranges, lemons, satsumas, clementines, etc.)
- Black currants
- Sweet potato
- Guava
- Mango
- Papaya
- Pepper
- Brussels sprouts
- Broccoli

Riboflavin (vitamin B_2)

Function

Riboflavin is part of two coenzymes that are both oxidizing agents: flavin mononucleotide (FMN) and flavin adenine dinucleotide (FAD). FMN and FAD are contained in flavoproteins, which are involved in many oxidation-reduction reactions in many metabolic pathways. The functions of riboflavin include:

- Promotion of normal growth;
- Assisting synthesis of steroids, red blood cells, and glycogen;
- Maintenance of mucous membranes, skin, eyes, and the nervous system;
- Aiding Fe absorption.

Measurement

Riboflavin status can be assessed by the measurement of urinary excretion or by measurement of erythrocyte glutathione reductase activity coefficient (EGRA). FAD is a co-factor for EGR and its activity is directly correlated to riboflavin status. EGRA is the method of choice as it reflects tissue saturation and long-term riboflavin status. Levels <1.3 are acceptable. Recently, doubts about the validity of EGRA in pregnancy and exercise have been expressed.

Deficiency

- Lesions of the mucosal surfaces of the mouth, angular stomatitis, cheilosis, glossitis, and magenta tounge, surface lesions of the genitalia, seborrhoeic skin lesions, and vascularization of the cornea.
- Riboflavin deficiency is often accompanied by other nutrient deficiencies, e.g. pellagra.
- In animal studies deficiency is associated with poor growth of the young and it is probable that similar effects occur in human neonates.
- Severe deficiency is unlikely in the UK but the elderly, anorexia nervosa sufferers, and chronic 'dieters' are at risk.

Sources in the diet

Riboflavin is unstable when exposed to ultraviolet light and up to 70% will be lost from milk during 4 h exposure to sunlight.

Requirement and intake

See Tables 6.9 and 6.10, and Box 6.6.

Toxicity

Toxicity is low due to the small amount that can be absorbed by the GI tract in a single dose.

Table 6.9 Reference nutrient intakes (RNI) for all ages and average daily intakes for adult men and women for riboflavin (mg/day)*

Age (years)	RNI
0–1	0.4
1–10	0.6–1.0
Males 11–14	1.2
Males 15+	1.3
Females 11+	1.1
Pregnancy†	+0.3
Lactation	+0.5
Average daily intake UK	
Men	1.88
Women	1.40

* Source for RNIs, Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. HMSO, London. Source for average daily intakes for adults, Bates, B., Lennox, A., and Swan, G. (2010). *The National Diet and Nutrition Survey: Headline results from Year 1 of the Rolling Programme (2008/2009)*. Food Standards Agency, London.

† Last trimester only.

Table 6.10 Contribution of foods to riboflavin intake*

Food group	% Daily intake
Milk and milk products	33
Semi-skimmed milk	16
Cereals and cereal products	24
Meat and meat products	15
Drinks	10

* Source: Henderson, L., Irving, K., and Gregory, J. (2003). *The National Diet and Nutrition Survey: adults aged 19 to 64 years*. Vol. 3 *Vitamin and mineral intake and urinary analytes*. HMSO, London.

Box 6.6 Good food sources of riboflavin

- Eggs
- Milk and milk products
- Liver and kidney
- Yeast extracts
- Fortified breakfast cereals

Niacin (nicotinamide, nicotinic acid, vitamin B_3)

Niacin is the generic term for a group of compounds that prevent pellagra. Nicotinamide and nicotinic acid both occur in food, but have different physiological properties. Approximately 50% of niacin in the body is synthesized from the amino acid tryptophan. Sixty milligrams of tryptophan are equivalent to one milligram of niacin or 1 NE.

Function

Nicotinamide is incorporated into the pyridine nucleotide coenzymes nicotinamide adenine dinucleotide (NAD) and nicotinamide adenine dinucleotide phosphate (NADP). The coenzymes are involved in numerous oxidoreductase reactions including glycolysis, fatty acid metabolism, tissue respiration, and detoxification.

Measurement

Niacin status is most often assessed by the measurement of its metabolites *N′*-methylnicotinamide (NMN) and *N′*-methyl-2-pyridone-5-carboxamide. These metabolites are ↓ in niacin deficiency. A deficiency should be considered when the NMN to creatinine ratio is <1.5 mmol/mol. This assay requires 24-h urine collection, which may be problematical. Other measures of niacin status include red cell NAD concentration and fasting plasma tryptophan.

Deficiency

Deficiency of niacin is known as pellagra and classically it is characterized by the three Ds.

- *Dermatitis:* skin that is exposed to the sun becomes inflamed, which progresses to pigmentation, cracking, and peeling. The neck is frequently involved and the distinctive distribution of skin lesions is known as Casal's collar.
- *Diarrhoea:* this is often accompanied by an inflamed tongue.
- *Dementia:* symptoms range from mild confusion and disorientation to mania, occasionally psychoses may occur that require hospitalization.

Pellagra is rare in the UK, but still occurs in parts of Africa.

Requirement and intake

See Tables 6.11 and 6.12, and Box 6.7.

Toxicity

Nicotinic acid intakes of 200 mg/day → flushing due to vasodilatation, higher doses → dilatation of non-cutaneous vessels and can cause hypotension. Doses of 1–2 g/day are used in the treatment of hypertriglyceridaemia and hypercholesterolaemia. Larger doses (3–6 g/day) cause reversible liver toxicity with changes in liver function, carbohydrate tolerance, and uric acid metabolism.

Table 6.11 Reference nutrient intakes (RNI) for all ages and average daily intakes for adult men and women for niacin provided by food (mg niacin equivalent/1000 kcal)*

Age (years)	RNI
All ages	6.6
Lactation†	+2.3 mg/day
Average daily intake UK	
Men	20
Women	20

*Source for RNIs, Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom.* HMSO, London. Source for average daily intakes for adults, Bates, B., Lennox, A., and Swan, G. (2010). *The National Diet and Nutrition Survey: Headline results from Year 1 of the Rolling Programme (2008/2009)*. Food Standards Agency, London.

†No increment is recommended during pregnancy.

Table 6.12 Contribution of foods to niacin intake*

Food group	% Daily intake
Meat and meat products	34
Chicken, turkey, and dishes including coated	15
Cereals and cereal products	27
White bread	7
Cheese	12

*Henderson, L., Irving, K., and Gregory, J. (2003). *The National Diet and Nutrition Survey: adults aged 19 to 64 years.* Vol. 3. *Vitamin and mineral intake and urinary analytes.* HMSO, London.

†No increment is recommended during pregnancy.

Box 6.7 Good food sources of niacin

- Beef
- Pork
- Chicken
- Wheat flour
- Maize flour
- Eggs
- Milk

Thiamin (vitamin B_1)

Function

Thiamin forms part of the coenzyme thiamine pyrophosphate (TPP), which is involved in major decarboxylation steps in the following pathways.

- Pyruvate → acetyl CoA at the entry to the citric acid cycle.
- α-Ketoglutarate → succinyl CoA, halfway round the citric acid cycle.
- Transketolase reactions in the hexose monophosphate shunt.
- Catabolism of branch chain amino acids, leucine, isoleucine, methionine, and valine.
- Thiamin is needed for the metabolism of fat, carbohydrate, and alcohol.

Measurement

Red cell transketolase assay is the most frequently used measure of thiamin status. It is essential to use fresh, heparinized whole blood. Thiamin deficiency is indicated by ↑ in transketolase activity after the addition of TPP. Higher values indicate greater deficiency; in Wernicke's encephalopathy activity can be ↑ by 70–100%.

Deficiency

Thiamin deficiency manifests as beriberi and Wernicke–Korsakoff syndrome. Beriberi is usually classified as either 'wet' (cardiac) or 'dry' (neurological). They rarely occur together.

- Wet beriberi is the acute form of the disease and is characterized by high output cardiac failure, bounding pulse, warm extremities, peripheral oedema, and cardiac dilatation.
- Dry beriberi is the chronic form of the disease and is characterized by progressive, peripheral neuropathy. Foot drop is accompanied by loss of sensation in the feet and absent knee jerk reflexes.
- Wernicke–Korsakoff syndrome is seen in chronic alcoholics who have a poor diet. It is characterized by confusion, low levels of consciousness, and poor co-ordination (Wernicke's encephalopathy). Paralysis of one or more of the external movements of the eye is a diagnostic criteria. Memory loss (Korsakoff's psychosis) often follows the encephalopathy.

Requirement and intake

See Tables 6.13 and 6.14, and Box 6.8. Thiamin requirements are related to energy metabolism.

Sources in the diet

Thiamin is widely distributed in the diet. In the UK and many other industrialized countries bread flour is fortified with thiamine by law and, in practice, it is added to many breakfast cereals.

Toxicity

Chronic intakes of more than 3 g/day are associated with symptoms of toxicity; these include headache, irritability, insomnia, weakness, tachycardia, and pruritis. Regular large intakes can → an allergic reaction.

Table 6.13 Reference nutrient intakes (RNI) for all ages and average daily intakes for adult men and women for thiamin provided by food (mg/1000 kcal)*

Age (years)	RNI
0–12 months	0.3
1–50 +	0.4†
Average daily intake UK	
Men	1.69 mg/day
Women	1.30 mg/day

*Source for RNIs, Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. HMSO, London. Source for average daily intakes for adults, Bates, B., Lennox, A., and Swan, G. (2010). *The National Diet and Nutrition Survey: Headline results from Year 1 of the Rolling Programme (2008/2009)*. Food Standards Agency, London.

†No increment for pregnancy or lactation.

Table 6.14 Contribution of foods to thiamin intake*

Food group	% Daily intake
Cereals and cereal products	34
White bread	9
Meat and meat products	21
Vegetables excluding potatoes	15
Potatoes and savoury snacks	13

*Henderson, L., Irving, K., and Gregory, J. (2003). *The National Diet and Nutrition Survey: adults aged 19 to 64 years*. Vol. 3. *Vitamin and mineral intake and urinary analytes*. HMSO, London.

Box 6.8 Good food sources of thiamin

- Cereal products (including breakfast cereals and bread)
- Yeast and yeast products
- Pulses
- Nuts
- Pork and other meats
- Vegetables
- Milk

Folate (folic acid)

Folic acid (pteroyl glutamic acid) is the synthetic form of the vitamin and is the parent molecule for a number of derivatives known as folates. Folic acid is a very stable molecule with high biological activity. It is used in the fortification of foods and in supplements. Folates occur naturally as a number of tetrahydrofolates, which have variable biological activities.

Function

- Folates are involved in a number of single carbon transfer reactions particularly in the synthesis of purines, pyrimidines, glycine, and methionine. It is ∴ essential for the synthesis of DNA and RNA.
- The folate derivative 5-methyl tetrahydrofolate requires vitamin B_{12} to enable the use of methionine synthase in the synthesis of methionine and tetrahydrofolate.

Measurement

Recent intake is assessed by serum folate; normal levels are 2.0–11.0 μg/l. Cellular status is reflected by red cell folate; normal levels are 150–700 μg/l.

Deficiency

Dietary deficiency is seen occasionally, but secondary deficiency is fairly common. Secondary deficiency can result from malabsorption, the use of certain drugs, and in late pregnancy and some disease states including leukaemia. Deficiency results in megaloblastic anaemia with abnormal neutrophil nuclei and giant platelets. There may also be infertility and diarrhoea.

Benefits of extra folate

Folate supplements in early pregnancy (before the neural tube closes at 24–28 days) have been shown to reduce neural tube defects (see 📖 Chapter 12, 'Pre- and periconceptual nutrition in women', p. 218). In many countries flour is supplemented with folate by law. SACN have recommended the supplementation of flour in the UK but this is waiting for government approval.

Large doses (200 μ/day) reduce plasma levels of homocysteine. Raised plasma homocysteine is a risk factor for cardiovascular disease.

Requirement and intake

See Tables 6.15 and 6.16, and Box 6.9.

Toxicity

The toxicity of folates is low. Folate supplements given to patients with developing vitamin B_{12} deficiency may obscure diagnosis.

Drug interactions

Chronic use of anticonvulsants has been associated with folate deficiency. Other drugs that interfere with folate metabolism include cytotoxic chemotherapy agents (methotrexate, aminopterin), and antimalarial (pyrimethamine) and antibacterial (co-trimoxazole) agents.

Table 6.15 Reference nutrient intakes (RNI) and average daily intakes for adult men and women for folate provided by food (μg/day)*

Age (years)	RNI
0–1	50
1–3	70
4–6	100
7–10	150
Males 11+	200
Females 11+	200
Pregnancy	+100†
Average daily intake UK	
Men	320
Women	242

*Source for RNIs, Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. HMSO, London. Source for average daily intakes for adults, Bates, B., Lennox, A., and Swan, G. (2010). *The National Diet and Nutrition Survey: Headline results from Year 1 of the Rolling Programme (2008/2009)*. Food Standards Agency, London.

†To prevent first occurrence of NTD: 400 μg during preconception and until the 12th week of pregnancy (on prescription or over the counter). To prevent recurrence of NTD: 5000 μg during preconception and until the 12th week of pregnancy (on prescription only).

Table 6.16 Contribution of foods to folate intake*

Food group	% Daily intake
Cereals and cereal products	33
Whole grain and high fibre breakfast cereals	8
Vegetables excluding potatoes	15
Drinks	14
Potatoes and savoury snacks	12

* Henderson, L., Irving, K., and Gregory, J. (2003) *The National Diet and Nutrition Survey: adults aged 19 to 64 years*. Vol. 3. *Vitamin and mineral intake and urinary analytes* HMSO, London.

Box 6.9 Good food sources of folate

- *Rich sources >100 μg per serving:* Brussels sprouts, kale, spinach
- *Good sources 50–100 μg per serving:* fortified bread and breakfast cereals, broccoli, cabbage, cauliflower, chickpeas, green beans, icebergs, lettuce, kidneys, beans, peas, spring greens
- *Moderate sources 15–15 μg per serving:* potatoes, most other vegetables, most fruits, most nuts, brown rice, wholegrain pasta, oats, bran, some breakfast cereals, cheese, yoghurt, milk, eggs, salmon, beef, game

Vitamin B_6

There are three naturally occurring forms of vitamin B_6: pyridoxine, pyridoxal, and pyridoxamine. These three vitamers are interconvertible in the body.

Function

The three vitamers can be converted to the coenzyme pyridoxal-5-phosphate, which is involved in amino acid metabolism. These reactions include:

- Transamination of amino acids to produce keto acids and synthesis of non-essential amino acids.
- Decarboxylation to yield biologically active amines, e.g. neurotransmitters (adrenaline, noradrenaline, serotonin, and γ–amino butyric acid) and histamine.
- Porphyrin synthesis, including haemoglobin.

Vitamin B_6 is also involved in the conversion of glycogen to glucose in muscles, the conversion of tryptophan to niacin, and in hormone metabolism.

Measurement

Vitamin B_6 status can be assessed by the measurement of plasma concentrations of pyridoxal phosphate (normal values are above 30 nmol/l) or total vitamin B_6. Activation of erthyrocyte transaminases can be a useful measure. Metabolism of test doses of methionine can be used but is technically difficult.

Deficiency

Severe deficiency of vitamin B_6 is rare. One outbreak was reported in 1954 due to errors in the manufacture of infant's formula feed. The affected infants suffered seizures that responded to treatment with vitamin B_6. Patients suffering from malabsorption, receiving dialysis, or alcoholics are at risk of deficiency. Clinical signs include lesions of the lips and corners of the mouth, and inflammation of the tongue. Peripheral neuropathy may be a sign of vitamin B_6 deficiency, but as vitamin B_6 deficiency is usually associated with other vitamin deficiency the neuropathy may be the result of thiamin deficiency. Sideroblastic (microcytic, hypochromic) anaemia due to poor haem synthesis is associated with vitamin B_6 deficiency.

Requirement and intake

See Tables 6.17 and 6.18, and Box 6.10. Due to the importance of vitamin B_6 in amino acid metabolism requirements are linked to protein intake.

Toxicity

Intakes of 500 mg/day and above have been associated with peripheral neuropathy and loss of sensation in the feet has been reported at higher doses (from supplements). The DH recommends that the daily dose of vitamin B_6 should not exceed 10 mg/day.

Drug interactions

Urinary excretion of vitamin B_6 is ↑ by isoniazid (used to treat tuberculosis). Penicillamine, L-dopa, and cycloserine are vitamin B_6 antagonists.

Table 6.17 Reference nutrient intakes (RNI) for all ages and average daily intakes for adult men and women for vitamin B_6 provided by food (μg/g protein)*

Age (years)	RNI
0–6 months	8
7–9 months	10
10–12 months	13
1–50 +	15†
Average daily intake UK	
Men	32
Women	30

*Source for RNIs, Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. HMSO, London. Source for average daily intakes for adults, Bates, B., Lennox, A., and Swan, G. (2010). *The National Diet and Nutrition Survey: Headline results from Year 1 of the Rolling Programme (2008/2009)*. Food Standards Agency, London.

†No increment is recommended for pregnancy or lactation.

Table 6.18 Contribution of foods to vitamin B_6 intake*

Food group	% Daily intake
Cereals and cereal products	21
Meat and meat products	21
Potatoes and savoury snacks	19
Drinks	11
Beer and lager	8

*Source for average daily intakes for adults, Henderson, L., Irving, K., and Gregory, J. (2003) *The National Diet and Nutrition Survey: adults aged 19 to 64 years*. Vol. 3. *Vitamin and mineral intake and urinary analytes*. HMSO, London.

Box 6.10 Good food sources of vitamin B_6

- Meat
- Wholegrain cereals
- Fortified cereals
- Bananas
- Nuts
- Pulses

Cobalamin B_{12}

Cobalamin is a complex molecule that contains cobalt; it occurs naturally in usual forms. Cyanocobalamin is the commercially available form, which is converted to the natural forms. It requires salivary haptocorrin and 'intrinsic factor' to be absorbed. 'Intrinsic factor' is secreted by the parietal cells of the stomach.

Function

The functions of vitamin B_{12} include:

- Recycling of folate coenzymes.
- Normal myelination of nerves.
- Synthesis of methionine from homocysteine.

Measurement

Serum B_{12} status is assessed by radioligand binding or microbiological assay. Levels >150 pmol/l are considered normal. Absorption of B_{12} is assessed by the Schilling test. Absorption of vitamin B_{12} labelled with radioactive cobalt is measured with and without 'intrinsic factor'.

Deficiency

Vitamin B_{12} does not occur in plant foods, and ∴ vegans and strict vegetarians are at risk of deficiency. Few exhibit deficiency symptoms as vitamin B_{12} is also manufactured by intestinal bacteria. Children on macrobiotic diets are at particular risk. The most common cause of deficiency is malabsorption due to atrophy of the gastric mucosa, which leads to inadequate production of 'intrinsic factor' or diseases of the ileum. Deficiency results in pernicious anaemia (megaloblastic) and/or neurological problems. The anaemia is morphologically the same as that seen in folate deficiency and biochemical tests are necessary to establish the cause. The neuropathy is characterized by loss of sensation and motor power in the lower limbs due to degeneration of myelin. Deficiency is easily corrected by monthly injections (100 μg/m).

Sources in the diet

Vitamin B_{12} is synthesized by micro-organisms and assimilated into the food chain. It occurs naturally in animal products but it can be found in fortified foods such as breakfast cereals.

Requirement and intake

See Tables 6.19 and 6.20, and Box 6.11.

Toxicity

Toxicity has not been reported in humans.

Table 6.19 Reference nutrient intakes (RNI) for all ages and average daily intakes for adult men and women for B_{12} provided by food (μg/day)*

Age (years)	RNI
0–1	0.3–0.4
1–3	0.5
4–6	0.8
7–10	1.0
11–14	1.2
Males 15+	1.5
Females 15+	1.5
Lactation	+0.5†
Average daily intake UK	
Men	6.2
Women	4.8

*Source for RNIs, Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. HMSO, London. Source for average daily intakes for adults, Bates, B., Lennox, A., and Swan, G. (2010). *The National Diet and Nutrition Survey: Headline results from Year 1 of the Rolling Programme (2008/2009)*. Food Standards Agency, London.

†No increment is recommended during pregnancy.

Table 6.20 Contribution of foods to vitamin B_{12} intake*

Food group	% Daily intake
Milk and milk products	36
Semi-skimmed milk	18
Meat and meat products	30
Fish and fish dishes	18
Oily fish	11

*Source for average daily intakes for adults, Henderson, L., Irving, K., and Gregory, J. (2003). *The National Diet and Nutrition Survey: adults aged 19 to 64 years*. Vol. 3. *Vitamin and mineral intake and urinary analytes*. HMSO, London.

Box 6.11 Good food sources of vitamin B_{12}

- Meat and meat products
- Eggs
- Milk and dairy products
- Fish and fish products
- Yeast products and fortified vegetable extracts
- Breakfast cereals (fortified)

Biotin (vitamin B_7)

Of the eight isomers of biotin only D-biotin is biologically active. Biotin is made by bacteria and yeasts. Biotin is obtained from the diet and synthesized by endogenous bacteria in the colon.

Function

Biotin is a coenzyme for several carboxylases involved in fatty acid synthesis and metabolism, gluconeogenesis, and the metabolism of branched chain amino acids.

Measurement

Microbiological assays are available to measure biotin in whole blood or urine. The normal range in whole blood is 0.22–0.75 μg/ml.

Deficiency

Biotin deficiency is rare, but has been reported in patients receiving total parenteral nutrition and should be added to the infusion solution. Deficiency is associated with a scaly dermatitis, glossitis, hair loss, anorexia, depression, and hypercholesterolaemia. It is possible to induce biotin deficiency by the ingestion of large amounts of raw egg white. Egg whites contain the protein avidin, which binds biotin and prevents absorption. The effect is prevented by heating the egg whites.

Requirement and intake

See Table 6.21 and Box 6.12. No studies are available on which recommendations on intake can be based. It is believed that intakes of between 10 and 200 μg/day are safe and adequate.

Toxicity

There have been no reports of biotin toxicity.

Table 6.21 Average daily intakes for adult men and women for biotin (μg/day) in the UK*

Men	41
Women	29

*Henderson, L., Irving, K., and Gregory, J. (2003). *The National Diet and Nutrition Survey: adults aged 19 to 64 years*. Vol. 3. *Vitamin and mineral intake and urinary analytes*. HMSO, London.

Box 6.12 Good food sources of biotin

- Liver
- Kidney
- Milk
- Eggs
- Dairy products

Pantothenic acid (vitamin B_5)

Function

- Pantothenic acid is part of coenzyme A (CoA) and as such is involved in the tricarboxylic acid cycle.
- The pantothenic acid derivative 4′-phosphopantetheine is part of acyl carrier protein.
- It is essential for reactions involved in carbohydrate and lipid metabolism.

Measurement Pantothenic acid can be measured in blood and urine. Normal urinary excretion is 1–15 mg/day and normal blood values are >100 μg/dl.

Deficiency

Spontaneous deficiency of pantothenic acid has not been described. It is possible to induce a deficiency with experimental diets or by administration of the antagonist ω-methypantothenic acid. During World War II malnourished prisoners in the Far East developed 'burning feet' parathaesiae, which responded to treatment with pantothenic acid.

The symptoms of deficiency include a burning sensation in the feet, depression, fatigue, vomiting, and muscle weakness.

Requirement and intake

See Table 6.22 and Box 6.13. Information is not available to derive recommended intakes but intake of 3–7 mg/day is considered adequate.

Sources in the diet Pantothenic acid is widely distributed in food, but highly processed foods do not contain the vitamin.

Toxicity

There are no specific toxic effects, although large doses may cause GI symptoms such as diarrhoea.

Table 6.22 Average daily intakes of pantothenic acid provided by food (mg/day) in UK*

Men	7.2
Women	5.4

*Henderson, L., Irving, K., and Gregory, J. (2003). *The National Diet and Nutrition Survey: adults aged 19 to 64 years.* Vol. 3. *Vitamin and mineral intake and urinary analytes.* HMSO, London.

Box 6.13 Good food sources of pantothenic acid

- Yeast
- Offal
- Peanuts
- Meat
- Eggs
- Green vegetables

Minerals and trace elements: introduction

Table 6.23 shows the minerals and trace elements known to be essential to humans. Fl is semi-essential in that no physiological requirement is known to exist, but there are known beneficial effects. Minerals are required in grams or milligrams, while trace elements are required in microgram amounts. Elements that are of biological importance, but are not currently considered essential include nickel, vanadium, cobalt, and boron.

Criteria for essentiality of minerals and trace elements

- Present in healthy tissues.
- Concentration must be relatively constant between different organisms.
- Deficiency induces specific biochemical changes.
- Deficiency changes are accompanied by equivalent abnormalities in different species.
- Supplementation corrects the abnormalities.

Table 6.23 Essential minerals and trace minerals

Minerals	Trace elements
Calcium	Copper
Phosphorus	Chromium
Magnesium	Manganese
Sodium	Molybdenum
Potassium	Selenium
Iron	Iodine
Zinc	
Fluorine	

Calcium

Ca is the most abundant mineral in the human body (1.4 g/kg) and 99% is deposited, usually as hydroxyapatite, in bones and teeth where it provides structural rigidity. Ca plasma levels are tightly controlled by parathyroid hormone, 1,25 dihydroxycholecalciferol, and calcitonin. Plasma Ca levels are also controlled by the vitamin D metabolite $1,25(OH)_2D_3$, which controls the active absorption of calcium from the intestine and osteoclastic resorption of bone. Causes of abnormal Ca plasma concentrations are shown in Table 6.24.

Function

- Structural rigidity of bones and teeth, as hydroxyapatite.
- Intracellular signalling control of muscles and nerves.
- Blood clotting.
- Co-factor for enzymes, e.g. lipase.

Measurement

The normal plasma range for Ca is 2.15–2.55 mmol/l and 50% of plasma Ca is bound to protein, principally albumin; ∴ plasma value is frequently given as a corrected value. Plasma Ca levels are tightly controlled and are not usually affected by dietary insufficiency in healthy adults. Hypocalcaemia results in symptoms, such as tetany and cardiac arrhythmias. Bone mineral concentration can be measured by neutron activation analysis and dual X-ray absorptiometry can be used to directly measure bone mineral density.

Deficiency

In early adulthood Ca deficiency can → stunted growth and failure to achieve peak bone mass. Peak bone mass is achieved in early adulthood and is determined by genetic factors, use of the skeleton, and nutritional factors including Ca intake. Failure to achieve peak bone mass is a risk factor for osteoporosis in later life. Poor Ca absorption due to vitamin D deficiency leads to rickets in children (see 📖 this chapter, 'Vitamin D (calciferols)', p. 100).

Requirement and intake

See Table 6.25.

Sources in the diet

See Tables 6.26 and Box 6.14. Ca absorption is variable; Ca in milk and dairy foods is more readily absorbed than Ca in plants. The presence of phytates in cereals and oxalates in leafy green vegetables inhibits absorption.

Toxicity

Accumulation in blood and tissues due to dietary excess is virtually unknown due to the tight homeostatic control of Ca. Hypercalcaemia is usually the result of an abnormality in this control as shown in Table 6.24. Milk alkali syndrome (MAS) results from excessive intake of Ca and alkali as antacid tablets, Ca supplements, and milk (which provides vitamin D and → ↑ absorption). MAS has been reported at Ca carbonate intakes of ≥4 g/day or more. A rare cause of MAS is excessive intake of Ca by the ingestion of betel nut paste containing oyster shells. Intakes of up to 2 g of Ca/day have been shown to be safe.

Table 6.24 Causes of abnormal plasma Ca*

Hypercalcaemia	Hypocalcaemia
Malignant disease	Vitamin D deficiency
Primary hyperparathyroidism	Hypoparathyroidism (and pseudohypoparathyroidism)
Sarcoidosis (and other granulomas)	
Vitamin D overdose	
Milk alkali syndrome	
Immobilization	
Thyrotoxicosis	
Hypercalcaemia of infancy	
Familial hypocalciuric hypercalcaemia	

*Reproduced from Garrow, JS., James, WPT., and Ralph, A. (1999) *Human Nutrition and Dietetics*. table 17.1, p. 135. With permission from Elsevier.

Table 6.25 Reference nutrient intakes for Ca (mg/day) for all ages and average daily intakes (mg) for adult men and women provided by food*

Age (years)†	RNI
0–12 months	525
1–3 years	350
46–years	450
7–10 years	550
Men	
11–18 years	1 000
19 + years	700
Women	
11–18 years	800
19 + years	700
Lactation	+ 550
Average daily intake UK	
Men	931
Women	718

*Source for RNIs, Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. HMSO, London. Source for average daily intakes for adults, Bates, B., Lennox, A., and Swan, G. (2010). *The National Diet and Nutrition Survey: Headline results from Year 1 of the Rolling Programme (2008/2009)*. Food Standards Agency, London.

†There is no recommendation for an increase in Ca intake during pregnancy, Ca absorption ↓ during pregnancy.

Table 6.26 Contribution of foods to Ca intake (NDNS)*

Food group	% Daily intake
Milk and milk products	43
Semi skimmed milk	17
Cheese	11
Cereals and cereal products	30
White bread	13

*Henderson, L., Irving, K., and Gregory, J. (2003). *The National Diet and Nutrition Survey: adults aged 19 to 64 years.* Vol.3. *Vitamin and mineral intake and urinary analytes.* HMSO, London.

Box 6.14 Food and portions that provide approximately 100 mg Ca

- 85 ml milk
- 15 g cheddar cheese
- 50 g yoghurt
- 100 g cottage cheese
- 20 g sardines
- 200 g baked beans
- 3 large slices white bread
- 125 g pulses (e.g. chickpeas)
- 20 g tofu
- 15 g tahini

Phosphorus

Phosphate is present in every cell in the body although 80–85% is found with Ca in hydroxyapatite.

Function

- Skeletal rigidity as the Ca compound hydroxyapatite.
- Energy for metabolism is derived from the phosphate bonds in adenosine triphosphate (ADP).
- Constituent of phospholipids and membranes.
- Constituent of nucleic acids.

Measurement

Serum total phosphate levels are measured by colorimetric methods. The normal adult range is 0.7–1.5 mmol/l.

Deficiency

P deficiency is unlikely to occur as it is present in all plant and animal foods. Hypophosphataemia does occur in poorly managed parenteral nutrition and re-feeding syndrome. Some studies have shown that P deficiency at birth is linked to rickets at a later age. Hypophosphataemia can occur in sepsis, liver disease, alcoholism, diabetic ketoacidosis, and excessive use of aluminium-containing antacids.

Requirement and intake See Tables 6.27 and 6.28 Dietary requirements for P are equal to those for Ca, i.e. 1 mg P:1 mg Ca or 1 mmol P: 1 mmol Ca.

Sources in the diet

Phosphate is present in all natural foods and is present in many additives. Good sources are shown in Box 6.15. Absorption is approximately 60% of intake; it is ↓ by non-starch polysaccharides (NSP). NSP rich diets are also rich in phosphate so compensating for the reduction in absorption.

Toxicity

Intakes above 70 mg/kg body weight may → high serum levels that are above any likely to be taken in foods. Generally ↑ intakes are balanced by ↑ excretion in urine; this is disrupted in renal patients (see 📖 Chapter 29, 'Renal disease', p. 631). The P:Ca ratio should not be above 2.2 mg P to 1 mg of Ca.

Table 6.27 Reference nutrient intakes for P (which are equivalent to those for Ca (mg/day) for all ages and average daily intakes (mg) for adult men and women provided by food*

Age	RNI
0–12 months	525
1–3 years	350
4–6 years	450
7–10 years	550
Men	
11–18 years	1000
19 + years	700
Women	
11–18 years	800
19 + years	700
Lactation	+550
Average daily intake UK	
Men	1493
Women	1112

*Source for RNIS, Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. HMSO, London. Source for average daily intakes for adults, Henderson, L., Irving, K., and Gregory, J. (2003). *The National Diet and Nutrition Survey: adults aged 19 to 64 years*. Vol. 3. *Vitamin and mineral intake and urinary analytes*. HMSO, London.

Table 6.28 Contribution of foods to P intake (NDNS)*

Food group	% Daily intake
Milk and milk products	24
Semi-skimmed milk	9
Cereals and cereal products	23
White bread	5
Breakfast cereals	5
Meat and meat products	21

*Henderson, L., Irving, K., and Gregory, J. (2003). *The National Diet and Nutrition Survey: adults aged 19 to 64 years*. Vol. 3. *Vitamin and mineral intake and urinary analytes*. HMSO, London.

Box 6.15 Good food sources of P

- Milk and dairy products except butter
- Cereals and cereal products
- Meat and meat products
- Fish
- Nuts
- Fruits and vegetables

Iron

There is approximately 4 g Fe in the body of an adult man of which 2.4 g is present as haemoglobin; adult women have approximately 2.1 g of which 1.6 g is haemoglobin. Haemoglobin consists of 4 units: each unit contains 1 haem group and 1 protein chain. Fe is also present in the non-haem form. Fe compounds in the body are shown in Table 6.29.

Transport and absorption

In the free state Fe is toxic; ∴ its transport and storage are closely controlled. Fe is actively absorbed in the duodenum. When the body needs Fe it passes directly through the mucosal cells and is transported by transferrin, with Fe released from old red blood cells, to the bone marrow (80%) and other tissues. If Fe is not required it is stored in the mucosal cells as transferrin. It will be lost in faeces when the mucosal cells are exfoliated. Excess Fe that is absorbed is stored as ferritin or haemosiderin in the liver, spleen, or bone marrow. Fe can be mobilized from these stores when demand is ↑. Haem Fe is absorbed directly into the mucosal cells where Fe is released by haem oxidase and then bound to transferrin. Haem Fe represents 10–15% of Fe intake, but contributes ≥40% total Fe absorbed (Fig. 6.1). Non-haem Fe is poorly absorbed (1–20% of the total absorbed) and is influenced by dietary constituents (see Table 6.30).

Function

- As haemoglobin:
  - transport of oxygen;
  - cell respiration.
- As myoglobin:
  - oxygen storage in muscles.
- Other functions:
  - component of enzymes, including those involved in immune functions, and cytochromes, which are essential for energy production.

Table 6.29 Fe compounds in the body (mg)

	Man (75 kg)	Woman (55 kg)
Functional Fe		
Haemoglobin	2400	1600
Myoglobin	350	230
Haem and non-haem enzymes	150	110
Transferrin-bound Fe	3	2
Total functional Fe	~2900	~1940
Storage Fe		
Ferritin and haemosiderin	500–1500	0–300
Total Fe	~4000	~2100

Table 6.30 Factors influencing Fe absorption

		Increased absorption	Decreased absorption
Haem	Physiological factors	Low Fe status	High Fe status
	Dietary factors	Low haem intake Meat	High haem intake Ca Tannins
Non-haem	Physiological factors	Depleted Fe stores Pregnancy Disease states (aplastic anaemia, haemolytic anaemia, haemochro- matosis)	Replete Fe stores Achlorhydria
	Dietary factors	Vitamin C Meat, fish, seafood Organic acids: ascorbic, citric, lactic, malic, tartaric	Phytate Fe-binding phenolic compounds Inorganic elements: Ca, Mn, Cu, Cd, Co

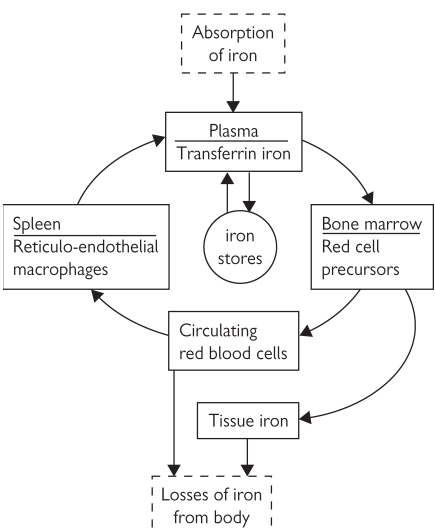


Fig. 6.1 Schematic representation of Fe metabolism.

Measurement

Fe deficiency develops in 3 stages and measurements are appropriate to each stage.

- *Fe depletion:* Fe stores are depleted and serum ferritin levels will fall below 12 μg/l. Other measures of Fe status will be normal.
- *Fe deficient erythropoiesis:* Fe stores are depleted and supply does not meet needs for haemoglobin production. Serum ferritin levels will be low, serum Fe concentration is low, and transferrin saturation is <16%. Haemoglobin within normal range.
- *Fe deficiency anaemia:* haemoglobin levels <11.5 mg/l in women and <13 mg/l in men. Red cells are microcytic and hypochromic. Mean corpuscular volume (MCV) <77 fl and mean cell haemoglobin (MCH) <27 pg.

Requirement and intake

See Tables 6.31 and 6.32.

Deficiency

Fe deficiency anaemia (IDA) is the most common nutritional deficiency in the world. It is estimated that up to 30% of women have IDA, with prevalence of ~8% in developed countries (see 📖 Chapter 13, 'Iron deficiency anaemia in infancy' (p. 264) and 📖 Chapter 20, 'Iron deficiency anaemia globally' (p. 395), and 📖 Chapter 16, 'Is a vegetarian diet risky for health?' (p. 312). Up to 15% of pregnant women are Fe deficient (see 📖 Chapter 12, 'Dietary reference values and dietary guidelines during pregnancy', p. 222).

Physical signs include:

- Pallor of finger nails and mucous membranes in the mouth and under eyelids.
- Koilonychia (spoon shaped nails).
- Tachycardia and in severe cases oedema.
- Fatigue, breathlessness on exertion, insomnia, giddiness, anorexia.
- Paraesthesia of fingers and toes.

Sources in the diet

See Table 6.33 and Box 6.16.

Toxicity

Due to the tight metabolic control dietary excess does not occur. Fe poisoning can occur due to overdose of supplements: the lethal dose in children is 200–300 mg/kg body weight and approximately 100 g in adults. High doses of Fe supplements cause GI symptoms especially constipation, although nausea, vomiting, and diarrhoea may occur. The absorption of other micronutrients, e.g. Zn, are reduced by high dose Fe supplements.

The hereditary disease primary idiopathic haemochromatosis is characterized by high levels of Fe being absorbed. Fe deposits in the liver and heart and may → cirrhosis, liver cancer, congestive heart failure, and eventually death. Treatment requires regular blood removal.

Table 6.31 Reference nutrient intakes for Fe (mg/day) for all ages and average daily intakes (mg) for adult men and women provided by food (NDNS)*

Age†	RNI
0–3 months	1.7
4–6 months	4.3
7–12 moths	7.8
1–3 years	6.9
4–6 years	6.1
7–10 years	8.7
Men	
11–18 years	11.3
19–50 + years	8.7
Women	
11–50 years	14.8
50 + years	8.7
Average daily intake UK	
Men	13.2
Women	10.0

*Source for RNIs, Department of Health (1991). *Dietary reference values for food and nutrients of the United Kingdom*. HMSO, London. Source for average daily intakes for adults, Henderson, L., Irving, K., and Gregory, J. (2003). *The National Diet and Nutrition Survey: adults aged 19 to 64 years*. Vol.3. *Vitamin and mineral intake and urinary analytes*. HMSO, London.

†There is no recommendation for pregnancy and lactation as women should have enough Fe stores that will be enhanced by increased absorption and cessation of menstruation. Women with low Hb levels at the start of pregnancy may require supplementation.

Table 6.32 Contribution of foods to Fe intake (NDNS)*

Food group	% Daily intake
Cereals and cereal products	44
Whole grain and high fibre breakfast cereals	13
Meat and meat products	17
Vegetables excluding potatoes	10

*Henderson, L., Irving, K., and Gregory, J. (2003). *The National Diet and Nutrition Survey: adults aged 19 to 64 years*. Vol. 3. *Vitamin and mineral intake and urinary analytes*. HMSO, London.

Box 6.16 Dietary sources of Fe

Very good sources
- Meat especially offal*
- Fish
- Eggs
- Meat extracts

Good sources
- Bread and flour
- Breakfast cereals
- Vegetables (dark green) and pulses
- Nuts and dried fruit—prunes, figs, apricots
- Yeast extract

*Liver is not recommended in pregnancy due to its high vitamin A content.

Table 6.33 Fe content of 50 g portions of foods

Food	Fe (mg)
Liver—cooked*	5
Liver pate	3
Roast or corned beef	1
Boiled egg	1
Sardines in tomato sauce	2
Wholemeal bread—1 slice	1
Bran flakes—30 g	6
Baked beans	1
Frozen peas	1
Lentils—cooked	1.5
Dark green leafy vegetables—cooked	0.5
Dried apricots	2
Tofu	0.5
Dry roasted peanuts	1

*Liver is not recommended in pregnancy due to its high vitamin A content.

Zinc

Function

- There are more than 200 Zn enzymes in plant and animal tissues including alcohol dehydrogenase, alkaline phosphatase, aldolase, and RNA polymerase. Zn is ∴ involved in digestion, carbohydrate metabolism, bone metabolism, and oxygen transport and it is a powerful antioxidant.
- Zn is important in the immune response.
- It has other vital functions including structural properties in some proteins. Zn stabilizes the structure of DNA, RNA, and ribosomes; it has a vital role in gene expression.

Measurement

<0.1% of the body's Zn is present in the blood and its measurement in plasma is not a good measure of Zn status. The measurement of thymulin activity is increasingly being used although it is labour-intensive and ∴ not widely available. Thymulin promotes T-lymphocyte maturation and requires Zn for it to be active. Zn supplementation and observation of the subject's response is the most reliable method of diagnosing deficiency.

Deficiency

- Severe deficiency results in growth retardation, failure to thrive, delayed sexual maturation.
- Sore throat and immune defects.
- Circumoral and acral dermatitis.
- Diarrhoea: Zn supplementation has been implemented in areas of the world where children are affected by persistent diarrhoea.
- Alopecia and neuropsychiatric symptoms.

Requirement and intake

See Tables 6.34 and 6.35.

Sources in the diet

See Table 6.36. Zn bioavailability is higher from animal sources than from cereals which contain phytate. Bioavailability is estimated to be 50–55% for an omnivorous diet in the UK; vegetarian and vegan diets have an estimated bioavailability of 30–35%.

Toxicity

Zn toxicity can occur following ingestion of water that has been stored in galvanized tanks or if this water is used for renal dialysis. Acute ingestion of 2 g or more of Zn results in nausea, vomiting, and fever. Intakes of 50 mg of Zn have been shown to interfere with Cu and Fe metabolism. Chronic intakes of 75–300 mg/day have been associated with symptoms of Cu deficiency including microcytic anaemia and neutropenia.

Table 6.34 Reference nutrient Intakes for Zn (mg/day) for all ages and average daily intakes (mg) for adult men and women provided by food (NDNS)*

Age†	RNI
0–6 months	4.0
7 months–3 years	5.0
4–6 years	6.5
7–10 years	7.0
11–14 years	9.0
Males	
15+ years	9.5
Females	
15+ years	7.0
Lactation	
0–4 months	+6.0
4+ months	+2.5
Average daily intake UK	
Men	10.1
Women	7.8

*Source for RNIs, Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom.* HMSO, London. Source for average daily intakes for adults, Bates, B., Lennox, A., and Swan, G. (2010). *The National Diet and Nutrition Survey: Headline results from Year 1 of the Rolling Programme (2008/2009)*. Food Standards Agency, London.

†No increase is recommended in pregnancy.

Table 6.35 Contribution of foods to Zn intake (NDNS)*

Food group	% Daily intake
Meat and meat products	34
Beef, veal, and dishes	11
Turkey, chicken, and dishes	5
Cereals and cereal products	25.5
White bread	6
Breakfast cereals	5
Milk and milk products	17
Cheese	6
Semi-skimmed milk	6

*Henderson, L., Irving, K., and Gregory, J. (2003). *The National Diet and Nutrition Survey: adults aged 19 to 64 years.* Vol. 3. *Vitamin and mineral intake and urinary analytes.* HMSO, London.

Table 6.36 Food sources of Zn

Source	Food
Very rich	Lamb, leafy and root vegetables, crabs and shellfish, beef, offal
Rich	Whole grains, pork, poultry, milk and milk products, eggs, nuts

Copper (Cu)

An adult has 80 mg of Cu in their body, 40% of which is present in muscle, 15% in the liver, 10% in the brain, and 6% in blood.

Function

Cu is incorporated in many metallo-enzymes, which are shown in Table 6.37.

Measurement

A totally reliable, sensitive method of assessing Cu status has yet to be established. Plasma Cu and caeruloplasmin (Cu-containing protein that normally binds 90% of the Cu present in plasma) are frequently used; they are both lowered in deficiency, but they plateau as levels of Cu ↑ and do not reflect high intakes. Neither is very specific. Normal serum Cu levels are 12–26 μg/l, but they are ↑ in late pregnancy and in women taking oestrogen-based contraceptives. Other methods include assessment of the activity of Cu enzymes in particular superoxide dismutase.

Deficiency

Cu deficiency is rare although it can occur in premature infants and in patients receiving total parenteral nutrition. Cu is accumulated in the fetus during the late stages of pregnancy and full-term babies have large stores in the liver. ↑ Cu losses can occur in diseases such as cystic fibrosis, coeliac disease, and in children with chronic diarrhoea. Cu deficiency occurs in the hereditary condition Menkes disease in which Cu transport is impaired.

The symptoms of Cu deficiency are:

- Failure to thrive in babies.
- Oedema with low serum albumin.
- Fe resistant anaemia.
- Impaired immunity with low neutrophil count.
- Skeletal changes including fractures and osteoporosis.
- Abnormal blood vessels due to defects in collagen and elastin.
- Hair and skin hypopigmentation with steely, uncrimped (kinky) hair.
- Neurological abnormalities.

Cu deficiency may be a risk factor for coronary heart disease as it has been associated with raised plasma cholesterol levels and heart-related abnormalities.

Requirements and intake

See Tables 6.38 and 6.39. No increment for pregnancy is recommended as any ↑ in demand is met by the mother's adaptive responses. The average intake is ↑ to 1.48 mg/day in men and 1.07 mg/day in women by the use of supplements.

Sources in the diet

See Box 6.17. Bioavailability ranges from 35 to 70% and ↓ with age. The bioavailability of Cu in milk based formulae is approximately 50%.

Toxicity

Cu toxicity occurs either by the deliberate ingestion of Cu salts or by drinking contaminated water. The symptoms of acute toxicity are nausea, vomiting, and diarrhoea and may be fatal in extreme cases. Chronic Cu poisoning, due to contamination by Cu water pipes or cooking utensils can → liver cirrhosis; infants and young children are particularly vulnerable. Wilson's disease is an inherited disease in which there is abnormal Cu transport that results in Cu accumulation in the liver, eyes brain, and kidneys and associated pathology.

Table 6.37 Functions of Cu metallo-enzymes

Enzyme	Functions
Blue proteins	Electron transfers
Cytochrome-c oxidase	Electron transport: reduction of O_2 to H_2O
Caeruloplasmin (ferroxidase I)	Fe oxidation and transport
Superoxidase dismutase	Antioxidant
Dopamine-hydroxylase	Hydroxylation of dopa in the brain
Diamine and monamine oxidase	Removal of amines and diamines
Lysyl oxidase	Cross-inking in collagen and elastin, cardiovascular and bone integrity
Tyrosinase	Melanin formation
Chaperone proteins	Intracellular Cu transport
Chromatin scaffold proteins	Structural integrity of nuclear material
Clotting factors V, VIII	Thrombogenesis
Metallothionein	Metal sequestration
Nitrous oxide reductase	Reduction of NO_2^- to NO

Taken from Reilly, C. (2004), *The nutritional trace metals*, table 41, p. 120. Reproduced with permission from Blackwell Publishing.

Table 6.38 Reference nutrient intakes for Cu (mg/day) for all ages and average daily intakes (mg) for adult men and women provided by food*

Age†	RNI
0–12 months	0.3
1–3 years	0.4
4–6 years	0.6
7–10 years	0.7
11–14 years	0.8
15–16 years	1.0
18+ years	1.2
Pregnancy	+ 0.3
Average daily intake UK	
Men	1.29
Women	1.08

*Source for RNIs, Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. HMSO, London. Source for average daily intakes for adults, Bates, B., Lennox, A., and Swan, G. (2010). *The National Diet and Nutrition Survey: Headline results from Year 1 of the Rolling Programme (2008/2009)*. Food Standards Agency, London.

Table 6.39 Contribution of foods to Cu intake (NDNS)*

Food group	% Daily intake
Cereals and cereal products	31
White bread	8
Meat and meat products	15
Potatoes and savoury snacks	10
Fruits	10

*Henderson, L., Irving, K., and Gregory, J. (2003). *The National Diet and Nutrition Survey: adults aged 19 to 64 years*. Vol. 3. *Vitamin and mineral intake and urinary analytes*. HMSO, London.

Box 6.17 Good food sources of Cu

- Offal
- Nuts
- Cereals and cereal products
- Meat and meat products

Iodine (I)

Function

- Iodine is a component of the thyroid hormones thyroxine (T4) and tri-iodothyronine (T3).
- Thyroid hormones maintain the body's metabolic rate by controlling energy production and oxygen consumption in cells.
- They are required for normal growth and development.
- In the foetus and neonate normal protein metabolism in the brain and central nervous system (CNS) requires iodine.

Measurement

Levels of thyroid-stimulating hormone (TSH) are the most sensitive indicators of iodine status. It is raised in iodine deficiency. In severe deficiency serum T3 and T4 decline.

Deficiency

- Iodine deficiency disorder (IDD) in adults results in hypothyroidism and raised levels of TSH, which cause hyperplasia of thyroid tissues resulting in goiter Hypothyroidism is characterized by lethargy, poor cold tolerance, bradycardia, and myxoedema.
- In the foetus IDD results in cretinism. This is characterized by mental retardation, hearing, speech defects, squint, disorders of stance and gait, and growth retardation. The degree of cretinism is variable and varying degrees of growth retardation and mental retardation are seen in infants and children with IDD.
- IDD is also linked to ↑ in the rates of still birth, miscarriage, and infertility.

IDD is now rare in the UK, although some areas were once associated with IDD, e.g. Derbyshire. It is estimated that 200–300 million people world-wide demonstrate some degree of IDD. Research is currently being conducted to look at the sufficiency of I intake during pregnancy.

Intake

See Tables 6.40 and 6.41, and Box 6.18. The amount of iodine in the diet is affected by the geography of the areas of cultivation. Areas with poor soil content, e.g. mountainous areas such as the Himalayas, are often associated with endemic IDD. This is due to iodine being washed from the soil. Supplementation of salt has been introduced in an attempt to reduce IDD. In the UK the iodine content of foods has gradually ↑ due to the supplements of cattle feeds, which are secreted in milk.

Absorption is reduced by the presence of goitrogens in some foods, e.g. brassica vegetables (cabbage, swede, Brussels sprouts, broccoli), cassava, maize, lima beans. Goitrogens are inactivated by heating.

Toxicity

High intakes can cause hyperthyroidism and toxic modular goitre; there is a weak relationship between persistently high intakes and thyroid cancer. The safe upper limit is 17 μg/kg/day.

Table 6.40 Reference nutrient intakes for iodine (μg/day) for all ages and average daily intakes (μg) for adult men and women provided by food*

Age	RNI
0–3 months	50
4–12 months	60
1–3 years	70
4–6 years	100
7–10 years	110
11–14 years	130
15+ years	140
Average daily intake UK	
Men	202
Women	141

*Source for RNIs, Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. HMSO, London. Source for average daily intakes for adults, Bates, B., Lennox, A., and Swan, G. (2010). *The National Diet and Nutrition Survey: Headline results from Year 1 of the Rolling Programme (2008/2009)*. Food Standards Agency, London.

Table 6.41 Contribution of foods to iodine intake (NDNS)*

Food group	% Daily intake	
	Men	Women
Milk and milk products	35	42
Skimmed milk	18	18
Drinks	19	9
Beer and lager	15	3
Cereals and cereal products	12	12
Fish and fish products	11	11

*Henderson, L., Irving, K., and Gregory, J. (2003). *The National Diet and Nutrition Survey: adults aged 19 to 64 years*. Vol. 3. *Vitamin and mineral intake and urinary analytes*. HMSO, London.

Box 6.18 Good food sources of iodine

- Milk and dairy products
- Sea fish, e.g. haddock, cod
- Seaweed
- Iodized salt

Selenium

Function

Selenium (Se) is an integral part of over 30 selenoproteins; the most important of which are:

- Glutathione peroxidases, which protect against oxidative damage.
- Iodothyronine deiodinases, which are involved in the production of tri-iodothyronine from thyroxine.
- Selenoprotein P, which is involved in antioxidant and transport functions.

Measurement

Se levels are measured in whole blood. There is considerable geographical variation in concentration: the range for the UK is 0.091–0.120 μg/ml.

Deficiency

Deficiency of Se is associated with two endemic causes: Keshan disease and Kashin–Beck disease.

- *Keshan disease:* outbreaks in Russia and several parts of Asia; it is characterized by a cardiomyopathy.
- *Kashin–Beck disease:* an endemic musculoskeletal disorder that has occurred in parts of Siberia and Asia.
- *Iatrogenic causes of Se deficiency:* include patients receiving total parenteral nutrition (TPN), phenylketonuric patients receiving a semi-synthetic diet. Patients exhibit symptoms of cardiomyopathy and/or musculoskeletal disorders.

Requirement and intake

See Tables 6.42 and 6.43. Se intake has ↓ over the last 20 years, due to ↑ consumption of European wheat that is low is Se, and the average intake is below the recommended intake. Epidemiological evidence suggests that this may contribute to ↓ risk of infection, cardiovascular disease and the incidence of some cancers. Low Se levels have been reported in AIDS/HIV patients and ↓ SE levels appear to be correlated to ↑ mortality. Interventional studies are being conducted to establish the functional consequences of small supplements. Due to the potential risk of toxicity self-administration of large supplements is not recommended.

Sources in the diet

See Box 6.19. Lacto-ova vegetarians and vegans may be at risk of Se deficiency.

Toxicity

Acute Se poisoning is characterized by hypersalivation, nausea, vomiting, and garlic-smelling breath. This may be accompanied by diarrhoea, hair loss, restlessness, tachycardia, and fatigue. Chronic poisoning (selenosis) is associated with nail and hair changes, skin lesions, and neurological, effects; numbness, pain, and paralysis may follow. Early nail changes have been observed at intakes of 900 μg/day and the recommended maximum safe intake is 6 μg/kg/day.

Table 6.42 Reference nutrient intakes for Se (μg/day) for all ages and average daily intakes (mg) for adult men and women provided by food*

Age	RNI
0–3 months	10
4–6 months	13
7–12 months	10
1–3 years	15
4–6 years	20
7–10 years	30
11–14 years	45
Men	
15–18 years	70
19+ years	75
Women	
15+ years	60
Lactation	+15
Average daily intake UK	
Men	55 mg
Women	43 mg

*Source for RNIs, Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. HMSO, London; source for average daily intakes for adults, Bates, B., Lennox, A., and Swan, G. (2010). *The National Diet and Nutrition Survey: Headline results from Year 1 of the Rolling Programme (2008/2009)*. Food Standards Agency, London.

Table 6.43 Contribution of foods to Se intake (MAFF 1995)*

Food	% Daily intake
Meat and meat products	15
Bread	15
Fish	12
Milk and milk products	10

*MAFF (1995). *MAFF UK—analysis of foods for selenium*, MAFF UK Food Surveillance Information Sheet no. 51, February, 1995. MAFF, London.

Box 6.19 Food sources of Se

- Offal
- Fish
- Brazil nuts
- Eggs
- Poultry
- Meat and meat products

Magnesium (Mg)

Function

- Mg is an integral part of bones and teeth; 60% is found in the skeleton.
- Intracellular energy metabolism; a co-factor for enzymes requiring ATP, in the replication of DNA, and synthesis of protein and RNA.
- Essential for phosphate transferring systems.
- Muscle and nerve cell function.

Measurement

Serum Mg is the most frequently used index of status. The normal range is 0.7–1.0 mmol/l.

Deficiency

Mg is found in all animal and plant foods and its concentration in the blood is tightly controlled; a dietary deficiency is unlikely to occur. Low serum Mg levels occur when there are ↑ renal losses, malabsorption, or changes in tissue distribution due to disease or use of some drugs, e.g. diuretics, re-feeding syndrome. Hypomagnesaemia has been associated with cardiac arrhythmias and cardiac arrest. Very low levels of Mg are associated with hypocalcaemia.

Requirement and intake

See Tables 6.44 and 6.45

Sources in the diet

See Box 6.20. Hard drinking water may make a significant contribution to Mg intake.

Toxicity

If renal function is normal, hypermagnesaemia is virtually impossible to achieve by dietary means; it can occur in renal or adrenal disease. Large quantities of some Mg salts (Epsom salts) are used for their cathartic effect.

Box 6.20 Good food sources of Mg

- Green vegetables
- Pulses and whole grain cereals
- Meats

Table 6.44 Reference nutrient intakes for Mg (mg/day) for all ages and average daily intakes (mg) for adult men and women provided by food*

Age†	RNI
0–3 months	55
4–6 months	60
7–9 months	75
10–12 months	80
1–3 years	85
4–6 years	120
7–10 years	200
11–14 years	280
15–18 years	300
Men	
19+ years	300
Women	
19+ years	270
Lactation	+50
Average daily intake UK	
Men	302
Women	229

*Source for RNIs, Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. HMSO, London. Source for average daily intakes for adults, Bates, B., Lennox, A., and Swan, G. (2010). *The National Diet and Nutrition Survey: Headline results from Year 1 of the Rolling Programme (2008/2009)*. Food Standards Agency, London.

Table 6.45 Contribution of foods to Mg intake (NDNS)*

Food	% Daily intake
Cereals and cereal products	27
Breakfast cereals	7
Sugar, preserves, and confectionery	17
Meat and meat products	12
Milk and milk products	11
Potatoes and savoury snacks	10

*Henderson, L., Irving, K., and Gregory, J. (2003). *The National Diet and Nutrition Survey: adults aged 19 to 64 years*. Vol. 3. *Vitamin and mineral intake and urinary analytes*. HMSO, London.

Manganese (Mn)

Function

- Mn is a component of several metallo-enzymes including arginase, pyruvate.
- It is needed for enzyme activity, including glutamine synthetase and various hydrolases, kinases, decarboxylases, and phosphotransferases.

Measurement

There is no accepted measurement of Mn status. Enzyme activity assays have been proposed, but none have been accepted into widespread practice.

Deficiency

Deficiency has only been observed in experimental studies: fingernail growth is slowed, black hair reddened, and a scaly dermatitis developed.

Requirement and intake

See Tables 6.46 and 6.47, and Box 6.21. No cases of nutritional Mn deficiency have been observed; ∴ no recommended nutrient intakes have been made in the UK. Safe levels are shown in Table 6.43.

Toxicity

Mn toxicity is low as absorption is ↓ when intake is high and any that is absorbed is excreted in bile and urine.

Table 6.46 Safe intakes for Mn for all ages and average daily intakes (μg) for adult men and women provided by food*

Age	RNI
Infants and children	>16 μg/kg/day
Adults	>1.4 mg/day
Average daily intake UK (mg/day)	
Men	3.32
Women	2.69

*Source for RNIs, Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. HMSO, London. Source for average daily intakes for adults, Henderson, L., Irving, K., and Gregory, J. (2003). *The National Diet and Nutrition Survey: adults aged 19 to 64 years*. Vol. 3. *Vitamin and mineral intake and urinary analytes*. HMSO, London.

Table 6.47 Contribution of foods to Mn intake (NDNS)*

Food	% Daily intake
Cereals and cereal products	50
Bread	26
Breakfast cereals	11
Biscuits, cakes, etc.	5
Drinks	17
Tea	12
Vegetables excluding potatoes	10

*Henderson, L., Irving, K., and Gregory, J. (2003). *The National Diet and Nutrition Survey: adult aged 19 to 64 years*. Vol. 3. *Vitamin and mineral intake and urinary analytes*. HMSO, London.

Box 6.21 Good food sources of Mn

- Cereals and cereal products
- Tea
- Vegetables

Molybdenum

Function

Co-factor in xanthine oxidase, sulphite oxidase, and aldehyde oxidase, and is ∴ involved in the metabolism of purines, pyrimidines, quinolines, and sulphites.

Measurement

Mo can be measured in whole blood and serum. Concentrations in whole blood vary widely although the mean con- centration is 0.5 μg/l.

Deficiency

Dietary deficiency of Mo has been reported in farm animals, but has not been observed in humans, although there is a single case reported following prolonged TPN. The symptoms included defects in sulphur metabolism, mental disturbance, and coma. An inborn error of metabolism results in abnormal production of the coenzyme. It is characterized by abnormal urinary metabolites, neurological and ocular problems, and failure to thrive. The genetic expression and symptoms are varied and in the most severe cases can be fatal at 2–3 years.

Requirement and intake

There are no RNIs, but safe intakes are believed to be between 50 and 400 μg/day in adults and 0.5 and 1.5 μg/day in children. Mean intakes in adults are reported as 0.12 mg/day.

Sources in the diet

Offal, nuts, cereals, and bread are good sources.

Toxicity

Little data are available for dietary excess, although intakes >100 mg/kg/day have been reported to cause diarrhoea, anaemia, and high blood uric acid levels; this is associated with gout.

Chromium

The essentiality of Cr is widely accepted, although this is still challenged by some scientists. In nutrition the trivalent state appears to have physiological functions but it is interchangeable with hexavalent Cr.

Function

- Cr is believed to be part of an organic complex known as the 'glucose tolerance factor' (GTF), which potentiates the action of insulin. The evidence for essentiality of Cr comes from observations of patients receiving TPN who develop diabetic symptoms. The symptoms respond to Cr treatment, but not insulin. Studies on the use of Cr in the management of type 2 diabetes are not conclusive.
- Cr may participate in lipoprotein metabolism.

Measurement There is no totally reliable measure of Cr. Urinary excretion, expressed in terms of creatinine, has been suggested as a measure of chromium status. Hair Cr levels have been used as a measure of long-term exposure although hair analysis is associated with several problems.

Deficiency Deficiency in humans has only been observed in patients receiving long-term TPN. The symptoms included impaired glucose tolerance, weight loss, neuropathy, elevated plasma fatty acids, depressed respiratory quotient, and abnormal nitrogen metabolism.

Requirement and intake

There are no RNIs for Cr but the theoretical requirement extrapolated from balance studies is 25–30 μg/day in adults. In children the safe intake is believed to be 0.1–1.0 μg/kg/day. The average daily intake of Cr for adults is estimated as 0.1 mg.[1]

Sources in the diet The richest sources of Cr in the diet are meat, whole grains, legumes, and nuts.

Toxicity

The trivalent form is not associated with toxicity, but the hexavalent form is very toxic. Two fatalities have been reported following acute ingestion of very large doses of hexavalent Cr as dichromate (75 mg/kg) and chromic acid (4.1 mg/kg). Symptoms included gastrointestinal haemorrhages, renal and liver abnormalities. Chronic toxicity is associated with renal failure, liver failure, haemolysis, and anaemia.

[1] Ysart, G., Miller, P., *et al.* (2000). 1997 UK Total Diet Study—dietary exposures to aluminum, arsenic, cadmium, chromium, copper, lead, mercury, nickel, selenium, tin and zinc. *Food Addit. Contam.* **17**, 775–86.

Fluorine

Fluorine (Fl) is considered semi-essential as it has biological functions, but its essentiality is still being debated.

Function Fluoride has a role in bone mineralization and protects against dental caries.

Deficiency Low intakes are associated with ↑ incidence of dental caries.

Requirement and intake See Tables 6.48 and 6.49, and Box 6.22. There are no RNIs for Fl, although safe intakes are given in Table 6.45. Total intakes depends on the level of fluoridation in water consumed. 10% of water in the UK is fluoridated or has a natural content above the recommended fluoridation rate of 1 ppm. A recent report (2006) by the FSA reports findings from the 2004 Total Diet Study.[1]

Toxicity Intake 3–5 times the normal intake is mildly toxic. Tooth mottling occurs in mild toxicity and chronic excess (10 mg/day) causes joint and bone abnormalities.

Table 6.48 Safe intakes of Fl (mg/kg/day)

Age	Safe intake
0–6 months	0.22
6–12 months	0.12
>1 year	0.05

Table 6.49 Mean adult intakes of Fl (mg/day)

	Intake
Non-fluoridated water areas	1.82
Fluoridated water areas*	2.90

*Assumes an average daily intake of 1.1 l daily.

[1] Ysart, G., Miller, P., *et al.* (2000). 1997 UK Total Diet Study—dietary exposures to aluminum, arsenic, cadmium, chromium, copper, lead, mercury, nickel, selenium, tin and zinc. *Food Addit. Contam.* **17**, 775–86.

Box 6.22 Sources of 1 mg Fl

- 1 l water (fluoridated at 1 ppm)
- 1 Fl tablet
- 1 g Fl toothpaste (accidental consumption)
- 5 ml Fl mouthwash (accidental consumption)
- 2–3 cups of tea (depends on strength)
- 1400 g cooked spinach
- 250 g tinned sardines

Food Standards Agency (2006). *Fluorinated chemicals: UK dietary intakes.* Food Survey Information Sheet No. 11/06. Available at:
℞ www.food.gov.uk/multimedia/pdfs/fsis1106.pdf

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Chapter 7

Electrolytes and fluid balance

Electrolytes: introduction

The monovalent electrolytes are Sodium (Na), Chlorine (Cl), and Potassium (K).

Sodium

An adult male (70 kg) has total body Na of 4 mol (92 g); 2000 mmol is in extracellular fluid (ECF), 1500 mmol in bone, and 500 mmol in intracellular fluid.

Function

- Cation in extracellular fluid.
- Regulation of blood pressure and transmembrane gradients.
- Acid–base regulation.
- Electrophysiological control of muscles and nerves.

Measurement

Na is easily measurable in plasma, with a normal range of 135–150 mmol/l.

Deficiency

Na losses requiring repletion can result from excess sweating in extreme conditions of heat and exertion.

Requirement and intake

See Table 7.1.

Sources in the diet

Na is present in many food additives, e.g. monosodium glutamate, but most Na in the diet is present as salt (NaCl). Levels are comparatively low in unprocessed foods. Salt is added to food as a preservative and flavour enhancer; it can also be used as a fermentation control agent in bread making, texturizer, binder, and colour developer. In the UK cereals and meat and their respective products provided 30 % & 28 % of sodium in the diet. 3 g salt ≈ 1.2 g Na.

Toxicity

It is a strong emetic, but excessive oral loads of Na are potentially fatal. Artificial intravenous load has severe and rapid effects.

Health implications of excess consumption

Excess Na intake has been linked to hypertension and heart disease. SACN has recommended that salt intake targets should be:

- 0–6 months, <1 g/day;
- 7–12 months, 1 g/day;
- 1–6 years, 2 g/day;
- 7–14 years, 5 g/day;
- >15 years, 6 g/day.

NB. Na can roughly be converted to NaCl by multiplying by 2.5.

FSA is working with food manufacturers to reduce the Na content of processed foods. Table 7.2 shows foods high in salt. The FSA has published voluntary salt reduction targets to encourage food manufacturers and retailers to reduce the amount of salt in a wide range of processed foods. Details can be found on the FSA website (www.food.gov.uk) and at Consensus Action on Salt and Health (www.actiononsalt.org.uk).

Clinical restriction of Na

Some disease states, e.g. renal disease, require the restriction of Na. The level of restriction can be classified.

- ***No added salt***: 80–100 mmol Na/day.
- ***Low salt***: 40 mmol Na/day.
- ***Low Na***: 22 mmol Na/day.

Table 7.1 Reference nutrient intakes for Na mg/d (mmol/day) for all ages and average daily intakes of Na(mg/d) and NaCl (g/day) for adult men and women provided by food (NDNS)*

Age	RNI	
0–3 months	210 (9)	
4–6 months	280 (12)	
7–9 months	320 (14)	
10–12 months	350 (15)	
1–3 years	500 (22)	
4–6 years	700 (30)	
7–10 years	1200 (50)	
11–14 years	1600 (70)	
15–50+ years	1600 (70)	
Average daily intakes UK		
	Na	**NaCl**
Men	2800	7.0
Women	2038	5.9

*Source for RNIs, Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. HMSO, London. Source for average daily intakes for adults, Bates, B., Lennox, A., and Swan, G. (2010). *The National Diet and Nutrition Survey: Headline results from Year 1 of the Rolling Programme (2008/2009)*. Food Standards Agency, London.

Table 7.2 Foods high in salt*

Foods where some brands/recipes are high in salt	
Bread products e.g. crumpets, bagels	Crisps
Pasta sauces	Pizza
Ready meals	Soup
Sandwiches	Sausages
Tomato ketchup, mayonnaise and other sauces	
Foods that are usually high in salt	
Anchovies	Bacon
Cheese	Chips (if salt added)
Gravy granules	Ham
Olives	Pickles
Pretzels Salted and dry roasted nuts	Salami
	Salt fish
Sausages	Smoked meat and fish
Soy sauce	Stock cubes
Yeast extract	

* Based on information from the Food Standards Agency, available at: www.eatwell.gov.uk.

Potassium

Function

Intracellular cation that is involved in acid–base regulation, electrophysiology of nerves and muscles, and is essential for the cellular uptake of molecules against concentration and electrochemical gradients.

Measurement

Normal plasma concentration is 3.5–5.0 mmol/l. Over 95% of total body K is found in cells; an adult male contains 40–50 mmol/kg (1.6–2.0 g/kg).

Deficiency

Lack of K alters the electrophysiology of cell membranes and causes muscle weakness. In cardiac muscle this leads to arrhythmias and cardiac arrest. Motility is lost in the intestine and mental depression and confusion can develop. Dietary deficiency of K is very unlikely as it is found in all foods. Causes of K depletion are shown in Box 7.1.

Requirement and intake

See Tables 7.3 and 7.4, and Box 7.2.

Toxicity

Toxicity due to dietary excess is unlikely. Acute intakes of supplements exceeding 17.6 g (450 mmol) may cause symptoms of hyperkalaemia. Hyperkalaemia causes paraesthesiae around the mouth and muscle weakness although these symptoms may be absent. There is a risk of cardiac arrest.

Box 7.1 Causes of K depletion

Gastrointestinal causes

- Diarrhoea
- Vomiting
- Small bowel or gastric drainage
- Ureterocolic anastomosis
- Purgatives

Urinary losses

- Chronic acidosis or alkalosis
- Osmotic diuresis, e.g. uncontrolled diabetes
- Renal disease (tubular)
- Diuretic drugs
- Steroid excess (Cushing's disease, primary and secondary hyperaldosteroidism)

Table 7.3 Reference nutrient intakes for K mg/day (mmol/day) for all ages and average daily intakes (mg) for adult men and women provided by food*

Age	RNI
0–3 months	800 (20)
4–6 months	850 (22)
7–12 months	700 (18)
1–3 years	800 (20)
4–6 years	1100 (28)
7–10 years	2000 (50)
11–14 years	3100 (80)
15–50+ years	3500 (90)
Average daily intakes UK	
Men	3251
Women	2593

*Source for RNIs, Department of Health (1991). *Dietary reference value for food and nutrients for the United Kingdom*. HMSO, London; source for average daily intakes for adults, Bates, B., Lennox, A., and Swan, G. (2010). The National Diet and Nutrition Survey: Headline results from Year 1 of the Rolling Programme (2008/2009). Food Standards Agency, London.

Table 7.4 Contribution of foods to K intake (NDNS)*

Food group	% Daily intake
Potatoes and savoury snacks	18
Meat and meat products	15
Drinks	15
Cereals and cereal products	13
Milk and milk products	13
Vegetables (excluding potatoes)	10

*Henderson, L., Irving, K., and Gregory, J. (2003). *The National Diet and Nutrition Survey: adults aged 19 to 64 years*. Vol. 3. *Vitamin and mineral intake and urinary analytes*. HMSO, London.

Box 7.2 Rich food sources of K

- Fruit especially bananas, apricots, blackcurrant, rhubarb, fruit juices
- Vegetables especially potatoes and potato snacks
- Chocolate, cocoa, and chocolate products
- Coffee and coffee products
- Malted milk drinks
- Yeast extracts and spreads, tomato ketchup, stock cubes, bottled sauces
- Table salt substitutes

Chlorine

Total body Cl is ~33 mmol (1.2 g)/kg. 70% is in extracellular fluid (ECF).

Function

Cl is the anion to the cations Na and K.

Measurement

Normal plasma concentration is 97–107 mmol/l.

Requirement and intake

There are no specific DRVs for Cl; it is recommended that Cl intake should equal Na intake in molar terms. The average intakes of Cl in foods are 4995 mg/day for men and 3481 mg/day for women.

Sources in the diet

Cl is usually consumed with Na as salt (NaCl).

Fluid balance

The human body is mainly water; a 70 kg man is comprised of approximately 45 l water; ECF 15 l, intracellular fluid (ICF) 30 l. 72% FFM is water. ECF is comprised of plasma and interstitial fluids. The monovalent electrolytes, Na, Cl, and K, determine the body's osmolality and their distribution determines the volume of ECF and ICF. ICF, plasma, and interstitial fluids are separated by semi-permeable membranes and are interdependent. Movement of fluid between the compartments is controlled by plasma osmolality and hydrostatic pressure gradients.

Regulation of fluid balance

Fluid balance is under tight homeostatic control and fluctuates by <1% per day despite large variations in fluid intake. Normally the osmolalities of plasma and interstitial fluids are similar. Plasma osmolality reflects serum Na which reflects total ECF.

Plasma osmolality

↑ In plasma osmolality → thirst and the hypothalamus will be stimulated to ↑ antidiuretic hormone (ADH). This leads to reabsorption by the distal tubules of the kidney which corrects the osmolality. ↓ plasma volume can also → raised osmolality which causes aldosterone to be released. This ↑ Na and water retention.

Hydrostatic pressure

Plasma volume is also controlled by hydrostatic pressure. At the arterial end of the capillaries blood pressure is exerted, but less osmotic pressure is exerted by plasma proteins resulting in the movement of fluid out into the interstitial fluid. At the venous end of the capillary the process is reversed and fluid passes back into plasma.

Water balance

See Table 7.5.

Thirst

Thirst usually plays only a small role in fluid balance of normal subjects. Fluid is usually consumed for reasons other than thirst, e.g. habit and customs. Local drying of the mouth and throat will also cause thirst, e.g. public speakers often require water to lubricate the mouth and throat.

Fluid losses

Fluid output in urine is controlled by the kidneys but there are also insensible water losses through the skin and lungs and in faeces.

- Sweat glands secrete water in sweat, which evaporates from the skin. This evaporation cools the skin. Water is also lost directly through the skin. 500–750 ml/day of fluid is lost through the skin and ↑ in fever and extreme temperatures or exertion. ↑ in 1°C in body temperature will ↑ fluid requirements by 500 ml/day.
- Healthy adults pass 50–400 g/day of faeces and approx. 75% of this will be fluid.
- Expired air contains 44 mg water/l and this will be ↑ in fever, with ↑ respiration rates, and by reduced water content of inspired air, e.g. at high altitudes.

Fluid requirements

The amount of fluid consumed is very variable. In normal conditions 30–35 ml/kg body weight is required daily. In some disease states, i.e. cardiac, hepatic, and renal disease, it may be necessary to restrict fluid intake to prevent fluid overload (see 📖 Chapter 23, 'Cardiovascular disease', p. 465, 📖 Chapter 28, 'Liver disease', p. 619, and 📖 Chapter 29, 'Renal disease', p. 631).

Table 7.5 People at risk of dehydration*

Reason	Cause
Increased fluid losses	Patients with tracheotomies or on ventilators
	Diarrhoea and/or vomiting
	Stomal losses
	Wound or burn exudates
	Pyrexia
	Diabetes insipidus
	Diabetic ketoacidosis
	Prolonged use of diuretics
	Patients receiving high protein high osmolar diets
Lack of awareness or inability to express the need for fluid	Patients who are unable to communicate, e.g. due to stroke
Low fluid intake	Poor food intake, e.g. anorexia, depression.
	Apathy, chronic illness, physical immobility, etc.
	Eating difficulties
	Swallowing difficulties
	Deliberate fluid restriction to avoid incontinence
	'Nil by mouth' regimens

*Reproduced from B Thomas & J Bishop (2007). *Manual of Dietetic Practice*, table 2.8.4, p. 220. Permission requested from Blackwell Publishing.

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Chapter 8

Food labelling, functional foods, nutrigenetics, and nutrigenomics and food supplements

Food labelling

Food labelling in the UK is currently controlled by the Food Labelling Regulations of 1996, subsequent amendments to these regulations, and also by European laws. Legally these regulations fall under the Food Safety Act of 1990. In the UK foods sold loose are exempt from many labelling regulations.

A new Food Information Regulation is being drafted, which will bring European rules on general and nutrition labelling together under a single regulation and will eventually replace current UK law.

Information required by law

Product name

This must be clearly stated and products with 'made up' names must give a description of the food. If the food has undergone processing, e.g. 'smoked', the process must be stated. The name must also distinguish between similar products. For example, 'orange drink' must contain oranges while 'orange flavoured drinks' can be made with artificial flavourings.

Ingredient list and quantity

Ingredients are listed in descending order of weight. All ingredients including additives and water must be listed, and the ingredients that make up a compound ingredient e.g. pepperoni on a pizza. The net quantity of a food ingredient must also be listed alongside the ingredient unless it is <5 g.

Quantitative ingredient declaration

An ingredient that is featured in a photograph or drawing on a pack or in the description of the product, e.g. potatoes in cheese and potato pie, must state the quantity of the ingredient declared as a percentage. This is required by European Union labelling law and is known as quantitative ingredient declaration (QUID).

Allergenic ingredients

Food and drink labels must state clearly if they contain ingredients to which people may be allergic or intolerant. The list of allergens that must be labelled is set down in European Union law (see Box 8.1). The manufacturer must also make it clear if ingredients are made-of or derived from the allergens, e.g. it is not enough to state 'glaze'; the label must state 'glaze made from eggs'. The exception arises for ingredients derived from allergens that have been processed and are no longer allergenic; these do not need to be labelled with reference to the parent allergen. For example glucose syrup made from wheat can be labelled just as 'glucose syrup'. Only ingredients that have been assessed and deemed safe by the European Food Safety Authority (EFSA) are exempt.

Shelf life

Labels must give information on how long the product will last once it has been bought or opened. This information is intended to ensure the safety and quality of the food and to prevent food poisoning or food-borne illnesses.

The 'use-by' date label must be present on perishable foods such as cooked meats, which deteriorate and can be dangerous to health after a relatively short period. 'Best before' must be expressed as a day, month, and year and is used to indicate that a food's flavour, colour, or texture may not be at its best beyond this period although it is probably still safe to eat. For products with a shelf life longer than 3 months 'Best before end' is used. 'Display until' is not required by law but is used by retailers to alert staff to the need to remove products from sale. Wine and spirits do not have to be date marked.

Box 8.1 Allergenic ingredient that must be listed

- Celery
- Cereals containing gluten—wheat, rye, oats, barley
- Crustaceans, e.g. lobster, crab
- Eggs
- Fish
- Lupin
- Milk
- Molluscs
- Mustard
- Nuts—almonds, pistachios, brazil nuts, walnuts, hazelnuts, cashews,
- Peanuts
- pecans, macadamia nuts
- Sesame seeds
- Soybeans
- Sulphur dioxide and sulphites at >10 mg/kg or l

Storage instructions

Details must be given on the conditions needed to ensure freshness. Following the instructions should ensure that the product's appearance and taste are optimum and prevent spoiling too quickly, so minimizing the risk of food poisoning.

Name and address of manufacturer, packer, or seller

These details must be stated on the package so that the consumers have a point of contact if they want to make a complaint or need more information.

Country of origin

The label must clearly state where the food is from if it would be misleading not to show it, e.g. French onion soup made in Scotland.

Weight or volume

The volume or weight of the product must be shown on the label. This enables consumers to compare the value of different brands. The symbol 'e' shows that the weight complies with EU requirements in that the average pack is at least the declared weight. Some foods, e.g. butter, tea, are sold only in standard amounts. Products that weigh <5 g do not have to have a stated weight, except for herbs and spices.

Instructions for use

Instruction on how to prepare and cook the product must be printed on the packaging when necessary. Oven temperature and cooking time are stated if the product needs heating; instructions on microwave cooking may also be given. The instructions are given so that the food can be consumed at its best and to reduce the risk of food poisoning by usually heating to a core temperature of 75°C.

Genetically modified ingredients

European Union (EU) regulations stipulate that foods that contain genetically modified organisms (GMO) or ingredients made from GMOs must be indicated on the label. Foods produced using genetically modified (GM) technology and animal products from animals fed GM feed do not have to be labelled. Loose GM food must be displayed next to information that states that it is genetically modified. More information is available from the FSA (www.food.gov.uk) and the Department for Environment, Food and Rural Affairs (http://www.defra.gov.uk).

Nutrition information labelling

Food manufacturers are not required by law to display nutrition labelling. If a nutrition claim, e.g. high fibre, is made this must be supported by nutrition information on the label. Labels may state recommended daily amounts or guideline daily amounts. EU regulations allow two nutrition labelling systems. The first system requires that the energy content of the food be given in kJ or kcal and the amount, in grams, of protein, carbohydrate, and fat per 100 g or 100 ml; manufacturers may also show values per item or average serving. The second system supplies these details together with information on sugars, fibre, sodium, and saturated fat. Details on starch, monosaturated fat, polyunsaturated fat, cholesterol, and some minerals and vitamins if they are present in significant amounts may also be given.

Nutrition and health claims

In July 2007 a new Regulation set out clear rules on nutrition and health claims to protect consumers from misleading or false claims. Table 8.1 defines what a nutrition claim is and what a health claim is. The regulation not only covers claims made in words e.g. high in fibre, but also claims made as pictures, graphics or symbols e.g. a heart shape. It regulates claims made to consumers whether on food packets, websites or leaflets.

Nutrition labelling must be given if a nutrition claim is made. Only authorized claims may be made. The list of nutrition claims has been published (see Table 8.2) and work continues on the list of health claims. Details can be found on the EFSA website (http://ec.europa.eu/food/food/labellingnutrition/claims/community_register/index_en.htm) including a list of rejected and authorized health claims. Details of the governing regulation Annex of Regulation (EC) No 1924/2006 can also be found on the FSA website www.food.gov.uk).

Organic

In the UK the Department for Environment, Food and Rural Affairs (DEFRA) regulates the bodies that can certify organic producers and manufacturers. 75% of certification is administered by the Soil Association. Organic food products can be sold in two categories:

- Category 1: Organic. Must contain at least 95% of organic ingredients by weight and can be labelled 'organic'.
- Category 2: Special emphasis. Product contains 70–95% organic ingredients by weight and can be labelled as 'made from organic ingredients'.

Organic food cannot legally contain GM or irradiated foods.

Signposting

Traffic light labelling

The FSA has recently introduced traffic light labelling as a front of package system that gives an at-a-glance indication of the total fat, saturated fat, sugars and salt content of pre-packaged foods. Quantities are given in weight per average serving. Red, amber and green are used to indicate a 'high' content; amber 'medium' content and green 'low' content. Further details are available from the FSA web site www.food.gov.uk.

Guideline daily amounts

Some pre-packaged foods provide information about guideline daily amounts GDAs. GDAs are derived from the EARs for energy for men and women, aged 19–50 years, and normal weight and fitness. The values for fat and saturated fatty acids are derived from DRVs and salt is based on Scientific Advisory Committee on Nutrition (SACN) recommendations. They are intended as a guide for the consumer when comparing products. Revised GDAs (Table 8.3) were published by the Institute of Grocery Distribution (IGD) in 2005. GDAs are now available for children. Further details are available from the IGD web site (www.igd.com).

Table 8.1 Definitions of nutrition and health claims

Claim	Definition	Example
Nutrition claim	'Any claim which states, suggests or implies that a food has particular beneficial nutritional properties due to (a) the energy it provides at a reduced or increased rate, or does not provide, and/or (b) the nutrients or other substances it contains in reduced or increased proportions, or does not contain'	'High fibre, 'low energy', 'light', 'source of protein', 'high in vitamin D'.
Health claim	'Any claim that states, suggests or implies that a relationship exists between a food category, a food or one of its constituents and health'	'Calcium helps build strong bones', 'good for you'

Table 8.2 Nutrition claims and the conditions applying to them

Nutrient	Claim	Conditions*
Energy	Low energy	≤40 kcal (179 kJ) per 100 g solids ≤20 kcal (80 kJ) per 100 ml liquids
	Energy reduced	≥30% reduction
	Energy free	≤4 kcal (17 kJ) per 100 ml
Fat	Low fat	≤3 g per 100 g solids ≤1.5 g per 100 ml liquids 1.8 g per 100 ml for semi-skimmed milk
	Fat free	≤0.5 g per 100 g or 100 ml 'X% fat free' prohibited
	Low saturated fat	≤1.5 g per 100 g solids ≤0.75 g per 100 ml liquids
	Saturated fat free	≤0.1 g per 100 g or 100 ml
	High unsaturated fat	≥70% of the fatty acids present derive from unsaturated fat under the condition that unsaturated fat provides >20% of energy of the product
	High polyunsaturated fat (PUFA)	≥45% of the fatty acids present derive from PUFA under the condition that PUFA provide >20% of energy of the product
	High monounsaturated fat (MUFA)	≥45% of the fatty acids present derive from MUFA under the condition that MUFA provide >20% of energy of the product
	Source of omega-3 fatty acids	≥0.3 g alpha-linolenic acid per 100 g and per 100 kcal, or ≥40 mg of the sum of eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) per 100 g and per 100 kcal
	High omega-3 fatty acids	≥0,6g alpha-linolenic acid per 100 g and per 100 kcal, or ≥80 mg of the sum of EPA and DHA per 100 g and per 100 kcal
Sugar	Low sugar	≤5 g per 100 g solids 2.5 g per 100 ml liquids
	Sugar free	≤0.5 g per 100 g or 100 ml

Table 8.2 (*Contd.*)

Nutrient	Claim	Conditions*
	With no added sugar	'does not contain any added mono- or disaccharides or any other food used for its sweetening properties. If sugars are naturally present in the food, the following indication should also appear on the label: 'CONTAINS NATURALLY OCCURRING SUGARS'.'
Sodium/salt	Low sodium/salt	≤0.12 g sodium or equivalent value for salt per 100 g or 100 ml
	Very low sodium/salt	≤0.04 g sodium or equivalent value for salt per 100 g or 100 ml
	Sodium or salt free	≤0.005 g sodium or equivalent value for salt per 100 g or 100 ml
Fibre	Source of fibre	≥3 g per 100 g solids ≥1.5 g per 100 kcal
	High fibre	≥6 g per 100g ≥3 g per 100 kcal
Protein	Source of protein	≥12% energy from protein
	High protein	≥20% energy from protein
Vitamins/ Minerals	Source of (name of vitamin/s) or (name of mineral/s)	at least a significant amount (as a rule, significant amount means 15% of the recommended daily allowance provided by 100g or 100ml of the food, or per package if contains only a single portion)
	High (name of vitamin/s) or (name of mineral/s)	at least twice the value of 'source of' as above
Comparative claims	Increased	Meets the conditions for 'source of' and the increase is ≥30% compared with a similar product
	Reduced	≥30% compared with a similar product 10% for micronutrients 25% for sodium or salt
	Light/lite	As for 'reduced'
Naturally/ natural	Where a food or drink naturally meets the conditions for the use of a nutrition claim, the term 'naturally/natural' may be used as a prefix to the claim	

*See Annex of Regulation (EC) No 1924/2006 for full details.

Table 8.3 Guideline daily amounts (GDAs) for adults as used in UK (IGD)

	GDA	
	Women	**Men**
Energy (kcal)	2000	2500
Fat (g)	<70	<95
Of which saturates (g)	20	30
Total carbohydrate (g)	230	300
Of which sugars (g)	<90	<120
% Total energy		
NSP/fibre (g)	>24	>24
Sodium (g)*	2.4	2.4
*Equivalent as salt (g)	6	6

Functional foods and nutraceuticals

- *Functional Foods* are foods that when consumed regularly, as part of the usual diet, exert a specific health-beneficial effect beyond their basic nutritional value.
- *Nutraceuticals* (nutrition and pharmaceutical) are products isolated or purified from foods, and generally sold in medicinal forms (such as a pill or capsule) and demonstrated to have a physiological benefit or provide protection against chronic disease.

Many nutrients and foods have nutrient or health claims as functional foods or nutraceuticals but the claims are not always proven. There is currently no law that controls these claims. It is generally accepted that health claims such as 'can help lower cholesterol as part of a low fat diet' can be made if the claim is supported by scientific research and is not misleading (📖 this Chapter, 'Food labelling'). Functional or 'novel' foods must go through a safety approval process before they are launched. Products that were on sale before 1997 do not have to undergo this process. In the UK the Joint Health Claims Initiative has established guidance on a voluntary scheme for health claims (🖱 www.jhci.org.uk).

A functional food may be:

- A natural food.
- A food to which a component has been added.
- A food from which a component has been removed.
- A food where one or more components has been modified.
- A food in which the bioavailability has been modified.

Categories of functional foods and nutraceuticals are shown in Table 8.4.

Functional food ingredients

The beneficial health effects of functional foods are due to the presence of a variety of bioactive components that elicit their effects via a number of different mechanisms. These substances (bio-actives) often originate from plant sources but some are also derived from animals and micro-organisms. Box 8.2 and Table 8.5 list examples of functional food ingredients.

Regulation of functional foods

The effects of functional foods on health must be scientifically proven. In Europe, the assessment of the scientific evidence to support health claims is the responsibility of the European Food Safety Authority (EFSA) (🖱 www.efsa.europa.eu). The process of assessment of claims is currently ongoing, but to date positive EFSA opinions on claims have included: plant sterol/stanols and cholesterol reduction, xylitol and caries reduction, alpha-linolenic acid (ALA) and brain development in children, long-chain polyunsaturated fatty acids and visual development in children.

Table 8.4 Categories of functional foods and nutraceuticals

Category	Example
Basic food	Tomatoes (rich in natural antioxidant lycopene)
Processed foods	Oat bran cereal
Processed food with added ingredients	Calcium-enriched fruit juice
Foods enhanced to have more of a functional component	Oat bran with higher levels -glucan
Isolated, purified preparations of active food ingredients	Isoflavones from soya

Adapted with permission from Arvanitoyannis, I.S. and Van Houwelingen-Koukaliaroglou, M. (2005). Functional foods: A survey of health claims, pros and cons, and current legislation. *Crit. Rev. Food Sci. Nutr.*, **45**, p. 390, table 3.

Box 8.2 Examples of Functional Foods

- Dairy spreads enriched with plant sterols/stanols
- Omega-3 enriched eggs and bread
- Yoghurts with probiotics and prebiotics
- Oat breakfast cereal rich in β-glucan

Table 8.5 Examples of bioactive functional food ingredients

Functional Ingredient	Food Source	Potential Effect on Health
β-glucan	Oats	↓ blood cholesterol; ↓ the postprandial glycaemic response; aid weight control
Conjugated linoleic acid (CLA)	Cheese, meat products	Improve body fat composition, enhance immune system; ↓ risk of cancer
Flavonoids (ex. catechins)	Tea, fruits, vegetables	Neutralize free radicals; ↓ risk of cancer
Lycopene (carotenoid)	Tomatoes	↓ risk of prostate cancer
Omega-3 fatty acids (ALA/EPA/DHA)	Salmon and fish oils;	↓ risk of cardiovascular disease (CVD); may improve mental and visual functions
Phytoestrogens (e.g. Isoflavones)	Soybeans and soy-based foods	Alleviate symptoms associated with the menopause; protect against heart disease and some cancers; ↓ LDL and total cholesterol; improve bone mineral density
Plant steols/stanols/ stanol esters	Corn, soy, wheat, wood oils	↓ absorption of cholesterol in the body and ∴ lower blood cholesterol levels (total and LDL-cholesterol by ~10%). ❗ May interfere with absorption of other fat-soluble components from the diet, data on effects of long-term consumption at levels >3g/day are limited
Prebiotics (e.g. inulin, Fructoligosaccharides)	Jerusalem artichokes, shallots, onion powder	Not digested by the gut and stimulate the growth of certain bacteria in the colon. Improve quality of intestinal microflora; GI health
Probiotics ('live' bacteria e.g. *Lactobacillus acidophilus*)	Yoghurt and other dairy products	Improve quality of intestinal microflora; GI health

Nutrigenetics and nutrigenomics

Nutrigenetics is the influence of the genotype on nutritionally related disease; nutrigenomics is the effect of diet on whole-body metabolism (Fig 8.1).

Nutri(epi)genetics refers to the study of heritable changes in gene expression that occur without a change in the primary DNA sequence. Epigenetic mechanisms of altering gene regulation are DNA methylation histone modifications and genetic imprinting (see Box 8.3).

Nutrigenomics refers to the study of interactions between dietary factors and genes that can promote health or cause disease. It involves the use of various molecular tools (genomics, transcriptomics, proteomics, metabolomics) to explore how dietary substances interact with the genome (see Box 8.4). Nutrigenomics views nutrients and bioactive food components as 'dietary signals' that can directly or indirectly alter the genomic structure or function and molecular events. The ultimate goal of nutrigenomics is to determine the dietary factors that are most compatible with health for a given individual (personalized nutrition).

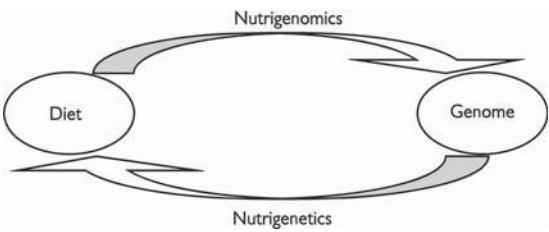

Fig. 8.1 The relationship between nutrigenomics and nutrigenetics.

Box 8.3 Nutrigenetics

- Aims to understand how the genetic makeup of an individual coordinates their response to diet
- Characterizes gene variants associated with differential responses to nutrients and relates this variation to disease states
- Typically characterizes SNPs (single nucleotide polymorphisms) with regards to their frequency in a given population and to their relevance for metabolic health disorders.

Box 8.4 Nutrigenomic technologies

- *Transcriptomics:* The simultaneous analysis of the mRNA transcripts from a cell's genome, including modifications that may occur to the transcripts. It provides a comprehensive system-wide view of gene-expression patterns in states of health and disease. Nutritional transcriptomics studies the influence of nutrients and bioactive food components on global gene expression and transcription.
- *Transcriptome:* The complete collection of gene transcripts (mRNAs) in a cell or tissue at a given time.
- *Proteomics:* The study of proteomes which attempts to determine their role inside the cells and molecules with which they interact. Proteomics refers to techniques for measuring global protein expression.
- *Proteome:* The complement of expressed proteins in a biological system whether it be a cell, its organelles, or the entire organism at a given time.
- *Metabolomics:* The study of small molecules or metabolites present in biological samples (such as biofluids, tissues and cellular extracts) which attempts to correlate the metabolomic profiles with known physiological or pathological states. The aim of metabolomics is to profile all the metabolites present in the samples to enhance the understanding of the effect of a particular stimulus on metabolic pathways. Metabolomics allows the analysis of hundreds of metabolites in a given biological sample.
- *Metabolome:* The full complement of metabolites in a sample at a given time. See Fig. 8.2.

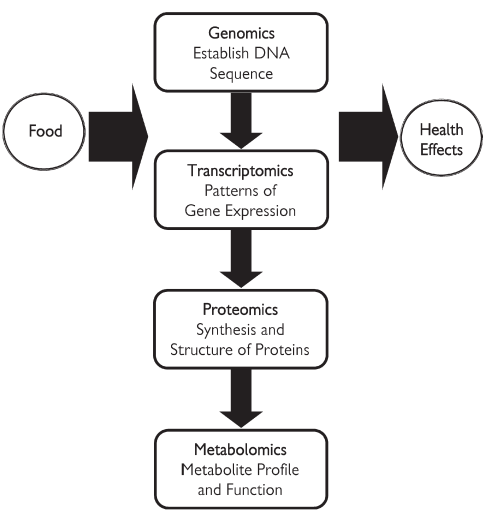

Fig. 8.2 Nutrigenomic technologies.

Nutrigenomics adheres to the following precepts[1]

- Poor nutrition can be a risk factor for diseases.
- Common dietary chemicals can act on the human genome (either directly or indirectly) to alter gene expression and/or gene structure.
- The degree to which diet influences the balance between health and disease depends on the individual's genetic makeup.
- Some diet-regulated genes (and their normal, common variants) are likely to play a role in the onset, incidence, progression and/or severity of chronic diseases.
- Dietary intervention based on knowledge of nutritional requirement, nutritional status, and genotype can be used to prevent, mitigate or cure chronic disease.

Practical applications of nutrigenomics[2]

- Identify the genes and proteins expressed differentially in health and disease that are modifiable by nutrients.
- Identify which genes, proteins and metabolites are influenced by specific nutrients that are known to be beneficial or harmful.
- Identify genetic variations that alter the nutrient-gene interactions in the applications above.

Gastrointestinal microbiota

It is estimated that there are 10^{14} bacteria in the human gut. Some components of the GI microbiota compete with enteropathogens, regulate innate and adaptive immune function and ferment non-digestible carbohydrates to produce short-chain fatty acids[3]. The GI microbiota can be modified using probiotics and prebiotics, and some studies have shown that this reduces the disease risk or severity.

Probiotics

Probiotics are defined as 'live microorganisms which when administered in adequate amounts confer a health benefit on the host.' The criteria for a probiotic include being safe for human use, able to survive transit through the gastrointestinal tract and having physiological capacity to provide health benefits. Probiotics are usually acid tolerant bacteria, such as lactobacilli and bifidobacteria, and are available in the form of functional foods (e.g. yogurts, fermented milks, juices) or as tablets, capsules, or powders.

The effect of probiotics has been investigated in numerous clinical settings. The results vary depending upon the strain and dose given, and therefore generic recommendations for probiotic use cannot be given. However, areas where evidence increasingly shows benefit for some probiotics include:

- Symptom of irritable bowel syndrome e.g. *Lactobacillus plantarum* 299V, *Bifidobacterium animalis* DN-173 010.[4]

[1] Kaput, J. and Rodrigues, R.L. (2004). Nutritional Genomics: the next frontier in the postgenomic era. *Physiol. Genom.*, **16**, 166–77.
[2] Kornman, K.S., Martha, P.M., and Duff, G.W. (2004). Genetic variations and inflammation: A practical nutrigenomics opportunity. *Nutr.* **20**, 44–9.
[3] Neish AS. (2009). Microbes in gastrointestinal health and disease. *Gastroenterology*, **136**, 65–80.
[4] Moayyedi P, Ford AC, Talley NJ, *et al.* (2010). The efficacy of probiotics in the treatment of irritable bowel syndrome: a systematic review. *Gut*, **59**, 325–32.

- Treatment and maintenance of inflammatory bowel disease e.g. *Escherichia coli* Nissle, *Saccharomyces boulardii*.[5]
- Prevention of antibiotic-associated diarrhoea e.g. *Lactobacillus caseii* DN114 011.
- Prevention of recurrent *Clostridium difficile* diarrhoea e.g. *S. boulardii*.

Prebiotics

Prebiotics are defined as non-digestible food components that "selectively stimulate the growth and/or activity of one or a limited number of microbial genera, species or strains in the gut microbiota that confer health benefits to the host". The major prebiotics are fructo-oligosaccharides (FOS), galacto-oligosaccharides (GOS) and lactulose. FOS are found naturally in chicory, wheat and onions, and GOS are found in beans and pulses. Studies have shown that in their powdered supplement form (3.5–15 g/day) prebiotics can result in the increased growth of colonic bifidobacteria.

There is emerging evidence of the role of prebiotics in irritable bowel syndrome and inflammatory bowel disease; however, further studies are warranted in these areas[6].

Food fortification

Some foods are supplemented with nutrients for their benefits above normal health benefits. In the UK flour is fortified with Ca and margarine is fortified with vitamins A and D.

Folate fortification

Folate is frequently added to cereals and there is considerable debate in the UK as to whether or not to fortify all flour and flour products. Flour is fortified with folate in USA, Canada, and Chile. The arguments for and against folate fortification are as follows.

- Arguments for the fortification of flour with folate:
  - prevention of neural tube defects;
  - low folate status is associated with elevated homocysteine which is a risk factor for cardiovascular disease and is linked to some cancers including colon and breast cancer.
- Arguments against the fortification of flour with folate:
  - there is uncertainty about the bioavailability of folate;
  - consumer choice;
  - high consumption of folic acid may mask the diagnosis of vitamin B_{12} deficiency in the elderly.

A recent SACN report has recommended the supplementation of flour in the UK although accompanied by some restrictions and a recommendation for careful monitoring of emerging evidence and dietary surveys of folate intake and status. This has not been ratified by the government. See SACN website for current status (www.sacn.org.uk).

[5] Hedin C, Whelan K, Lindsay JO. (2007). Evidence for the use of probiotics and prebiotics in inflammatory bowel disease: a review of clinical trials. *Proc. Nutr. Soc.*, **66**, 307–15.
[6] Roberfroid M, Gibson G R, Hoyle L, *et al.* (2010). Prebiotic effects: metabolic and health benefits. *Br. J. Nutr.*, **104**(Suppl. S2), S1–S63.

Food supplements

Food supplements

Food supplements are defined by EU law as 'foodstuffs the purpose of which is to supplement the normal diet and which provide concentrated sources of nutrients (vitamins and minerals) or other substances with a nutritional or physiological effect, alone or in combination, marketed in dose form . . . designed to be taken in measured small unit quantities'.

The growing interest in diet and health has stimulated the market for dietary supplements. In 1991, the market for dietary supplements was £194 million and this had risen to £396 million in 2009. The range of supplements available in health food shops, chemists, and supermarkets is growing with vitamins, minerals, fish liver oils, and evening primrose oil being the most popular. In 2008/09, 30% of women and 18% of men reported taking supplements in the UK.

Health information

The average diet in the UK supplies adequate vitamins and minerals to prevent deficiency and for the majority of people supplements are not necessary. Vulnerable groups, e.g. vegetarians, the elderly and pregnant women may benefit from supplements. There is little evidence to support the blanket use of supplements. There are a few well-established cases, e.g. additional folate in pregnancy (see 🕮 Chapter 12, 'Diet before and during pregnancy', p. 217), additional calcium in osteoporosis. Most supplements are bought over the counter and are self-prescribed.

The range of nutrition supplements is vast and cannot be adequately covered in this format. Information on the health benefits of specific supplements can be obtained from the Health Supplements Information Service (🖰 www.hsis.org).

Micronutrient supplements

In 2003, the Expert Group on Vitamins and Minerals published its report on the safety of vitamins and minerals in food supplements and fortified foods. Safe upper limits were set for all vitamins and most minerals. The full report can be found at: 🖰 www.food.gov.uk/multimedia/pdfs/vitamin2003.pdf.

Regulation

Food supplements fall between medicines and foods and it has been difficult to regulate their marketing. However in 2002 the EU issued the Food Supplements Directive and the directive and regulations have applied since August 2005. The directive lists vitamins and minerals that can be used in supplements. Work was undertaken to permit additions to the originally listed vitamins and minerals, but at this time, tin, nickel, cobalt, and vanadium are excluded.

A second list gives details of the chemical forms that may be used. These forms are considered safe. Work on setting maximum and minimum dose levels for use in food supplements is still in progress.

Labelling

Manufacturers must not make claims referring to the prevention, treatment, or curing of diseases or refer to such properties. The label must display:

- The words 'food supplement'.
- Names of the nutrients and substances.
- Portion required for daily use.
- Warning not to exceed stated dose.
- Statement to the effect that supplements should not replace a varied diet.
- Statement that the product should be stored away from children.
- It should not be implied that a balanced and varied diet cannot provide adequate amounts of nutrients.
- Amounts of nutrients available in the recommended dose.

Oral nutritional supplements

A wide range of liquid, semi-solid or powder supplement products are available that provide either one or more macronutrients or, more usually, a combination of macro and micronutrients. In many cases oral nutritional supplements are nutritionally complete (nutritionally balanced) as such they can be used as a sole source of nutrition if taken in sufficient quantities. See 🕮 Chapter 25, 'Nutrition Support' (p. 501).

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Chapter 9

Non-nutrient components of food

Alcohol

The alcohol present in alcoholic drinks is ethanol (ethyl alcohol), C_2H_5OH. Ethanol is produced by the fermentation of glucose in plants. Sugars in grapes and apples are fermented to produce wine and cider; barley starch is hydrolysed to glucose in the production of beer. Other fruits and cereals are used to produce alcoholic drinks, e.g. rice for sake and rye for whisky. The resultant alcohol is diluted to produce the appropriate alcohol content of drinks. By law, drink labels must show the strength of alcohol present; this is expressed as the percentage alcohol by volume (abv). 10% abv is equivalent to 7.9 g of alcohol per 100 ml (Box 9.1).

Box 9.1 Alcohol by volume in different alcoholic drinks

- *Ciders and beers:* 4–6% abv
- *Wines:* 9–13% abv
- *Fortified wines:* 18–25% abv
- *Liqueurs:* 20–40% abv
- *Alcopops:* 4–13% abv
- *Spirits, e.g. gin:* 40% abv

Low alcohol and strong variations of some drinks are now produced. Other substances are added to provide flavor, such as juniper berries in gin and hops in beer. Alcoholic drinks may also contain sugars, small amounts of other alcohols, e.g. propyl alcohol, potassium, and small amounts of riboflavin and niacin. In the UK 1 unit of alcohol drink contains 8 g or 10 ml of pure alcohol (see Table 9.1). Other systems are used by other countries.

Alcohol metabolism

Ethanol is quickly absorbed in the stomach and jejunum and distributed throughout total body water including blood. Alcohol is distributed via the blood to the brain and the liver where it is metabolized by alcohol dehydrogenase (ADeH) to acetaldehyde which is converted to acetate by the enzyme aldehyde dehydrogenase (ALDH). ADeH is the rate limiting step in the metabolism of alcohol and there is a great deal of variation between individuals in their ability to metabolize alcohol. On average approximately 5–10 g of alcohol (1/2–1 unit of alcoholic drink) is metabolized per hour. The stages of alcohol intoxication are shown in Table 9.2. Alcohol absorption can be slowed by the presence of food in the stomach. Smaller people have smaller livers and ∴ metabolize alcohol more slowly; women have smaller livers than men and ∴ become intoxicated more quickly. Disulfam (Antabuse®) antagonizes ALDH and is a drug used to treat alcoholism. The build up of acetaldehyde leads to headache, nausea, and vomiting. Alcoholics have an induced system of alcohol metabolism known as the microsomal ethanol-oxidizing system (MEOS); this system is thermogenic. Some alcohol is excreted in breath, which provides an easy monitor of alcohol intoxication.

Table 9.1 Drink measures equivalent to 1 unit of alcohol

Drink	Bar measure	ml
Spirit, e.g. whisky, gin, vodka	I single optic measure	25
Sherry or fortified wine, e.g. port or vermouth	1 small glass or schooner	50
Wine	1 small glass	125
Strong lager, beer, cider	¼ pint	142
Ordinary strength lager, beer, cider	½ pint	284
Low alcohol lager, beer, cider	2 pints	1136

Table 9.2 Stages of acute alcohol intoxication

Blood alcohol concentration (mg/100 ml)	Stage	Effects
Up to 50	Feeling of well being	Relaxed, talkative
50–80	Risky	Fine movements and judgement affected
80		Legal limit for drink driving prosecution in UK
80–150	Dangerous	Slurred speech, balance affected, blurred vision, drowsiness, nausea and vomiting
200–400	Drunken stupor	Dead drunk, loss of bladder and bowel control, unconscious
450–600	Death	Shock and death

Nutritional value of alcohol

Alcohol is an energy source providing 29 kJ (7 kcal) per gram. Alcoholics do not obtain this level of energy from alcohol due to the thermogenic nature of MEOS.

Recommendation on alcohol intake

In the UK the Department of Health recommends that men consume no more than 3–4 units of alcohol/day and women no more than 2–3 units. Standard measures of drinks providing 1 unit of alcohol are shown in Table 9.1. It is also recommended that 2 day/week should be alcohol free.

Alcohol consumption

It is difficult to estimate alcohol consumption due to the social stigma associated with excessive drinking and the effects of alcohol on mental capacity. The National Diet and Nutrition Survey[1] found that on average

[1] Bates, B., Lennox, A., and Swan, G. (2010). *The National Diet and Nutrition Survey: Headline results from Year 1 of the Rolling Programme (2008/2009)*. Food Standards Agency, London.

adult men consumed 29.2 units per week and women consumed 12.4 units per week; this figure was calculated including non-drinkers. The average alcohol consumption by children aged 11–18 years was 3.8 units per week.

Acute effects of alcohol

The response to alcohol is variable; this variation is due to the extent of stimulation of the sympathetic nervous system and the rate of production of acetaldehyde and acetate. The most common effects are ↑ in heart rate and peripheral vasodilatation. As a result of the peripheral vasodilatation some people feel excessively warm and some experience facial flushing. The psychological and physiological responses to acute alcohol excess are shown in Table 9.2

Alcohol is a central nervous system depressant and acts as an anaesthetic. Diuresis (increased urine production) results from the action of alcohol on the pituitary gland and leads to dehydration.

Effects of alcohol on health

Light to moderate consumption of alcohol has been shown to have beneficial health effects in reducing the risk of coronary heart disease in men and post-menopausal women. The most established mechanism is that this level of alcohol consumption ↑ plasma high-density lipoprotein. An additional proposed mechanism is that alcohol ↓ platelet aggregation and ∴↓ the risk of thrombosis. It has been proposed that polyphenolic compounds in wines have antioxidant properties that reduce the plasma levels of low-density lipoproteins.

Excessive alcohol consumption has been linked to ↑ risk of breast cancer in women and oesophageal and liver cancer in men and women. High intakes of alcohol are strongly associated with ↑ risk of liver disease. Hypertension risk is ↑ by high levels of alcohol intake.

The harmful physical effects of alcohol abuse are extensive and some are listed in Table 9.3. Thiamin deficiency can result from chronic, excessive alcohol intake as thiamin is required for ethanol metabolism and dietary intake may be poor. This can → Wernicke's encephalopathy and Korsakoff's psychosis. Other vitamin deficiencies are rare but do occur in alcoholics, e.g. folate and vitamin C. Alcohol is estimated to be a factor in 20–30% of accidents and has socio-economic consequences including domestic violence.

Alcohol and vulnerable groups

Pregnant women Alcohol may reduce the ability to conceive and excessive consumption is associated with a greater risk of miscarriage. Pregnant women are advised not to drink and to consume no more than 1–2 units per week. Excessive alcohol consumption during pregnancy can → foetal alcohol syndrome which may → facial deformities and growth problems (see 🕮 Chapter 12, 'Dietary reference values and dietary guidelines during pregnancy', p. 222).

Diabetes People with diabetes are advised not to drink excessively as this can → ↑ hypoglycaemic episodes.

Table 9.3 Physical health problems associated with excessive alcohol consumption

Body system	Effects
Nervous system	Acute intoxication, dementia, Wernicke—Korsakoff syndrome, cerebellar degeneration
Cerebrovascular system	Strokes, nerve and muscle damage
Liver	Fatty liver, cirrhosis, hepatitis, liver failure, cancer
Gastrointestinal system	Reflux, oesophageal rupture, oesophageal cancer, pancreatitis, gastritis, malabsorption
Nutrition	Reduced food intake and absorption leading to weight loss, obesity in early stages of heavy drinking
Heart and circulatory system	Arrhythmias, hypertension, heart muscle damage leading to heart failure
Respiratory system	Pneumonia from inhalation of vomit
Endocrine system	Overproduction of cortisol, hypoglycaemia, stimulation of the pituitary to cause diuresis
Reproductive system	Loss of libido, atrophy of testicles, reduced sperm count, menstrual abnormalities

Biologically active dietary constituents

Foods contain many chemicals that have no nutrient value but have physiological or pharmacological properties; these are often referred to as 'phytochemicals'. Some of these chemicals have protective properties but some are toxic or may become toxic if taken in excess.

Antioxidants and anticarcinogenic phytochemicals

Epidemiological studies have shown that fruit and vegetables have positive effects on health, particularly the prevention of cancer and heart disease, due to the presence of the antioxidant vitamins, vitamin C, vitamin E, and β-carotene, and probably some as yet to be identified antioxidants or anticarcinogenic compounds. These chemicals prevent damage to body tissues by free radicals. This group of chemicals includes the carotenoids, polyphenols, glucosinolates, phytoestrogens, and sulphides.

Carotenoids

The carotenoids consist of approximately 100 compounds that occur naturally in plants; they often give fruits and vegetables a yellow or orange colour. Some can be converted into retinol (vitamin A) (see 🕮 Chapter 6, 'Vitamin A (retinol)', p. 94); the most important of the carotenoids for retinol production is β-carotene. Carotenoids act as antioxidants by reacting with unpaired electrons in free radical and so neutralizing them. Another carotenoid that has received a lot of attention is lycopene; 85% of dietary lycopene is derived from tomatoes. Some studies have shown that lycopene may have anticarcinogenic properties and reduce the risk of heart disease.

Polyphenols

Phenolic compounds are found in many foods and beverages and act as a plant's defence system against animals and insects. Polyphenols are either antioxidants or potentiate the effects of other antioxidants. The following chemicals are types of polyphenols.

- Phytosterols are found in seeds and oils and inhibit cholesterol absorption.
- *Flavanoids:* over 4000 types have been identified. They are found in fruits, vegetables, nuts, and seeds. Onions, apples, and black tea are particular rich sources. Quercetin and catechins are examples of flavanoids.
- Tannins are present in red wines and tea adding colour and flavour. They are antioxidants but bind to Fe and inhibit Fe absorption.
- Phytoestrogens are plant chemicals that are chemically similar to the animal hormone oestradiol. They compete for oestradiol receptors and can either ↑ or ↓ the effects of oestradiol. Isoflavones are phytoestrogens that are found in soya beans and products.
- Soya isoflavones have been shown to have hormonal effects and have been used to alleviate menopausal symptoms.
- Soya isoflavones have been shown to have anticarcinogenic properties in *in vitro* studies but the effects are variable in humans. Some studies have suggested that excessive intakes of isoflavones may in fact be carcinogenic; ∴ concentrated supplements are not recommended.

- Sulphides are present in foods such as onion, leek, and garlic. They have been shown to have anticarcinogenic properties in animal studies. There is some epidemiological evidence to suggest that sulphides reduce the risk of colorectal and gastric cancers.
- Glucosinolates are present in plants of the brassica family such as cabbage, broccoli, kale, Brussels sprouts, and cauliflower. There is some evidence of anticarcinogenic properties of glucosinolates in experimental studies, but large intakes may actually be carcinogenic; more studies are required.

Caffeine and methylxanthines

Methylxanthines are a group of chemicals that includes caffeine, theophylline, and theobromine. They occur naturally in foods and drinks such as tea, coffee, cola drinks, and cocoa products (Box 9.2); they are also added to 'energy' drinks. Caffeine is a mild stimulant although there is individual variation in this response. It is mildly addictive and abrupt stoppage of caffeine intake can → mild withdrawal symptoms of headache, fatigue, and irritability. A daily intake of 4–5 cups of coffee is considered moderate; caffeine intake is dependent on the size of cup, the fineness of grinding, brewing method, roasting of beans, and type of coffee beans used. Arabica coffee beans contain less caffeine than Robusta beans. Tea has higher caffeine content than coffee on a dry weight basis but less tea is used to produce a drink.

By law drinks containing caffeine in excess of 150 mg/l must carry a declaration in the same part of the label as the name of the food. Caffeine content, with the amount of caffeine expressed in mg per 100 ml, should be given to identify high levels of caffeine in some drinks (Box 9.2). This law does not apply to drinks based on tea or coffee, or coffee or tea extract.

Box 9.2 Caffeine content of beverages

300 mg of caffeine is roughly equivalent to

- 4 average cups or 3 average size mugs of instant coffee
- 3 average cups of brewed coffee
- 6 average cups of tea
- 8 cans of regular cola drinks
- 4 cans of so-called 'energy' drinks
- 400 g (8 standard 50 g bars) of plain chocolate

Household measures of caffeine:

- Average cup of instant coffee—75 mg
- Average mug of coffee—100 mg
- Average cup of brewed coffee—100 mg
- Average cup of tea—50 mg
- Regular cola drink—up to 40 mg
- Regular energy drink—up to 80 mg
- Plain bar of chocolate—up to 50 mg. Caffeine in milk chocolate is about half that of plain chocolate

Caffeine is a mild diuretic if taken in quantities above that considered moderate, 4–5 cups per day. The evidence that caffeine reduces fertility is inconclusive. The Food Standards Agency recommends that pregnant women should not have more than 200 mg of caffeine per day (see 📖 Chapter 12, 'Diet before and during pregnancy', p. 217). Some studies suggest that higher intakes are linked with miscarriage, low birth weight, and premature delivery. This is disputed by some researchers who have suggested that high caffeine intake is an indication of low hormone levels. Many women have a reduced desire for coffee during pregnancy, which is believed to be due to high placental hormone levels, ∴ high coffee intake may be a marker of low hormone levels. The FSA recommends that coffee intake during breastfeeding should not be stopped but that caffeine should only be taken occasionally as caffeine passes into breast milk causing the baby to be restless and agitated (see 📖 Chapter 13, 'Breast versus bottle feeding', p. 242).

Decaffeinated brands are not totally free of caffeine, but usually contain <5 mg caffeine per cup; they contain smaller amounts of the other methylxanthines than normal brands.

Theobromine levels are low in beverages except chocolate products. High cocoa (70% cocoa beans) content brands have higher levels of caffeine and theobromine than average brands.

Vasoactive amines

Tyramine, histamine, tryptamine, and serotonin are all present in foods and are normally deactivated in the body. High intakes and intake in individuals with an impaired ability to deactivate them can → vasoconstrictive effects. Vasoactive amines can trigger migraine in susceptible people. People taking monoamine oxidase inhibitor type A drugs may experience a dangerous hypertensive interaction with vasoactive amine (see 📖 Chapter 38, 'Drug–nutrient interactions', p. 738).

Food additives

Food additives are substances added to food for technological reasons, which may be their organoleptic properties. They are classified into groups according to their purpose. The numbering system is being adapted for international use by the Codex Alimentarius Commission. The International Numbering System (INS) will use the same numbers used within the European Community but without the E prefix. The use of food additives is controlled by the Food Standards Agency in the UK and European Scientific Committee for Food (SCF) in Europe. Food additives must gain approval before their use in food manufacture is permitted at specified levels. The approval process is lengthy and detailed with most of the research being funded by the food manufacturer. Some additives are naturally occurring substances but they must also undergo safety testing and approval before they can be used in food manufacture. Approximately 3500 additives are in use today. Additives are used for the following reasons:

- the need to keep foods fresh until eaten so widening food choice and availability;
- convenience of packaging. storage, preparation, and use;
- attractive presentation;
- economic advantage, e.g. longer shelf life or reduced cost;
- nutritional supplementation.

E numbers

E numbers identify permitted food additives regarded as safe for use within the European Union. Some additives have a number but no E prefix as they are under consideration by the European Commission. All food labels must show the additive's name or E number in the list of ingredients.

Additive groups

Colourings (E100–180)

Food is coloured to restore losses that occur in manufacture and storage, to meet consumer expectations, and to maintain uniformity of products. An example of this is that oranges have green patches when picked and are coloured orange before sale. Azo, coal tar based, dyes are frequently linked to food allergy (see 📖 Chapter 37, 'Food hypersensitivity', p. 730). Lists of some natural and synthetic colours are shown in Tables 9.4 and 9.5.

Preservatives (E200–290)[1]

Preservatives are used to prevent food spoilage and enable the consumers to have a wide range of goods that are available out of the usual season. Traditional preservatives include salt, vinegar, alcohol, and spices. Acetic acid is the major component of vinegar and may be considered as a natural additive but it has undergone extensive testing and has an E number E260. Benzoic acid and benzoates occur widely in fresh foods, e.g. peas, bananas, and berries. Although rare, adverse reactions to benzoates have been seen. Some commonly used preservatives are shown in Table 9.6.

[1] The preservative lysozyme has the E number, E1105.

Sulphur dioxide destroys thiamin and ∴ is not permitted in foods that are significant sources of thiamies. Sulphur dioxide is used to destroy yeasts which can cause fermentation in food products.

Nitrates and nitrites kill the bacteria that cause botulism, a potentially lethal form of food poisoning. They preserve the red colour in meat and are ∴ used in meat products. A major source of these chemicals in the body is fertilizers that use these chemicals. Nitrites may react with other chemicals in the gut to form nitrosamines, which have been shown to cause cancer in experimental animals. There is no evidence to support the suggestion that these preservatives play a role in causing cancer in man.

Antioxidants (E300–322)[2, 3]

These additives prevent the unpleasant taste and smell that occur when fats and oils go rancid. The most widely used antioxidants, butylated hydroxyanisole (BHA) and butylated hydroxytoluene (BHT), are used in a wide variety of foods. Table 9.7 shows the permitted antioxidants.

Table 9.4 Examples of natural colours*

Name	E number	Food use
Riboflavin (yellow)	E101	Processed cheese
Chlorophyll (green)	E140	Fats, oils, canned & dried vegetables
Carbon (black)	E153	Jams, jellies
α Carotene (yellow/orange)	E160	Margarine & cakes

* Reproduced with permission from Webster–Gandy, J. (2000). *Understanding Food and Nutrition*. Part of the Family Doctor Series Ltd in association with the British Medical Association.

Table 9.5 Examples of synthetic colours*

Name	E number	Food Use
Tartrazine (yellow)	E102	Soft drinks
Sunset (yellow)	E110	Orange drinks
Amaranth (red)	E123	Blackcurrant products
Erythrosine (red)	E127	Glace cherries
Indigo carmine (blue)	E132	Savoury food mixes
Green S	E142	Tinned peas, mint jelly and sauce

* Reproduced with permission from Webster–Gandy, J. (2000). *Understanding Food and Nutrition*. Part of the Family Doctor Series Ltd in association with the British Medical Association.

[2] The antioxidant 4-Hexylresorcinol is E586.
[3] Agents with E numbers outside this grouping: lecithins E322, invertase E1103.

Emulsifiers, stabilizers, thickeners, and gelling agents (E400–495)[6]

These additives are needed to ↑ the shelf life of some foods and are shown in Table 9.8. They affect the texture and constituency of products. This is the largest group of additives and many are natural substances, e.g. carrageenan, which is derived from seaweed. Polyphosphates have received a great deal of attention from consumer groups as they enable products to retain water so ↑ the product's weight. They are used in products such as frozen poultry and cured meats.

Sweeteners

Sweeteners are divided into 2 groups:

- *Caloric sweeteners:* mannitol (E421), sorbitol and sorbitol syrup (E420), isomalt (E953), maltitol and maltiol syrup (E965), xylitol (E967), erythritol (968), and lactitol (E966). These additives add energy to the diet;
- *Non-caloric sweeteners:* acesulfame K (E950), aspartame (951), cyclamic acid and its salts (E952), saccharine and its salts (E954), thaumatin (E957), neohesperidine (E959), sucralose (E955) and the salt of aspartame—acesulfame (E962). Sucrose, glucose, fructose, and lactose are all classified as foods rather than sweeteners or additives.

Other additives

These include:

- flavour enhancers, e.g. monosodium glutamate (E621);
- anti-foaming agents that prevent frothing during processing;
- propellant gases, e.g. in aerosol cream.

A full list of E numbers is available from the Food Standards Agency website http://www.food.gov.uk.

Table 9.6 Commonly used preservatives*

Name	E number	Food use
Sorbic acid and derivatives	E200–E203	Cheese, yogurt, soft drinks
Acetic acid	E260	Pickles, sauces
Lactic acid	E270	Margarine, confectionery, sauces
Propionic acid and derivatives	E280–E283	Bread, cakes, flour
Benzoic acid and derivatives	E210–E219	Soft drinks, pickles, fruit products, jams
Sulphur dioxide	E220	Soft drinks, fruit products, beer, cider, wine
Nitrites	E249, E250	Cured meats, cooked meats, meat products
Nitrates	E251, E252	Bacon, ham, cheese (not cheddar or cheshire)

* Reproduced with permission from Webster–Gandy, J. (2000). *Understanding Food and Nutrition.* Part of the Family Doctor Series Ltd in association with the British Medical Association.

Table 9.7 Permitted antioxidants*

Name	E number	Food use
Ascorbic acid (vitamin C) and derivatives	E300–E305	Beer, soft drink, powdered milks, fruit, meat products
Tocopherols (vitamin A) and derivatives	E306–E309	Vegetable oils
Gallates	E310–E320	Vegetable oils and fats, margarine
BHA	E320	Margarine, fat in baked products, e.g. pies
BHT	E321	Crisps, margarine, vegetable oils, convenience foods

* Reproduced with permission from Webster–Gandy, J. (2000). *Understanding Food and Nutrition*. Part of the Family Doctor Series Ltd in association with the British Medical Association.

Table 9.8 Examples of emulsifiers, stabilizers, thickeners, and gelling agents

Name	E Number	Food use
Lecithins (may be used as an antioxidant)	E322	Chocolate, margarine, potato snacks
Citric acid and derivatives	E472c	Pickles, dairy products, baked products
Tartaric acid and derivatives	E472d–f	Baking powder
Alginic acid	E400–E405	Ice cream, instant desserts, and puddings
Agar	E406	Tinned ham, ice cream
Carrageenan	E407	Ice cream
Gums	E410–E418	Ice cream, soups, confectionery
Pectin	E440	Preserves, jellies

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Chapter 10

Nutrition and catering in institutions

Introduction

In the UK approx. £2 bill/y is spent on providing food and drink in public institutions to service users, staff and the general public. However the quality and safety of the food is equally as important as the cost. In 2006 the National Audit Office (www.nao.gov.uk) published a report on how public sector organizations could become more effective procurers of food whilst maintaining or improving quality and reducing costs.[1] This report encompassed food provision in the National Health Service (NHS), schools and nurseries, children in residential accommodation, residential care for adults and older people, the prison service, and the armed services.

As a result of this report the Food Standards Agency (FSA) commissioned the Caroline Walker Trust (CWT) (www.cwt.org.uk) to prepare a report on nutritional guidelines for food served in public institutions.[2] Following the CWT report the FSA produced food-based guidelines for adults in UK institutions and guidance for caterers. The guidance is applicable to the NHS, prisons, and residential care for adults and older people.

[1] National Audit Office (2006). *Smarter food procurement in the public sector*. The Stationery Office, London.

[2] Crawley, H., on behalf of the Caroline Walker Trust. (2007). *Food Standards Agency*. Available at: www.food.gov.uk/multimedia/pdfs/walkertrustreport.pdf.

Hospital catering

In the UK, NHS hospital catering provides ~300 million meals each year. These are consumed by individuals who vary from those who are in good health to those who are very sick and may have highly specialized nutritional requirements; food may be provided in the form of a single meal or as an individual's exclusive intake over a long period of time (>3 months) or even indefinitely. The nutritional needs of such individuals vary considerably and yet the importance of providing optimum nutrition is imperative to promote healing and recovery, to minimize complications associated with poor nutrition, and to maintain and optimize health and quality of life. Meeting all these aims, providing food people want to eat and achieving a balance between and under- and overnutrition in an environment of financial constraint, and where food is often regarded as being of little significance is a major challenge. Provision of food is one important factor amongst many that influence the nutritional status of hospital patients (see Box 10.1, and 📖 Chapter 25, 'Nutrition support', p. 501).

Box 10.1 Factors influencing nutritional status

Missing meals contributes to inadequate intake

Hypothetical case

- Patient X, admitted for investigations prior to major surgery
- Weight loss ±12 kg in last 3 months, now BMI = 18.1 kg/m^2
- Misses breakfast for three consecutive mornings as nil by mouth prior to investigative procedures
- Misses lunch on two of these days as off ward at meal time and on third day as asleep following sedation
- Early evening ward round followed by student teaching curtails time available to eat evening meal
- Eats small amount of evening meal but unable to achieve adequate nutrient intake for whole day at one meal.

Optimizing nutritional intake through hospital food service

- *WARD KITCHEN SERVICES* means tea, toast, fruit juice available 24 h so that an early breakfast can be provided before nil by mouth restrictions start;
- *SNACK BOX* can be ordered so that a patient missing lunch due to visits to X-ray, physiotherapy, etc., can eat on their return, e.g. sandwiches, cheese and biscuits, fruit and drink;
- *PROTECTED MEAL TIMES* mean that non-urgent clinical activity stops so that patients can eat without interruptions and that ward staff can provide food-related assistance, e.g. feeding, when it is required. This includes ward rounds, teaching and visiting times.

Better Hospital Food

Better Hospital Food (BHF) was introduced by the Department of Health to ensure consistent delivery of high quality food and food services to patients. Although the BHF initiative has finished, elements introduced have been continued. These include:

- Flexible menus providing greater choice of meals.
- Protected meal times to allow hospital patients to eat their meals without interruption.
- 24-h catering providing food at any time of the day or night.
- Sustainability to help reduce the environmental impact of food production.
- Nutrition that is adequate and appropriate.

The initiative now sits with the Hospital Caterers Association.
http://www.hospitalcaterers.org.

Council of Europe (CoE)

The CoE report, *Food and Nutritional Care in Hospitals: How to Prevent Undernutrition* (2003), makes 117 recommendations which include topics covered elsewhere, e.g. nutritional screening and nutritional support (see Chapter 25, 'Nutrition support', p. 501), as well as aspects of ordinary food, the distribution of responsibility for nutritional care, communication, nutrition education, organization of food service (including contract food services), eating environments, food hygiene, and economic cost. The implementation of these recommendations requires agreement at national and local level.

STILL Hungry to be heard

A campaign initiated by Age Concern (now part of Age UK) to address inadequate nutrition in older people in hospital.
http://www.ageuk.org.uk.

Red tray initiative

Some UK hospitals have adopted a system where patients who are at nutritional risk are given their food on an easily identifiable tray (e.g. coloured red; see Box 10.2) so that their intake can be monitored and appropriate eating/nutritional support given at meal times. Staff training is also provided. The introduction of red trays in an acute teaching hospital resulted in significant increases in energy and protein intakes in 64 patients at high nutritional risk and needing assistance with feeding.[1]

Box 10.2 Red tray education at Milton Keynes General Hospital NHS Trust

Remember the vulnerable patient in need of extra help at mealtimes
Encourage and assist patients where necessary
Dietary intake may be improved by extra attention at mealtimes

Tell patients and relatives about the benefits of the Red Tray Project
Remove red tray ONLY after recording food consumption
Assess and weigh patients regularly
You can improve the patients' mealtime experience!

[1] Newall, S., *et al.* (2010). The impact of a red tray system on feeding assistance and dietary intake in an acute teaching hospital. *J. Hum. Nutr. Dietet.* **23**, 446–7.

Types of hospital food production

- *Cook–Serve*: food is prepared within the hospital site and served more or less immediately to patients and staff.
- *Cook–Chill*: food is prepared either within the hospital site or at another venue and rapidly chilled to ~0–4°C and stored for up to 5 days before regeneration (reheating) either in the hospital kitchen or at ward level.
- *Cook–Freeze:* food is prepared either within the hospital site or at another venue and rapidly frozen to approximately –18°C and stored for up to 3 months before regeneration (reheating) usually in the hospital kitchen.
- *STEAMPLICITY*® (Compass Group, Uxbridge): a commercially developed system that uses raw, semi, or fully cooked chilled foods that are either served on plates or in bulk packs, which are encapsulated in plastic film with a special steam valve. These are heated in a microwave for a specific time before serving.

Types of hospital food service

- *Plated meal service*: individual trays are served out for each patient in the main hospital kitchen and transported to the ward either in insulated trolleys (if hot and cold food) or chilled trolleys (if regeneration takes place at ward level).
- *Bulk service*: large containers of food are sent from the kitchen to each ward/dining area, where staff, e.g. Nursing, care, or catering assistants or ward hostesses, serve out the meals for each patient. Food sent in bulk containers may be already hot, or still be chilled and regenerated at ward level.

Schools

Increasing levels of childhood obesity, and attempts to protect nutritionally vulnerable groups and children receiving free school meals have resulted in many government policies and initiatives in the UK and other countries. In UK primary schools the take up rate for school lunches is approx. 45% and in secondary schools it is approx. 40%. School lunches represent a valuable opportunity to improve the nutritional intake of children and for nutrition education. However the initiatives also encompass children who receive their lunches outside of the school system. See 🕮 Chapter 14, 'School-aged children and adolescents' (p. 279) for nutrition, food problems and factors that influence food choice in this age group.

The Healthy Schools Programme was launched in 1999 and since 2000 these initiatives have included:

- Food in Schools Programme (FSP) (2001);
- Education Act (2002) amended to widen the eligibility criteria for free school meals;
- in 2005 the FSP launched the 'Food in Schools Toolkit';
- the School Food Trust (SFT) was established in 2005.

The School Food Trust

SFT (www.schoolfoodtrust.org.uk) is an independent, non-departmental public body that 'promotes the education and health of children and young people by improving the quality of food supplied and consumed in schools'. It has produced food- and nutrient-based standards for lunch and non-lunch food (Table 10.1); all schools, including special schools and pupil referral units, are expected to meet these standards.

❗ A new UK government took office in April 2010 and information on the SFT web site may not reflect current polices. Policies are currently under review; future policies can be found at the Department for Education (www.education.gov.uk).

Nutrient based standards

These should be applied to an average school lunch, rather than an individual. The average school lunch must provide:

- the amount of energy shown in Table 10.1 (±5%);
- < max. total fat, sat. fat, NMES, and sodium;
- ≥ min. carbohydrate, protein, fibre, vitamins C and A, folate, calcium, iron, and zinc.

Table 10.1 School Food Trust nutrient-based school standards*

Nutrient	Minimum or maximum	Primary	Secondary*
Energy (kJ)		2215 ± 5% (111)	2700 ± 5% (136)
(kcal)		530 ± 5% (26.5)	646 ± 5% (32.3)
Carbohydrate (g)	Min	70.6	86.1
Non-milk extrinsic (NME) sugars (g)	Max	15.5	18.9
Fat (g)	Max	20.6	25.1
Saturated fat (g)	Max	6.5	7.9
Protein (g)	Min	7.5	13.3
Fibre (g)	Min	4.2	5.2
Sodium (mg)	Max	499	714
Vitamin A (μg)	Min	175	245
Vitamin C (mg)	Min	10.5	14.0
Folate (μg)	Min	53	70
Calcium (mg)	Min	193	350
Iron (mg)	Min	3.0	5.2
Zinc (mg)	Min	2.5	3.3

* Source School Food Trust www.schoolfoodtrust.org.uk/the-standards.

Food-based standards

The standards have three food groupings: foods that are allowed, food or food groups where the frequency or amount is restricted, and food or food groups that are no longer allowed (Table 10.2).

Table 10.2 School Food Trust food-based standards for school lunches

Food/food groups	Standard	
Fruit and vegetables	≥2 portions per day per pupil	≥1—fruit ≥1—vegetables or salad
Meat, fish, and other non-diary sources of protein	No standard	
Red meat	No standard	
Fish	No standard	
Oily fish	≥Once every 3w	
Salt and condiments—restricted	Salt—none available after cooking Ketchup, mayonnaise, etc., only available in sachets not >10 g or 1 tsp.	
Snacks—restricted	No snacks such as crisps	Nuts, seeds, vegetables and fruit with no added salt or sugar are allowed. Savoury crackers and breadsticks can only be served with fruit, vegetables or dairy food as part of school lunch.
No confectionary	No chocolate, chocolate coated or flavoured biscuits, sweet or cereal bars.	
Cakes and biscuits—restricted	Allowed at lunchtime, but must not contain confectionery.	

Drinking water	Free, fresh and provided at all times	
Healthier drinks	Includes low-fat milk, lactose reduced milk; fruit juice, veg.; juice; soya, rice or oat drinks enriched with calcium, yoghurt drinks, flavoured low-fat milk	Tea, coffee and hot chocolate with <5% added sugar or honey.
Meat products—categorized and restricted	1 product from each of the following 4 groups <once per 2w: 1 Burger, chopped, or corned meat. 2 Sausage, sausage meat, luncheon meat. 3 Individual meat pie, meat pudding, pork pie, game pie, pasty, or sausage roll. 4 Any other shaped or coated meat product.	Must meet min. meat content and prohibited offal standards.
Starchy foods	No standard	If cooked in oil/fat <3 times per week.
Bread	Bread with no added fat or oil must be provided daily	
Deep-fried foods—restricted	<2 items per w	
Milk and dairy food	No standard	

Prisons

The FSA (http://www.food.gov.uk/multimedia/pdfs/catererguide.pdf) produce nutrient- and food-based guidelines, which are based on the Eatwell Plate (see 📖 Chapter 2, 'Dietary reference values and food-based dietary guidelines', p. 19) and guidance for food served in major institutions. These documents are aimed at caterers in all major institutions including prisons and the guidance includes example menus compared with dietary reference values (DRVs). Unlike other institutions, prisons provide all the foods and drinks, apart from some snacks and soft drinks, consumed by prisoners. The provision of meals and food is seen as a key issue in helping to maintain order in prisons and to improving prisoners' health. In the UK Her Majesty's Prison Service order 5000 (2008) details prison catering services. All prisons use a pre-select, multi-choice, cyclical menu for lunch and dinner, which covers a 2–5-week cycle. Where queuing is in operation, measures are taken to ensure that prisoners at the end of the queue are able to obtain their chosen meals; this reduces conflict and confrontation at the serving point. Portion sizes are tightly controlled to meet budgetary demands and for consistency.

❗ Some studies have shown a link between poor diet and antisocial behaviour; however, this is equivocal. A large UK government funded study is currently underway.

Dietetic practice in prisons

There is no guidance for dietitians working in prisons or how to deal with situations such as patients in restraints, lack of privacy, and the prison regimen. However the BMA's (2009) guidance for providing medical care in hospitals (see Box 10.3) may be followed.

Box 10.3 Guidance for providing medical care to detained prisoners in hospitals (BMA, 2009)*

- Detained prisoners must have the same standards of care as the rest of society including patient's respect for dignity and privacy.
- Risk assessment must be carried out prior to a prisoner going into hospital to determine the degree of supervision. Risk assessment includes: the prisoner's condition, any medical objection to the use of restraints, nature of the prisoner's offence, security of the consulting room and risk of violence to self or others.
- Where escape is unlikely, escort and bed watch by one prison officer, without restraints is sufficient.
- Hospitals should be informed in advance about levels of escort and restraint envisaged and hospital staff should have the opportunity to discuss when level of restraint is clinically unacceptable.

* Source BMA (2009). *Health care of detainees in police stations.* Available at: www.bma.org.uk/images/healthdetainees0209_tcm41-183353.pdf.

The Armed Forces

Food is provided in 3 areas;
- Non-operational (UK bases),
- Operational (ration packs, overseas bases, and active theatres.
- Civilian.

The Defence Food Services (DFS) Team (www.mod.uk) are responsible for food provision, meal type, and quality for the armed forces in all areas, including ships and submarines. The DFS also help develop operational ration packs (ORP). The Defence Catering Manual (Joint Services Publication 456) covers every aspect of food provision.

A system known as the Pay as You Dine (PAYD) provides catering for non-operational food service. The PAYD system replaced the daily food charge, which was deducted from service personnel's salary regardless of whether or not it was taken. PAYD is currently charged at £3.76. It is based on the provision of 3 core meals (providing a minimum of 3300 kcals in total) and personnel pay only for food that is taken; they also have access to other healthy foods. The core menu must have at least a 6 item breakfast, lunch and a 3-course dinner.

The Ministry of Defence (MoD) has developed Military Dietary Reference Values (2008) for macro- and micro-nutrients and all advice is centred around the FSA's Eatwell Plate. The Expert Panel on Armed Forces Feeding (EPAFF (UK) produce nutrition and hydration guidance for personnel that emphasizes increasing/maintaining performance, reducing the chance of injury, staying healthy and fit, and quick recovery from activity. All new entry trainees are provided with nutrition education materials. The Armed Forces Nutrition Advisory Service offers expert advice and information on nutrition, diet and military feeding; this is only available to UK military personnel and MOD civil servants.

Operational Ration Packs

All personnel are issued with ORP when operational, but the MoD provides field-catering facilities as soon as possible after deployment. ORPs are available for 10-man groups and individuals. They provide between 3788 and 4996 kcal as approx. 57% carbohydrate, 33% fat, and 10% protein. The menus are varied and provide ethnic alternatives. Core items in each pack include fruit biscuits, brown biscuits, instant coffee, tea, chewing gum, tabasco sauce, and water purification tablets. Matches are also included for lighting the hexiburner cooker that is carried by each soldier.

UK personnel working as part of a North Atlantic Treaty Organization (NATO) force are also covered by the NATO Nutrition Science and Food Standards For Military Operations (http://ftp.rta.nato.int).

Regulation and monitoring

NHS and care settings

The Care and Quality Commission (CQC) (www.cqc.org.uk) are charged with regulating nutrition in care provided by the NHS in England and all care settings, including care homes, must be registered. CQC set standards, hold organizations to account when they fail to meet these standards, and protect service users from malnutrition and dehydration. NHS and care settings regulations vary across the home countries. More information for Scotland can be found at The Scottish Commission for the Regulation of Care (www.carecommission.com), for Northern Ireland at the Regulation and Quality Improvement Authority (RQIA) (www.rqia.org.uk) and in Wales by the Care Standards Inspectorate for Wales (www.cssiw.org.uk).

Schools

The FST standards are mandatory under the School Food Regulations (2007), which are laid down in UK law. They form part of the Ofsted (Office for Standards in Education, Children's Services and Skills) schools inspect framework (www.ofsted.gov.uk).

Prisons

The prison governor has overall responsibility for catering standards, and the kitchens are inspected daily by the prison governor or one of his assistants. The catering manager at each prison is responsible for implementing standards. Her Majesty's Inspectorate of Prisons and the Independent Monitoring Board are responsible for monitoring all aspects of prison life including catering.

The Armed Forces

Each unit commanding officer is ultimately responsible for standards of catering and the catering manager monitors provision of foods and patterns of consumption of high fat foods, salt and sugar. The DFS Quality Assurance team ensures the quality of food supplied and inspects all food premises.

Chapter 11

Popular diets

Popular diets

This section is included to help orientate health-care professionals to diets that their patients may initiate or possibly seek advice about. It does not validate their efficacy. For many, evidence of benefit in the form of a randomized controlled trial is not available. However, the concerns, described below, about potential harm resulting from some diets are based on scientific principles.

- *Atkins diet:* a low carbohydrate, high protein diet for weight reduction (see 📖 Chapter 21, 'Management: dietary aspects', p. 424).
- *Beverley Hills diet:* based on the belief that enzymes are required to break down specific foods and that certain foods provide these enzymes while undigested food in the gastrointestinal tract leads to the gain of fat. There is no scientific evidence to support this hypothesis.
- *Blood Group diet or Eat Right for Your Type:* this diet is based on the idea that blood groups evolved at different times during evolution and that a diet that reflects this period is optimum for health and weight control. There is no evidence that different types of blood group relate to different historical eating patterns or that modifying current dietary intake on the basis of blood groups influences energy balance and thus weight loss.
- *Bristol Approach to Healthy Eating:* developed from the Bristol Diet, these nutritional guidelines are recommended by Penny Brohn Cancer Care for people living with cancer (see 📖 Chapter 24, 'Other dietary approaches to cancer treatment', p. 496).
- *Cabbage Soup diet:* advocates short-term rapid weight loss through consuming large quantities of home-made cabbage soup plus very limited other food. The soup recipe provides little energy but also few other nutrients. Whilst this regime may lead to rapid weight loss through a very low energy intake, nutrient intake is very likely to be inadequate and following the diet is incompatible with established nutritional principles.
- *Detox diets:* recommend strict avoidance of all potential dietary 'toxins', e.g. wheat, dairy, alcohol, caffeine, food colouring, and preservatives, as a means of reducing body weight. Perceived 'natural' foods, e.g. organic produce including fruit, vegetables, and nuts, are usually allowed, although short-term fasting is also recommended on some regimes. No formal studies have been undertaken to evaluate efficacy but it is likely that weight loss associated with this type of diet is due to a reduction in energy intake as a result of the limited foods permitted. The total nutrient intake is likely to be inadequate overall, especially as foods providing protein and calcium are often restricted.
- *Food combining diets:* the rationale is based on the theory that overweight occurs as a result of defective digestion caused by eating the wrong types of food at the same time, e.g. eating protein-rich food and carbohydrate at the same meal. The diet ∴ advises careful separation of these foods so that they are not consumed together. There is no scientific basis for this theory and no evidence that a food combining diet is effective in reducing excess body weight. If weight loss occurs while following the regime, it will be because total energy intake falls below energy expenditure which probably occurs because

the complexity of the rules discourages food intake. Although the diet could be potentially adequate and nutritious, it is complex and time-consuming to follow and does not address long-term changes in eating habits.

- *Gerson diet:* a regime used in treating cancer (see 🕮 Chapter 24, 'Other dietary approaches to cancer treatment', p. 496).
- *Glycaemic index (GI) diet:* uses the sound scientific principle that foods with a low GI (see 🕮 Chapter 5, 'Glycaemic index', p. 77), e.g. apples, lentils, yogurt, are more satiating and have other health benefits compared to high GI foods, e.g. white bread, cornflakes, sugar, and that the former will help followers to limit their total energy intake and thus reduce overweight. Weight loss will only occur if total energy intake is < total energy expenditure rather than by eating low GI foods *per se*, but these are compatible with a well-balanced and varied diet. Systematic review of six studies shows that people following a GI diet lose significantly more weight (1 kg) than those on a control diet.[1]
- *Grapefruit diet:* a low fat, very low energy diet to promote rapid weight loss in 1–2 weeks. Originally described as enzyme-dependent and 'fat-burning' in the 1930s, one recent study has reported greater weight loss associated and reduced insulin resistance with consuming grapefruit three times daily compared with a placebo. Potentially interesting but mechanistic evidence is needed.
- *Hay diet:* see 'Food combining diets', four bullets above.
- *LighterLife:* a commercial weight loss and weight management programme providing a very low calorie diet (~600 kcal/day), transactional analysis and cognitive behavioural therapy techniques which are delivered through foodpacks and a network of counsellors. Peer-reviewed abstracts from the company report successful weight loss after 2 years. Very low calorie diets are associated with health risks, especially if followed for more than 12–16 weeks and their use for >12 weeks is not recommended[2] (see 🕮 Chapter 21, 'Very low calorie diets', p. 426). The high financial cost may preclude use.
- *Meal replacements:* many commercial meal replacements for use in weight loss are available in the form of milk shakes, soups and bars and also as ready-meals or complete food provision. Brand names include Diet Chef, Jenny Craig Programme, Rosemary Conley Solo Slim and Slimfast (see 🕮 Chapter 21, 'Meal replacement/meal provision', p. 427).
- *Macrobiotics:* describes a philosophical approach to life that includes balancing yin and yang elements. The dietary element is based on predominantly vegetarian, high carbohydrate, low fat food with regular consumption of soya and sea vegetables. Although some aspects of the diet follow healthy eating principles, more extreme versions

[1] Thomas D, Elliott EJ, Baur L (2009). Low glycaemic load diets for overweight and obesity. *Cochrane Database of Systematic Reviews* 2007, Issue 3, Art. No. CD005105. DOI: 10.1002/14651858.CD005105.pub2.

[2] NICE (2006) *Obesity guidance on the prevention, identification, assessment and management of overweight and obesity in adults and children*. Available at: 🖱 www.nice.org.uk/CG43.

are nutritionally inadequate and cannot be recommended for health reasons; the low energy and protein density is a particular concern in patients with a poor appetite.

- *Plant diet:* a regime used in treating cancer (see 📖 Chapter 24, 'Other dietary approaches to cancer treatment', p. 496).
- *Protein Power diet:* a weight loss regime based on carbohydrate restriction.
- *South Beach diet:* Originally designed for heart health, this is now regarded as a weight loss diet which is based on an initial very restricted 2-week phase followed by a regime which encompasses the principles of the GI diet described above. While the weight loss phase is compatible with healthy guidelines, the nutritional information presented with the diet has been criticized.
- *Sugar Busters* advocates avoidance of refined sugar and processed grains and eating high fibre vegetables and wholegrains to promote weight loss. Although avoiding refined carbohydrate is compatible with a healthy, weight reducing diet, avoiding sugar on its own is insufficient to ensure long-term successful weight loss.
- *Weight Watchers:* a commercial structured weight loss programme delivered either through local meetings or online. Energy restriction is achieved by using calorie-based points to construct a flexible intake which is compatible with health eating guidelines and education about food and exercise. Successful weight loss has been reported although the perceived complexity may compromise adherence in some people.
- *Zone diet* is based on the theory that an optimum diet should comprise a fixed proportion of macronutrients at each meal: carbohydrate 40%, fat 30% and protein 30%. These values differ from values currently recommended for healthy adults (50, 35, and 15%, respectively) and there is no evidence that either health or weight loss are optimized by the proposed quantities.

Chapter 12

Diet before and during pregnancy

Pre- and peri-conceptional nutrition in women

Why is nutrition important at preconception?

A mother's nutritional status is critical prior to conception, (preconception is 3 months before conception), and immediately afterwards, (peri-conception is 2–3 months after conception). The foetus is most vulnerable to nutritional deficiencies in the first trimester of pregnancy, often before a woman realizes that she is pregnant.

There is evidence that poor maternal nutrition has both immediate (e.g. low birth weight) and long-term consequences. The so-called 'foetal origins' or 'Barker' hypothesis proposes that foetal growth plays a major role in determining the risk of some dietary related non-communicable disease, e.g. cardiovascular disease and type 2 diabetes in adulthood.

Dietary advice for preconception

What dietary changes can the mother make to increase the likelihood of conceiving and giving birth to a healthy infant?

Eat a varied diet. Refer to 'The Eatwell Plate' in 📖 Chapter 2, p. 27. The main points are:

- include 5 portions of fruit and vegetables a day;
- eat a variety of different foods from all food groups;
- restrict foods containing too much saturated fat and sugar.

Folic acid and preconception

- Take folic acid supplements to protect against neural tube defects (NTDs). In the UK the DH[1] recommends:
  - *to prevent first occurrence of NTD:* 400 µg during preconception and until 12th week of pregnancy (on prescription or over counter);
  - *to prevent recurrence of NTD:* 5 mg during preconception and until the 12th week of pregnancy (on prescription only).
- Foods rich in folic acid should be chosen (see Box 12.1).

Box 12.1 Foods rich in folic acid

- *Rich sources:* >100 µg per serving: Brussels sprouts, kale, spinach
- *Good sources:* 50–100 µg per serving: fortified bread and breakfast cereals, broccoli, cabbage, cauliflower, chickpeas, green beans, iceberg, kidney, lettuce, peas, spring greens
- *Moderate sources:* 15–15 µg per serving: potatoes, most other vegetables, most fruits, most nuts, brown rice, wholegrain pasta, oats, bran, some breakfast cereals, cheese, yoghurt, milk, eggs, salmon, beef, game

[1] SACN (2009) *Folic acid and colorectal cancer risk: Review of recommendation for mandatory folic acid fortification*. SACN, London.

Foods to avoid at preconception

Alcohol

Reduce alcohol consumption and ideally exclude altogether.

- Alcohol intake may be associated with decreased fertility and can affect the growing foetus.
- UK Department of Health (DH) recommends ≤1–2 units, once or twice a week.
- Binge drinking in particular is not recommended. Advise women not to get drunk!

Vitamin A supplements and liver

Avoid excessive intake of retinol/vitamin A (β-carotene is not toxic).

- Avoid vitamin A supplements or fish liver oils, liver, liver pâté, or sausage as retinol is teratogenic at extreme intakes (8000–10000 μg).
- Avoid drugs that contain vitamin A or its analogues, such as cystic acne medications (isotretinoin; treinoin).

Certain fish

- Women can eat most types of fish, whilst they are trying to conceive; however, the DH (England) advice for pregnant women should be followed as a precautionary measure (advice for other home countries may vary):
  - ♀ should avoid eating shark, swordfish and marlin because of the mercury content, which can affect neural development of foetus;
  - ♀ should limit the amount of tuna they eat (high in mercury) to no more than 2 fresh tuna steaks a week or 4 medium size cans (drained weight of 140 g);
  - ♀ should not eat more than 2 portions of certain fish a week, including fresh (but not canned) tuna. See Box 12.2 for these.

Box 12.2 Types of fish to limit during pregnancy

The following should be limited to 2 portions/week (1 portion = 140 g cooked weight) because of their levels of dioxins and PCBs:

- oily fish, including mackerel, sardines, salmon trout and fresh tuna;
- sea bream, sea bass, turbot, halibut, rock salmon (also known as dogfish, flake, huss, rig, or rock eel);
- brown crabmeat.

Food safety

Women should be encouraged to follow the food safety advice for pregnant women (see this 📖 Chapter, 'Food safety in pregnancy', p. 226 and 'Maternal weight gain', p. 228) as a precautionary measure for when conception occurs.

Peanuts and preconception

Women trying to conceive are no longer advised to avoid peanuts due to lack of an evidence base that this reduces likelihood of infant peanut allergy. Obviously, ♀ who have peanut allergy should still avoid them! Previously it was recommended to avoid eating peanuts in pregnancy to reduce risk of allergy. Advice in UK changed in 2009.

Healthy weight for conception

- Achieve and maintain ideal weight at preconception (BMI 18.5–24.9 kg/m^2).
- Weight needs to be stabilized 3 months before attempting conception.
- Low body fat content of <22% of body weight can prevent ovulation (average body fat content of post-pubertal women is 28%).
- Obesity (BMI ≥30) can inhibit ovulation due to associated changes in insulin activity and its effect on hormone activity.
- Obesity at conception can influence the *pregnancy* (high blood pressure, impaired blood sugar metabolism, gestational diabetes; pre-eclampsia), *delivery* (preterm delivery; prolonged labour; unplanned Caesarean), and *infant's health* (stillborn foetus; difficulty initiating and sustaining breastfeeding).
- Obese and overweight women should be advised to lose weight before conception (NICE guidance 2010[2]).
- Underweight (BMI <18.5) at conception can increase the risk of pre-term delivery and of delivering a low-birth weight infant.

[2] NICE (2010) *Weight management before, during and after pregnancy*. NICE public health guidance 27.

Dietary reference values and dietary guidelines during pregnancy

Dietary recommendations are the same as for a normal healthy diet (see 📖 'The Eatwell Plate' in Chapter 2, p. 27) except for additional requirements for 6 nutrients of protein, energy, folic acid, vitamins A, C and D (see Table 12.1).

Caffeine and pregnancy

- May contribute to low birth weight by increasing foetal heart rate and ↑ risk of miscarriage.
- Pregnant women do not need to completely cut out caffeine, but limit intake to <200 mg/day (this is 100 mg less than previous UK recommendation before 2008).
- Tea, coffee, cocoa, and cola-type drinks are advised in moderation (equivalent to <200 mg/day). Suggest decaffeinated tea and coffee or other alternatives, such as fruit tea, fruit juice, or water. Tea and coffee also reduce iron absorption.

The following contain ~200 mg of caffeine:

- 1 mug of brewed or filter coffee (140 mg each);
- 2 mugs of instant coffee (100 mg each);
- 2 mugs of tea (75 mg each);
- 5 cans of cola (up to 40 mg each);
- 2 cans of 'energy drink' (up to 80 mg each);
- 4 (50g) bars of plain chocolate (up to 50 mg each). Caffeine in milk chocolate is about half that of plain chocolate.

Alcohol in pregnancy

- Current optimal advice is abstinence in pregnancy, especially important in the first trimester; however, occasional drinking of small quantities, i.e. ≤1–2 units, once or twice a week but ≤2 units at any 'sitting' (see 📖 Chapter 9, 'Alcohol' (p. 186), Table 9.1) is unlikely to harm the foetus.[1]
- Excessive binge drinking is most dangerous and can have teratogenic effects leading to foetal alcohol syndrome which affects 1–2/1000 births/year. Risk is elevated in women drinking >8 units/day. Symptoms in the infant are growth retardation, craniofacial, and CNS defects, cardiac and genitourinary abnormalities. Advise women not to get drunk!

[1] Deparment of Health (2009). *The pregnancy book*. Department of Health, London. Available at 🖱 www.dh.gov.uk.

Eating fish in pregnancy

- Women can eat most types of fish whilst they are pregnant, however the DH advice has become more complex recently:
  - ♀ should avoid eating shark, swordfish and marlin because of the mercury content which can affect neural development of foetus.
  - ♀ should limit the amount of tuna they eat (high in mercury) to no more than 2 fresh tuna steaks a week or 4 medium size cans (drained weight of 140 g).
  - ♀ should not eat more than 2 portions of certain fish (1 portion = 140 g cooked weight) a week, including fresh (but not canned) tuna. (see Box 12.2).

Table 12.1 RNI for pregnant women

Nutrient	Daily RNI (pre-pregnancy)	↑ in pregnancy
Energy (kcal)	1940–110	+200 (3rd trimester)
Folic acid (μg)	200	+400 (1st trimester) +100 (2nd and 3rd trimesters)
Protein (g)	51	+6
Vitamin C (mg)	40	+10 (3rd trimester)
Vitamin D (μg)	0 (assumed gained from sun exposure)	+10
Vitamin A (μg)	600	+100

NB. No increase recommended for intake of calcium and iron as evidence insufficient that this is needed above RNI. See 🕮 Appendix 6 (p. 780) for RNI for adult women.

Vitamin and mineral supplements in pregnancy

Women should try and obtain nutrients from a balanced diet (see 'The Eatwell Plate' in 📖 Chapter 2, p. 27) and need to be advised against taking high dose multivitamin and mineral supplement (see Table 12.2).

Vitamin A (and liver products)

Vitamin A supplements and fish liver oils can quickly reach toxic levels, and may have teratogenic effects. Women in the UK should not use vitamin A supplements or fish liver oils. Liver, liver pâté, and other foods containing liver should not be eaten as they are a very rich source of vitamin A. However, in areas of the world where vitamin A deficiency is prevalent, supplementation may be beneficial for pregnant women.

Folic acid

Folic acid (400 µg/day) is the only supplement recommended for 'blanket' use by women until the 12th week of pregnancy (see this 📖 Chapter, 'Pre- and peri-conceptional nutrition in women', p. 218).

Iron tablets

In the UK, iron supplements are advised only if there is evidence of iron deficiency anaemia (see 📖 Chapter 6, 'Iron', p. 128). Iron stores should be verified preconceptually and in pregnancy. Iron supplements can cause constipation and other GI changes, and may interfere with zinc absorption.

Vitamin D supplements

The DH recommends vitamin D supplements (10 µg/day) during pregnancy and breastfeeding in addition to sunlight between April and September (see 📖 Chapter 6, 'Vitamin D (calciferols)', p. 100) and that only those on a restricted diet need extra vitamin D. Some Asian women could be at risk of vitamin D deficiency due to insufficient skin exposure (see 📖 Chapter 16, 'Minority ethnic communities', p. 306) → neonatal hypocalcaemia and rickets, ∴ may need extra vitamin D supplements.

Table 12.2 Nutritional supplements in pregnancy

Supplement	Recommendation
Vitamin A	Not advised in well nourished populations
Folic acid	400 µg/day for all women until the 12th week of pregnancy
Iron	Only if there is evidence of iron deficiency anaemia
Vitamin D	10 µg/day during pregnancy and breastfeeding

Food safety in pregnancy

Box 12.3 Food items to avoid in pregnancy

Besides following normal safe food hygiene practices, pregnant women should be advised to avoid additional practices that have been specifically linked to micro-organisms that can lead to foetal malformations.

Avoiding salmonellosis

In severe cases can cause premature labour and miscarriage.

- Avoid raw or undercooked eggs due to salmonella risk. White and yolk should be hard boiled. Raw egg may be found in home-made mayonnaise, ice-cream, mousse. Mayonnaise and salad cream made with cooked eggs is fine
- Avoid raw/partially cooked meat, e.g. poultry, sausages and burgers; they should be cooked thoroughly until brown on the inside.

Avoiding listeriosis

Caused by *Listeria monocytogenes*. Rare, but even mild infection can lead to miscarriage, still birth, or ill newborn. Women should avoid:

- All types of pâté (including vegetable)
- Mould ripened soft cheese, e.g. brie, camembert, chevre
- Blue veined cheese, e.g. stilton, roquefort, and other unpasteurized cheese
- Unpasteurized milk, including cow, goat, and sheep's, and associated milk products
- Eating uncooked or undercooked ready-prepared meals or leftovers >24 h old

Avoiding toxoplasmosis

Caused by *Toxoplasma gondii*—the mother will have flu symptoms and it can cause blindness and mental retardation in the infant.

- Avoid cats as they can be carriers—wear gloves when gardening or changing cat litter and wash hands afterwards.
- Cook poultry and meat thoroughly.
- Wash salads, fruit, and vegetables to remove all soil.
- Thoroughly reheat ready prepared meals and leftovers.

Reducing likelihood of developing peanut allergy

Pregnant women are no longer advised to avoid peanuts due to lack of an evidence base that this reduces likelihood of infant peanut allergy. Obviously, ♀ who have peanut allergy should still avoid them! Previously, it was recommended to avoid eating peanuts in pregnancy to reduce risk of allergy. Advice in UK changed in 2009.

Avoiding food poisoning

Avoid raw shellfish. Cooked shellfish including prawns are fine.

❗ Advise caution about herbal supplements, as these are not generally evaluated for safety in pregnancy.

► Honey is fine for pregnant and lactating women to eat, but not for infants <12 months.

Maternal weight gain

How much weight should a woman gain during pregnancy? Weight gained in pregnancy is a combination of maternal/foetal tissues and fluid, as well as maternal fat stores. Rate of weight gain is usually not constant: around 2 kg (5 lbs) are gained in the first trimester and the rest throughout the second and third trimesters at a rate of ~ 0.4 kg (1 lb) per week.

To account for these ↑ energy demands, the DH (England) (see Table 12.1) makes a blanket recommendation for women to consume an extra 200 kcal/day in the last trimester, but the best advice is to encourage ♀ to eat to appetite in pregnancy and monitor weight gain within the appropriate ranges.

An average weight gain of 10–12.5 kg (22–28 lbs) is recommended for women of normal BMI in the UK, but a higher average weight gain of 11.5–16 kg (25–35 lbs) is seen as acceptable in the US.[1]

❗ Both too little and too much weight gain can adversely affect the foetus.

- *Too much maternal weight gain during pregnancy* can → post-partum maternal obesity; possibility of caesarean; infant macrosomia; and ↑ risk of gestational diabetes. See 'The Eatwell Plate' in 📖 Chapter 2, p. 27 for healthier eating to prevent weight gain.
- *Too little maternal weight gain* can → low birth weight baby with subsequent effects on long-term health (see 📖 this chapter, 'Pre- and peri-conceptional nutrition', p. 218).

Weight gain in overweight and obese women

Women who are overweight or obese should not attempt to lose weight during pregnancy. NICE (2009)[2] makes no recommendation regarding acceptable weight gain for women who were overweight or obese pre-pregnancy, stating insufficient evidence. However, the US Institute of Medicine (IOM)[1] recommends that, based on pre-pregnancy BMI, overweight women should limit weight gain to 7–11.5 kg (15–25 lbs) and obese women should limit weight gain to 5–9 kg (11–20 lbs).

Overweight and obese pregnant women need regular monitoring as there is ↑ risk of pre-eclampsia, gestational diabetes mellitus (DM) and hypertension (HT); the risk ↑ with BMI. At birth there is ↑ likelihood of caesarean section, post-operative complications, low apgar score, excessive birth weight of newborn (macrosomia), ↑ perinatal mortality (3-fold), and neural tube defects (NTDs).

[1] IOM (Institute of Medicine). (2009). *Weight Gain During Pregnancy: Re-examining the Guidelines.* The National Academies Press, Washington, DC. Available at: 🖱 www.iom.edu

[2] NICE (2010). *Weight management before, during and after pregnancy.* NICE public health guidance 27.

Weight gain with multiple pregnancies

Multiple births account for 1 in 6 of all births in the UK. Women carrying twins (or more!) will gain even more weight than women carrying one foetus. In the absence of UK guidelines, the IOM[1] recommendations are used that were revised in 2009. They advise that normal weight women carrying twins should gain 17–25 kg (37–54 lbs), overweight ♀ should gain 14–23 kg (31–20 lbs), and obese ♀ should gain 11–19 kg (25–42 lbs) during the pregnancy. A healthy weight gain is particularly important in multiple pregnancies as they carry a higher risk of premature birth and low birth weight.

Dietary problems in pregnancy

Pregnancy sickness (also known as morning sickness)

During the first trimester, ~70% of women have pregnancy sickness (nausea and vomiting) as the woman adjusts to higher hormone levels, especially human chorionic gonadotrophin and high oestrogen levels. Although often referred to as 'morning sickness' vomiting can occur at any time of the day: it varies from slight nausea to frequent and severe vomiting. Most cases are mild, but it impacts on the pregnant woman's sense of well-being and daily activities. *Hyperemesis gravidarum* is the most severe form and is defined as persistent nausea and vomiting leading to dehydration, ketonuria, electrolyte imbalance, and weight loss greater than 5% of pre-pregnancy weight.

Advise[1]

- Frequent small meals and snacks every 2 h, avoiding large meals.
- High carbohydrates (CHO)/low fat foods are best tolerated, e.g. toast, dry biscuits, crackers, low sugar breakfast cereals.
- Avoid smells and foods that exacerbate nausea, e.g. high fat foods. However, these foods will depend on each woman.
- Taking food and drinks separately can help ↓ nausea in some women.
- Encourage plenty of fluid, especially as water and other sugar-free fluids, as dehydration may occur in extreme cases. Recommend at least 35 ml/kg body weight/daily; equivalent to 9 mugs of fluid in a 65-kg woman (1 mug = 250 ml).
- Avoid foods or smells that trigger symptoms. Eat cold meals, rather than hot ones because they do not smell as strongly, which may provoke nausea.
- Eat plain biscuits before getting up.
- Avoid drinks that are cold, tart (sharp), or sweet.
- Some evidence that ginger supplements may help reduce the symptoms of nausea and vomiting in some pregnant women. Ginger products are unlicensed in UK, so advise purchase from a reputable source, such as a pharmacy or supermarket.
- Wear comfortable clothes without tight waistbands, which can sometimes cause discomfort.
- Take time to rest and relax; take fresh air.
- Reassure women that most cases resolve spontaneously in the first 16–20 weeks of pregnancy.
- When symptoms are persistent, severe, and prevent daily activities, drug treatment should be considered.

Food aversions and cravings

Aversions are relatively common especially for tea, coffee, fried food, and eggs. Food cravings can be strong but depend on the individual. There are no nutritional implications as long as craving does not involve eating a lot of energy-dense foods that result in excessive weight gain.

[1] Further information is available at www.nhs.uk/Conditions/Morning-sickness.

Pica

Pica is the persistent craving for non-food substances, ranging from coal, clay, candles, matchboxes, to soil. Pica can be harmful if the item craved and eaten is toxic or eaten in large enough quantities to have an impact on nutritional status. Eating soil could carry the risk of toxoplasmosis (see 🕮 this Chapter, 'Food Safety in Pregnancy', p. 226). Evidence for a physiological basis of need is inconclusive. Pica is often associated with iron deficiency but it is uncertain whether iron deficiency causes pica or conversely whether pica causes iron deficiency because of its effect on ↓ iron absorption.

Iron deficiency anaemia in pregnancy

Women with diets poor in iron prior to pregnancy and a history of anaemia will need haemoglobin and ferritin status verifying to assess whether supplements are required. Anaemia is most likely to affect women on a low income (see 🕮 Chapter 16, 'Eating on a low income', p. 318), those with low BMI, or vegetarians with an unbalanced diet (see 🕮 Chapter 16, 'Vegetarians', p. 312). In the UK, iron supplements are advised only if there is evidence of iron deficiency anaemia (see diagnosis of anaemia in 🕮 Chapter 6, 'Iron', p. 128).

However, care should be taken not to 'blanket' prescribe iron supplements (can result in nausea and constipation), as in later pregnancy many women experience haemodilution and ∴ physiological changes may resemble iron deficiency (↓ haemoglobin and ↓ ferritin). See 🕮 Chapter 6, 'Iron', p 128, for good dietary sources of iron.

Gestational diabetes

Estimated prevalence is 3–5% of pregnancies in the UK. Abnormal glucose intolerance occurs in pregnancy and usually disappears after birth, although there is evidence that it is a marker for development of type 2 diabetes in later life. Diagnosis is made at fasting blood glucose >7 mmol/l (see Table 22.1 for further information on diagnosis). Women who are obese/overweight, aged ≥30 y, and have a family history of type 2 diabetes are at greater risk of developing gestational diabetes, increased risk of macrosomia at birth, and increased likelihood of Caesarean.

A meta-analysis of cohort studies[2] reported a 7.5 fold ↑ risk of developing type 2 diabetes for women who had been diagnosed with gestational diabetes. See 🕮 Chapter 22, 'Gestational Diabetes', p. 460 for advice on management.

Constipation in pregnancy

35–40% of women suffer during pregnancy, as peristalsis is slower.

Encouraging fresh and dried fruit and vegetables for pectins, and wholemeal bread and breakfast cereals for cereal fibre will relieve symptoms, and plenty of fluid, preferably as water, should be taken. Faecal bulking agents may help.

[2] Bellamy, L., Casas, J.P., Hingorani, A.D., *et al.* (2009). Type 2 diabetes mellitus after gestational diabetes: a systematic review and meta-analysis. *Lancet*, **373**, 1773–9.

Women may intentionally restrict their fluid intake to reduce frequency of micturition; this could be a factor in them becoming constipated. See 🕮 Chapter 26, 'Constipation' (p. 602).

Heartburn in pregnancy

Heartburn is common and 30–50% of pregnant women experience symptoms. This can occur at any stage of pregnancy, but usually in the 3rd trimester.

Suggestions for relief

- Small, frequent meals.
- Eat earlier in the evening and avoid late night meals.
- Chew food thoroughly and slowly.
- Take fluids between meals, not at mealtimes.
- Dairy foods may relieve symptoms.
- Avoid spicy and acidic foods that may irritate gastrointestinal (GI) mucosa. Food causing symptoms varies a lot in different women; examples include chilli, vinegar, pepper, acidic fruit juices.
- Avoid foods that relax oesophageal muscles before bedtime, e.g. chocolate, fatty foods, alcohol, and mint.
- Sleep propped up with cushions.
- Avoid bending after eating.

Vulnerable groups in pregnancy

Adolescents and pregnancy

Pregnancy in adolescence increases risk to the:

- *Foetus* of low birth weight, perinatal mortality, and premature delivery;
- *Mother* suffering anaemia, difficult labour, and hypertension.

As adolescents are still growing, optimal weight gain is unknown, but it is likely to be higher than for adult women (see 🕮 this Chapter, 'Maternal weight gain', p. 228). They are less likely to eat healthily and have higher RNIs for calcium and iron than women >18 years ∴ they are less likely to meet requirements for calcium and iron (see 🕮 Chapter 14, 'Nutrient deficiencies in children', p. 286). Iron deficiency anaemia can result in low birth weight and preterm delivery.

Social problems may have an influence and will compound pregnancy outcome, including:

- Reducing energy intake to try and hide pregnancy;
- Low income;
- Smoking;
- Alcohol consumption;
- Substance abuse;
- Previous dieting leading to low nutrient stores;
- Less knowledge and skills to eat a healthy diet.

UK government set targets to reduce the number of under teenage pregnancies with a consequent reduction of 13.3% between 1998 and 2008.[1]

Vegetarians

Being vegetarian should pose no problem in pregnancy, if the woman is well informed and eating a balanced lacto-ovo and lacto-vegetarian diet. Pregnant vegan, fruitarian, and macrobiotic women should be seen by a dietitian to assess overall nutrient adequacy of their diets. They may require supplementation of vitamin B_{12}, iron, vitamin D, or calcium (if <600 mg/day consumed). Some fortified soya milks contain these nutrients. (see 🕮 Chapter 16, 'Vegetarians', p. 312).

Asian vegetarian women could be at risk of vitamin D deficiency if insufficient skin exposure → neonatal hypocalcaemia and rickets ∴ may need additional Vitamin D supplements (see 🕮 Chapter 16, 'Minority ethnic communities', p. 306). Pregnant vegetarian adolescents are at particular risk of inadequate diet if they are the only 'veggie' in the house, as they may tend to eat the same as the rest of the family except 'remove' the protein aspect of the meal or replace it with cheese, ready-prepared vegetarian sausages, and burgers.

Low income and pregnancy

Although it is difficult to generalize, UK women on low incomes may find it harder to achieve an adequate diet (see 'The Eatwell Plate' in 🕮 Chapter 2, p. 27 and 🕮 Chapter 16, 'Eating on a low income', p. 318). Key nutrients at risk of low intakes are: zinc and iron, and vitamins A, C, and E and

[1] Department of Health (2010). *Teenage Pregnancy Strategy: Beyond 2010*. DoH, London.

essential fatty acids (EFAs) needed for foetal neural and vascular system development. EFAs are found in green vegetables, oily fish (e.g. tuna, sardines, mackerel, salmon, herring, pilchards, trout, and kippers), and certain vegetable oils (e.g. corn, sunflower, and soya oils). Cheaper blended vegetable oils and margarine are often consumed but they contain less EFAs.

Healthy start scheme

In the UK, the welfare food scheme has been replaced by the healthy start scheme. The healthy start scheme allows beneficiaries to exchange tokens for fresh fruit and vegetables through general retail (see 📖 Chapter 13, 'Healthy start scheme', p. 256). Further information on Healthy start see 🖱 www.healthystart.nhs.uk.

Closely-spaced pregnancies

Women having closely spaced pregnancies may have low nutrient stores at conception and in early pregnancy, so taking a dietary history would be useful to assess previous and current diet for nutrient adequacy (including iron status).

Diabetic women

Regular glucose monitoring and good compliance will result in the same outcome as for non-diabetic mothers. However, poor control can ↑ risk of pre-eclampsia, ↑ foetal problems, and ↑ infant mortality.

Useful websites

DH, *The pregnancy book 2009*; www.dh.gov.uk
www.eatwell.gov.uk/
www.healthystart.nhs.uk

Chapter 13

Infants and preschool children

Infant growth and development

Dietary recommendations for infants and preschool children

In the first year of life birth weight increases by 300%, doubling in the first 4–6 months, and height increases by 50%. Growth then slows down. This rapid growth involves tissue and organ maturation that mean that energy and nutrient requirements are high relative to body size, especially during the first year (see Table 13.1). In the first 3 months of life, 35% of energy requirements are utilized for growth, but by 12 months age, this has dropped to 3%. High protein synthesis rates in the newborn contribute to high energy and protein requirements.

Children <5 years need a diet that is higher in fat and lower in fibre than that presented in the 'The Eatwell Plate' model (see 'The Eatwell Plate' in 📖 Chapter 2, 'Food-based dietary guidelines', p. 27) as they need fat for growth and central nervous system (CNS) development. However, by the age of 5 they can follow the dietary guidelines presented. If children are growing normally then >2 years, parents can gradually start introducing low fat/high fibre choices towards 'The Eatwell Plate' recommendations at age 5.

Growth reference charts

Monitoring children's' growth is essential to identify any faltering growth. Length/height, weight and occipito-frontal head circumference (OFC) should be plotted on a growth reference curve. An infant's growth should follow the direction of the growth curves. Serial measurements are necessary to determine adequacy of growth as a one-off measurement is only a reflection of size. The chart (Box 13.1) can be a useful tool for communicating with parents so that they understand the importance of monitoring growth. Parents with naturally short children will need reassuring that s/he is growing well if progressing in parallel with the same centile throughout infancy and childhood.

Box 13.1 Calculating adult height potential

Parental height plays a role in determining eventual height and mid-parental height (target centile range) is useful to estimate the genetic height potential of a child. To calculate adult height potential:
(a) = father's height
(b) = mother's height
(c) = sum of (a) + (b)
(d) = (c) ÷2
(e) = (d) –7cm if girl or +7cm if boy (mid parental height)
(f) = (e) ±8.5 cm (target centile range)

Where growth is unimpaired, adult height can be estimated in children age 2–4 years from the child's current height centile, using the adult height predictor for both boys and girls, in the 0–4 years UK-WHO growth charts (see 📖 Appendix 2, p. 764).

Table 13.1 RNI for infants and preschool children*

Nutrient	0–3 months	4–6 months	7–9 months	10–12 months	1–3 years	4–6 years
Energy (kcal)						
♂	545	690	825	920	1230	1715
♀	515	645	765	865	1165	1545
Energy (kcal/kg/day)	100–115	95	95	95	95	90
Protein (g)	12.5	12.7	13.7	14.9	14.5	19.7
Protein (g/kg/day)	2.1	1.6	1.5	1.5	1.1	1.1
Fluid (ml/kg)	150	130	120	110	95	85
Vitamin C (mg)	25	25	25	25	30	30
Vitamin A (μg)	350	350	350	350	400	400
Folic acid (μg)	50	50	50	50	70	100
Thiamine (mg)	0.2	0.2	0.2	0.3	0.5	0.7
Riboflavin (mg)	0.4	0.4	0.4	0.4	0.6	0.8
Niacin (mg)	3	3	4	5	8	11
Vitamin B_{12} (μg)	0.3	0.3	0.4	0.4	0.5	0.8
Iron (mg)	1.7	4.3	7.8	7.8	6.9	6.1
Calcium (mg)	525	525	525	525	350	450
Phosphorus (mg)	400	400	400	400	270	350
Magnesium (mg)	55	60	75	80	85	120
Zinc (mg)	4.0	4.0	5.0	5.0	5.0	6.5
Selenium (μg)	10	13	10	10	15	20
Copper (mg)	0.2	0.3	0.3	0.3	0.4	0.6

* Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. HMSO, London.

If there are any concerns that the child may be under- or over-weight, it is recommended that from the age of 2 years BMI is calculated [weight (kg)/height (m^2)], which should be adjusted for age and gender using BMI centile charts (see 🕮 Appendix 2, p. 751). Alternatively the BMI centile can be estimated using the weight-height to BMI conversion chart in the new 0–4-year UK-WHO charts (see 🕮 Appendix 2, p. 751).

Which growth charts should be used?

In April 2006, the World Health Organization (WHO) published new Child Growth Standards for infants and children up to the age of 5 years. They are based on the growth of around 8500 healthy, non-deprived children from six different countries (USA, Norway, India, Ghana, Brazil, Oman), who were breastfed exclusively for the first 4 months, weaned by 6 months, by healthy mothers who did not smoke.[1] It is believed that all charts should be based on breastfed infants, as this is the biological norm and all infants should be compared to this whatever their ethnic origin and however they are fed in infancy. This standard was adopted by the UK for children under 4 years and used to construct the UK-WHO charts[2] (🔊 www.growthcharts.rcpch.ac.uk).

The existing UK-90 growth reference charts were constructed using measurements from a large number of British children at different ages, collected in the late 1980s and were the main charts in use until 2009.[3] Because the WHO charts do not include preterm data, the UK 1990 data have been used to make the birth section of the UK-WHO charts and new low birth weight charts, as well as charts for use after the age of 4 years. They are a description of typical, but not necessarily healthy, growth in UK children from 1980–90.

All charts have 9 reference centiles of 0.4th, 2nd, 9th, 25th, 50th, 75th, 91st, 98th, and 99.6th that mean, for example:

- 98th centile curve, below which 98% of UK children lie (2 in 100 children will be as tall/heavy as this);
- 50th centile curve, below which 50% of UK children lie (average weight and height for a child of that age);
- 2nd centile curve, below which only 2% of UK children lie (2 in 100 children will be as small/light as this).

These are available from Harlow Healthcare (🔊 http://www.health-forallchildren.co.uk).

New UK-WHO growth charts are to be used in all children born in the UK from May 2009, using appropriate girls' or boys' charts:

- *Low birth weight:* 23 weeks gestation to 2 years (UK90 data).
- *Birth to 1 year* (WHO data + birth UK90 data).
- *1–4 years* (WHO data).
- *4–18 years* (UK90 data).

There is no need to re-plot for children born before May 2009. Six separate charts covering birth to 4 years are included in the UK Personal Child Health Record (PCHR) issued to each newborn. It is anticipated that charts for children over 4 years will be produced to be added to the PCHR. (See growth charts in 🕮 Appendix 2, p. 764).

[1] De Onis M, Garza C, Victora CG, *et al.* (2004) The WHO Multicentre Growth Reference Study: planning, study design, and methodology. *Food Nutr. Bull.*, **25**, S15–S26.
[2] Department of Health (2007). *Application of the WHO Growth Standards in the UK*. Department of Health, London. Available at: 🔊 http://www.sacn.gov.uk/.
[3] Wright, C. M., Booth, I.W., Buckler, J.M., *et al.* (2002). Growth reference charts for use in the United Kingdom. *Arch, Dis, Child*, **86**, 11–14.

How often should infants be measured?

As part of the birth assessment, all babies should be weighed and OFC measured and plotted, and these measurements repeated at the 6–8 week check for healthy babies (sick babies may need more frequent weighing).[4,5] Babies and young children can be further opportunistically weighed at immunization (8, 12, and 16 weeks, and 1 year age) and surveillance contacts. It has recently been recommended that as part of the DH Healthy Child Programme, 2-year review (2009)[6] both height and weight should be measured to calculate BMI. Previously it was not considered routine to measure length/height in healthy, term children until school entry at around 5 years of age.[4] Length, however, should be measured and plotted in any child whose health, growth or feeding pattern is causing concern.[4]

Measuring too frequently may cause parental anxiety and it is recommended that babies should be weighed no more than monthly <6 months, once every 2 months at 6–12 months, and quarterly >1 year age.

[4] Hall, D.M.B. and Elliman, D. (ed). (2006). *Health for all children*, 4th edn (revised). Oxford University Press, Oxford.
[5] NICE (2006). *Routine postnatal care of women and their babies*. NICE clinical guideline 37.
[6] DH (2009). *Healthy Child Programme—The two year review*. Available at: www.dh.gov.uk/publications.

Breast versus bottle feeding

Breastmilk is the best choice for infant feeding for many reasons (see Box 13.2). Infant formulae have a different composition to breastmilk and do not provide all the same benefits, particularly the immunological active components, nor the same nutritional profile and bioavailability.

The composition of breastmilk is not homogeneous: colostrum is produced 1–3 days postpartum, eventually becoming mature milk after 3 weeks. The immunological factors are not only present in colostrum produced during the first few days of lactation, but continue throughout breastfeeding.

Box 13.2 Protective factors in breastmilk

- *Immunological active components:* lactoferrin; cytokines; T- and B-lymphocytes; neutrophils; macrophages; immunoglobulins; lysozymes; growth factors; thyroxin; antiviral lipids; antiprotozoan factors; and bifidus factor (promotes growth of protective *Lactobacillus bifidus* in infant's gastrointestinal (GI) tract)
- *Essential long chain fatty acids (LCP's):* amino acids (AA) and docosahexaenoic acid (DHA) important for cell membrane structure, especially CNS and retina development
- *Structured fats* (palmitic acid in psn 2 on TG) result in better fat and calcium absorption, producing softer stools, less constipation, and improved bone development
- *Proteins:* predominantly α-lactalbumin (rich source of essential amino acids) and free amino acids.
- *Nucleotides:* supports development and maturation of the gut and immune system.
- *Oligosaccharides (prebiotics):* breastmilk contains >100 different oligosaccharides that help normal brain development and produces softer stools, reducing constipation

Benefits of breastfeeding

For the mother

- Encourages bonding between mother and infant.
- Helps women lose excess weight gained during pregnancy.
- Breastfeeding stimulates uterine contractions that help return the uterus to normal size.
- Exclusive breastfeeding suppresses ovulation, helping iron stores return to normal.
- Breast milk is free, except that the mother needs extra nourishment (see 📖 this chapter 'Dietary recommendations for lactation', p. 252).
- Convenience; no preparation required.
- Reduces mother's risk of developing premenopausal breast cancer.

For the infant

- Breastmilk offers complete nutrition for the first 6 months and high bioavailability of nutrients.
- Infants are less likely to experience GI infections, as there is no need for access to clean water, which can be a problem in low-income countries in particular (may also be due to protective factors).
- Prevention of other infectious diseases, especially respiratory, ear and urinary tract infections (greatest impact is for infants exclusively breastfed for first 6 months).
- Breastfed babies are less likely to be obese in later childhood.

Potential obstacles to breastfeeding

- Frequent myth of 'not enough milk': usually results from incorrect breastfeeding technique (see 🕮 this chapter 'Promoting and establishing breastfeeding', p. 246). This is a common reason for women stopping breastfeeding.
- Freedom of the mother: she can feel exhausted as she takes complete responsibility for feeding ∴ she will need support of others with housework, especially in the first few weeks.
- Mothers may be concerned that she cannot see how much milk the baby is taking.
- Engorged breasts and sore nipples can discourage some mothers; need to make sure that the right position is being used for feeding and latching on.
- High stress levels: mother's mental state will affect the letdown reflex; anxiety → oxytocin ↓. Encourage her to rest more and relax when breastfeeding.
- Glamorous image of infant formula portraying healthy, beautiful babies via advertising.
- Social taboo of breastfeeding in public in some cultures, UK included.
- Lack of public facilities for breastfeeding, especially needed in colder months.
- Lack of employment legislation supporting breastfeeding mothers in some countries. NB. Mothers are entitled to express breastmilk at work in the UK.
- May be perceived as offensive by some women, their partners, and older children.
- Breastfeeding of boys may be encouraged more in some cultures. Need to reinforce that breastfeeding is best for girls and boys.

Contraindications to breastfeeding

- HIV+ women: by vertical transmission from mother to infant, breastfeeding increases risk of transmission by up to 20%. The WHO states that health authorities should decide on the strategy that is most likely to give infants the greatest chance of HIV-free survival, i.e. whether health services will principally counsel and support mothers known to be HIV infected to either (i) breastfeed and receive antiretroviral (ARV) drugs for up to 12 months or (ii) avoid all breastfeeding.[1] In developed, well-resourced countries such as the UK, the latter is encouraged. DH advice is that HIV+ women living in the UK should not breastfeed.[2]
- Mothers with untreated TB should not breastfeed.
- Mothers with hepatitis C who have cracked or bleeding nipples should not breastfeed.
- Women who smoke or occasionally drink alcohol *can* still breastfeed; however smoking will lower the vitamin C content of breastmilk. Even so, it is still preferable to infant formula.
- *Certain drugs:* illegal drugs will pass into breastmilk, ∴ users should not breastfeed. Other medicines should be checked for suitability in the *British National Formulary* (℔ www.bnf.org/bnf/).
- Some types of breast surgery.
- Infants with galactosaemia (see Table 36.1) as they cannot metabolize galactose present in breastmilk. Lactose free infant formulae should be used.
- Phenylketonuria (PKU) infants should alternate breastmilk with phenylalanine-free formula.

[1] WHO (2009). *HIV and Infant Feeding: Revised Principles and Recommendations*. Available at: ℔ http://whqlibdoc.who.int/publications/2009/9789241598873_eng.pdf.
[2] DH (2004). HIV and Infant Feeding: guidance from the UK CMO's expert advisory group on AIDS. Available at: ℔ http://www.dh.gov.uk/dr_consum_dh/groups/dh_digitalassets/@dh/@en/documents/digitalasset/dh_4089893.pdf.

Promoting and establishing breastfeeding

Promoting breastfeeding

The DH (2003) recommends exclusive breastfeeding (nothing else but breastmilk, not even water) for the first 6 months (26 weeks) of life, following a systematic review conducted by the WHO (2001). However, exclusive breastfeeding remains infrequent in the UK.[1] Data from 2005 found 63–78% mothers breastfed at birth, but only 45% were breastfeeding exclusively at one week, 21% by 6 weeks and by 6 months the proportion of mothers exclusively breastfeeding was negligible (<1%).

Breastfeeding rates vary greatly and are highest among mothers from managerial and professional occupations, those with highest educational levels, those aged 30 or over and first time mothers. Young women in low-income areas with lower educational levels are least likely to initiate and continue breastfeeding.

The DH has targeted increasing breastfeeding rates at 6–8 weeks.[2] It has undertaken to support the NHS by:

- supporting a National Helpline for breastfeeding mothers;
- encouraging adoption of UNICEF's Baby Friendly Initiative (BFI—see Box 13.3)
- rolling out of the UK-WHO growth charts (see 📖 'Which growth charts should be used?', p. 240);
- developing a code of best practice for employers and businesses to support employees and customers who breastfeed;
- investing in an information campaign to promote breastfeeding benefits.

Promotion of breastfeeding needs to be part of an effective multifaceted approach across different settings.[3] Training needs to target all health professionals to emphasize both the enormous benefits of breastfeeding and appropriate techniques, so that women receive consistent messages throughout their care. The use of breastfeeding peer-support programmes is emphasized by NICE (2008),[4] with joint working between health professionals and peer supporters.[3]

Maternal education needs to begin antenatally by local health-care services providing breastfeeding classes and written support including leaflets. The father, family, and/or friends should be encouraged to participate so that the woman can be offered support.[4] Focus should be on changing attitudes and knowledge of the technique and the recommended length of time to continue feeding. Many young mothers from lower income areas lack access to key sources of advice and information, such as antenatal classes, peer support programmes, friends, family, and other

[1] Bolling, K., *et al.* (2007). *Infant feeding Survey 2005*. The Information Centre. BMRB International.
[2] DH (2008). *PSA Delivery Agreement 12: Improve the health and well-being of children and young people*. HM Treasury. TSO, London.
[3] DH (2009). *Healthy Child Programme. Pregnancy and the first five years of life.*
[4] NICE (2008). *Improving the nutrition of pregnant and breastfeeding mothers and children in low-income households*. NICE Public Health Guidance 11.

support networks. Support and education should be targeted at these groups.[3,4]

Common reasons given by women in the UK for the choice of feeding method and reasons for stopping breastfeeding[1] are useful targets for public health measures (see Tables 13.2 and 13.3). Three-quarters of breastfeeding mothers who gave up said they would have liked to continue, suggesting that they are committed if obstacles can be overcome.

Table 13.2 Reasons for choice of feeding method

Breast	Bottle
• Best for baby	• Others can feed baby
• Convenient	• Dislike idea of breastfeeding
• Closer bond	
• Cheaper	

Table 13.3 Reasons for stopping breastfeeding

First 2 weeks	Later months
• Rejecting the breast	• Returning to work
• Painful nipples	
• Insufficient milk	
• Takes too long/tiring	

Baby friendly initiative (BFI)

To improve breastfeeding rates in accordance with the DH (2008),[2] both maternity hospitals and community health care settings are seeking baby friendly accreditation (🖉 www.babyfriendly.org.uk/). This is awarded to services that adopt the '10 steps to successful breastfeeding' (hospital) or '7 point plan for protecting, promoting and supporting breastfeeding in community health care settings' (community) (see Box 13.3). In addition, there are now Baby Friendly University Standard awards (usually for universities involved in training midwives), and work is underway for neonatal units. Infant Feeding Advisors are being employed to help achieve baby friendly accreditation.

Box 13.3 Baby Friendly Initiative—10 steps

1 Have a written breastfeeding policy that is routinely communicated to all healthcare staff
2 Train all healthcare staff in skills necessary to implement this policy
3 Inform all pregnant women about the benefits and management of breastfeeding
4 Help mothers to initiate breastfeeding within an hour after birth
5 Show mothers how to breastfeed, and how to maintain lactation even if they are separated from their infants
6 Give newborn infants no food and drink other than breastmilk, unless medically indicated
7 Practice rooming-in—allow mothers and infants to remain together 24 h a day
8 Encourage breastfeeding on demand
9 Give no artificial teats or dummies to breastfeeding infants
10 Foster the establishment of breastfeeding support groups and refer mothers to them on discharge from the hospital or clinic

Plus: welcome breastfeeding in healthcare premises and support mothers to breastfeed in public places.

Establishing breastfeeding (Box 13.4)

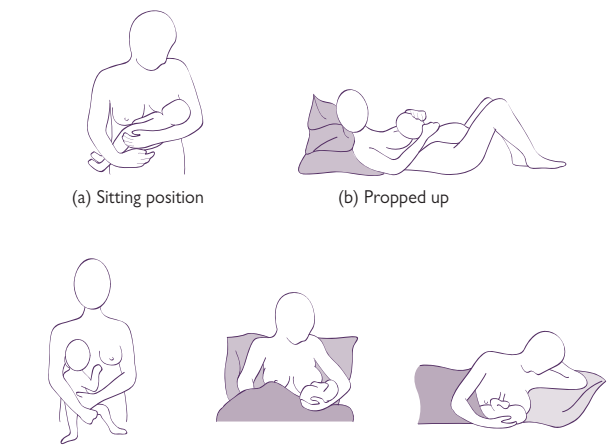

Fig. 13.1 Breastfeeding positions. (From Vinther, T. and Helsing, E. (1997). *Breastfeeding: how to support success. A practical guide for health workers*. World Health Organization, Geneva. Reproduced with permission (Available at: www.euro.whoint/document/e57592.pdf.).)

Box 13.4 Patients' FAQs for establishing breastfeeding

- How soon after the birth should I put my baby on the breast?

Start as soon after birth as possible (preferably within 1h) as suckling stimulates the let down response.

- How often should I feed?

Feed as often as the infant wants; not restricting frequency or duration will help fully establish the milk supply initially. Infants usually feed 8–12 times a day including at night. The first 3 weeks are crucial. Dummies should be avoided, as will diminish frequency of baby sucking.

- How long should I let my baby feed on each breast?

Always offer both breasts at each feed. Let the baby finish the first breast completely as incomplete emptying of the breasts means the baby may just drink 'foremilk' and not the fat dense 'hind milk'. If this is habitual practice, it may affect infant growth. Babies may seem sleepy but can often coax awake to feed for longer. Start on a different breast from the one last emptied.

- Which position is best for feeding my baby?

The most comfortable and convenient position of the baby on the breast will depend on the mother (see Fig. 13.1 and DH breastfeeding leaflet 'Off to the best start' for positions). If baby is restless at the breast and seems unsatisfied, it hurts to breastfeed, or the mother gets cracked nipples, adjust position of baby.

- How do I know if my baby is getting enough milk?

Plenty of wet nappies; bright yellow, regular stools (after the first week or two), contented baby after a feed; baby gains weight and looks well.

- Does my baby need extra drinks?

No. Foremilk is more watery and thirst quenching and in hot weather, babies tend to take shorter, more frequent feeds.

- My breasts are swollen, hard, and painful. Is this normal?

This is known as breast engorgement which occurs when your milk comes in about day 3 and can occur if there has been a delay between feeds. Feeding on demand should prevent it. If there is a lump, it's likely to be a blocked duct. Feed from the breast and massage the lump towards the nipple. If there is a red, hot painful patch it may be a sign of mastitis. Keep feeding from the breast and avoid wearing a bra at night.

- I want to carry on breastfeeding but I'm going back to work full-time when the baby is 3 months; what can I do?

If returning to work, exclusive breastfeeding will be challenging, unless the mother is extremely motivated and expresses and freezes breastmilk for use when at work. (NB. mothers are entitled to express breastmilk at work in the UK). A high quality breast pump is essential. Mixed bottle/breast may be a more realistic solution in this situation and the woman can continue with pre- and post-work breastfeeds. See 🕮 'Combining breast and bottle' in 'Establishing bottle-feeding', this chapter, p. 254.

Breastfeeding help and support further information

Association of Breastfeeding Mothers (ABM). Available at: www.abm.me.uk.
BDA Paediatric Group fact sheet 'Breastfeeding—best for baby'. Available at: http://www.bda.uk.com/foodfacts/090331Breastfeeding.pdf.
Breastfeeding network supporter line (0300 100 0210). Available at: www.breastfeedingnetwork.org.uk.
Department of Health (2007). '*Off to the best start*' leaflet.
Department of Health (2010). *Start4Life*. '*Off to the best start*' leaflet.
Department of Health *Bump to Breastfeeding (Best Beginnings)* DVD, given during pregnancy or available at: www.bestbeginnings.info.
Department of Health (2009). *The Pregnancy* book.
Department of Health (2009). *Birth to Five* book.
Department of Health publications web address: www.dh.gov.uk/publications.
IBFAN (International Baby Food Action Network). Available at: www.ibfan.org.
FSA (2002). *Eating for Breastfeeding*.
La Leche League (UK), (0845 120 2918). Available at: www.laleche.org.uk.
National Breastfeeding Helpline (0300 100 0212) or available at; www.nationalbreastfeedinghelpline.org.uk.
National Childbirth Trust (NCT), (0300 330 0771) www.nct.org.uk.

Dietary recommendations for lactation

Lactating women should follow general healthy eating guidance in 'The Eatwell Plate' (see 📖 Chapter 2, 'Food-based dietary guidelines', p. 24), but care should be taken to meet the extra requirements for energy, protein, 3 fat-soluble vitamins, 5 water-soluble vitamins and 6 minerals (Table 13.4). On a practical level, this can be achieved by women eating larger quantities of the healthy diet suggested earlier. In addition, women should:

- Limit alcohol to 1–2 units once or twice a week as alcohol will pass into breastmilk affecting its smell and potentially the sleep patterns and digestion of the baby. Try to avoid breastfeeding for at least 2–3 hours after drinking;
- Avoid too much caffeine as this will pass into breastmilk → infant hyperactivity and sleeplessness. Tea, coffee, cocoa, and cola -type drinks are best avoided (<4 cups a day of these combined). Suggest decaffeinated tea and coffee;
- Consume at least 2 l of fluid a day to avoid dehydration (35 ml/kg body weight). See 📖 Chapter 7 'Fluid balance', p. 162;
- Avoid spicy foods that may alter the taste of breastmilk if the infant appears to reject milk as a result;
- Can eat peanuts or foods containing peanuts as part of a balanced diet, even if a family history (siblings, mother, father) of allergy (asthma, eczema, hay fever) exists.
- Take a vitamin D supplement (10 µg)[1,2] whilst pregnant and breastfeeding. Those eligible for the UK Healthy Start scheme can obtain free vitamins (see 📖 this Chapter, 'Establishing bottle feeding,' 'Healthy Start Scheme', p. 256). Supplements of other vitamin and minerals are not usually necessary. Exceptions are vegan, macrobiotic, or fruitarian women who may need B12 supplements and women following dietary restrictions, e.g. cow's milk free diet. These and women with poor dietary intakes should see a dietitian.

When a woman is breastfeeding exclusively, her nutritional status will be compromised before that of the infant.

[1] DH (2008). *Healthy Child Programme. Pregnancy and the first five years of life.*
[2] NICE (2008). *Improving the nutrition of pregnant and breastfeeding mothers and children in low-income households.* NICE Public Health Guidance 11.

Table 13.4 RNIs for lactating mothers*

Nutrient	Daily RNI (15–50 years)	↑ in lactation
Energy (kcal)	1940–2110	+450 (month 1) +530 (month 2) +570 (months 3–6) if exclusively breastfed
Protein (g)†	51	+11 (0–4 months) +8 (>4 months)
Vitamin C (mg)	40	+30
Vitamin D (μg)	0 (assumed gained from sun exposure)	+10
Vitamin A (μg)	600	+350
Folic acid (μg)	200	+60
Thiamine (mg)	0.8	+0.2
Riboflavin (mg)	1.4	+0.5
Niacin (mg)	13–14	+2
Vitamin B_{12} (μg)	1.5	+0.5
Calcium (mg)	700–800	+550
Phosphorus (mg)	550–625	+440
Magnesium (mg)	270–300	+50
Zinc (mg)†	7.0	+6.0 (0–4 months) +2.5 (>4 months)
Selenium (μg)	60	+15
Copper (mg)	1.0–1.2	+0.3

*Department of Health (1991). *Dietary reference values for Food and nutrients for the United Kingdom*. HMSO, London.

† The RNI for zinc and protein falls after 4 months, which was based on previous DH advice of exclusive breastfeeding up to 4 months. It is likely that the current advice of exclusive breastfeeding up to 6 months will mean these will be revised upwards accordingly.

Establishing bottle-feeding

Women who choose to bottle-feed should not be made to feel guilty or inadequate as a result of their decision. Once this choice is made they need to be supported accordingly. Mother's breasts will return to normal quickly. Modified cows' milk infant formula will meet all nutrient requirements, but all protective immunological factors will be absent.

Combining breast and bottle

After breastmilk is well established (4–6 weeks), it may be possible to combine breast and bottle where circumstances dictate: e.g. woman returns to work; woman is exhausted (physically or mentally) from feeding continuously; or male partner needs to bond/help with the baby. Introducing 1 or 2 bottle feeds during the day, for example, and regularly continuing with breastmilk before and after returning from work should not affect the woman's ability to breastfeed. ▶NB. This is preferable to stopping breastfeeding entirely, but is not the 'ideal' option as exclusive breastfeeding is recommended for the first 6 months of life.

Choice of infant formula

In the UK, there are three main types of cows' milk formula:

- *Whey dominant* (most companies label as first milk): ratio of whey to casein of ~60:40. Whey-based formula is recommended as it most closely resembles the protein structure of breastmilk. Formulas vary, with 'extras' added based on components in breastmilk (see Box 13.2 box on protective factors of breastmilk, in breast versus bottle section); some have a higher ratio of α-lactalbumin, most contain added long chain polyunsaturated fats (docosahexaenoic acid and arachidonic acid), some have structured fats, many contain nucleotides and prebiotics, and some are organic. The wide choice can be confusing for parents.
- *Casein dominant* (most companies label as stage 2 milk). Whey to casein ratio of ~20:80; similar to doorstep cows' milk. They are marketed and perceived by parents as milks that can help fill up babies as they get 'hungrier' ~6–10 weeks (they contain the same energy, but >protein than whey dominant milks). Do not usually contain 'extras'.
- *Follow-on and toddler milks* (most companies label as stage 3, although one company confusingly has started labelling follow-on as stage 2 and toddler as stage 3). In UK, follow-on infant formulae are widely used for infants >6 months. Some parents wish to change their infant's milk at 4–6 months as a symbolic 'developmental milestone'. In these cases, follow-on milk is preferable to introducing solids early or doorstep cows' milk. Follow-on formulae are higher in iron and toddler milks contain additional vitamins and minerals, with a lower protein and sodium content compared with doorstep cow's milk. There is no evidence of nutritional benefit from changing milks, compared with remaining on first stage whey based infant formula during the first year of life, in infants and children who are following an otherwise well balanced diet. However, 'follow on' and 'toddler' milks may be useful in nutritionally at risk infants, particularly due to their higher iron content.

Infant formulae and energy

Although the energy content of breastmilk is reported to be 70 kcal/100 ml, this is based on the energy content of milk obtained by completely expressing the first breast. This is unlikely to be a true representation of suckled human milk, and doubly labelled water studies of suckled human milk have suggested values of 53–58 kcal/100 ml at 6 weeks and 3 months, respectively. The Scientific Committee for Food (SCF 2003) recommends maximum energy content of 60–70 kcal/100 ml for both standard and follow-on infant formulae.

Soya-based infant formulae

Infant formulae based on soya are not recommended for infants under 6 months of age, due to both the phytoestrogen content which may impact on future reproductive health, and risk of sensitization to soya protein. They should not be used in premature or low birth weight infants, or babies with impaired renal function. They should only be used in exceptional circumstances, e.g.

- vegan mothers who do not breastfeed;
- infants with cows' milk protein allergy, who consistently refuse elemental formulae;
- galactosaemia

If soya infant formula is used, it should be consumed as the main milk drink until ~2 years of age, when standard soya milk alternatives fortified with calcium, with energy content similar to semi-skimmed milk, can be used.

Goats' milk infant formulae

Infant formulae based on goats' milk protein are *not* recommended for infants as there is a lack of data on growth in infants fed on these. They are also *not* suitable for infants who are allergic to cows' milk formulae or who are lactose-intolerant.

Preparation of infant formula

Powdered infant formula is not a sterile product and could contain bacteria such as *Cronobacter sakazakii*. Formula should be made up with water between 70–80°C (equivalent to boiling a kettle and cooling for approx. 30 min). Higher temperatures will denature vitamin C. Microwaves should not be used. Bottles should ideally be made up one at a time, as storing bottles for any length of time increases risk of bacterial growth. A practical compromise would be to make up 1 bottle in advance and store in the back of a fridge (which should run between 2–4°C according to DH advice) to facilitate on demand feeding.

Bottle feeding support literature for parents

Department of Health (2008). *Bottle feeding*' leaflet.
Department of Health (2009). *Birth to Five* book.
Department of Health publications. Available at: www.dh.gov.uk/publications.

Healthy Start scheme

In the UK, the welfare food scheme, established in 1940 as a wartime measure, entitled pregnant women and children <5 years, receiving certain benefits, to tokens for either cows' milk or cows' milk based infant formulae per week and free supplements of vitamins A, C, and D. The NHS Plan 2000 reformed the welfare food scheme 'to use the resources more effectively to ensure that children in poverty have access to a healthy diet, with increased support for breastfeeding and parenting'. As a result, the welfare food scheme was replaced by 'Healthy Start' in 2006.

Healthy Start involves a broader range of foods; fresh fruit and vegetables, as well as cows' milk and infant formula. Fixed value vouchers are issued so they can be exchanged in a wide range of outlets and are of equal value for both breastfeeding and non-breastfeeding mothers. The age of children eligible has fallen from 5 to 4 years. Milk or fruit (not both) are available in nurseries. Free vitamin supplements continue to be available as part of the scheme, but now there are 2 versions; one for pregnant and breastfeeding women, and one for infants and children (Table 13.5).

Table 13.5 Nutrient content of free healthy start vitamin supplements

Nutrient	Pregnant and breastfeeding women	Infants and Children
Vitamin D (μg)	10	7.5
Vitamin C (mg)	70	20
Vitamin A (μg)	0	233
Folic acid (μg)	400	0

All pregnant and breastfeeding women should take 10 μg vitamin D. All breastfed infants >6 months and infants taking < 500 ml infant formula should commence vitamins A, C, and D, and continue until they are 4–5 years.

Healthy start is open to pregnant women and families with children <4 years who are in receipt of certain income support benefits or are <18 years (for further information see www.healthystart.nhs.uk or leaflet 'A Healthy Start for Pregnant Women and Young Children').

Weaning

When to introduce solids

Six months is the recommended age for the introduction of solid foods for infants (Department of Health, 2003), whether they are breastfed, fed solely on infant formula milk, or taking breast and infant formula combined. The choice of milk should continue >6 months, alongside complementary feeding.

The change in recommendation for introducing weaning from 4 to 6 months in 2003 generated much debate, partly due to lack of evidence to suggest that weaning after 4 months is detrimental to health in the developed world. ESPGHAN (2008) state that exclusive or full breast-feeding for *about* 6 months is a desirable goal.[1] Complementary feeding should not be introduced before 17 weeks, and all infants should start solids by 26 weeks. They also state that it is important to avoid both early (<4 months) and late (≥7 months) introduction of gluten and to introduce gluten gradually while the infant is still breastfed, as this may reduce the risk of developing coeliac disease and type 1 diabetes. EFSA (2009) support this more flexible approach, stating that starting complementary feeding between 4–6 months in the EU is safe and does not pose a risk for adverse health effects.[2]

In practice, commencing complementary feeding is appropriate once neuromuscular co-ordination is sufficiently developed to enable the child to eat solid foods. This means being able to sit up with support, control their head, move food around their mouth, have the ability to chew and show an interest in family food. Weaning at 6 months is necessary to desensitize the mouth and delayed weaning can result in food refusal, especially of lumpy foods. Some nutrient requirements also increase (see Table 13.1), especially for iron, B vitamins, energy, and protein, and cannot be met by milk alone.

Which foods to introduce

The overall aim of weaning is to introduce the infant gradually to a range of foods, textures, and flavours, so that normal family foods are taken by 12 months. The stages of weaning are summarized in Table 13.6.

If parents choose to start weaning before 6 months of age, foods should be introduced gradually, starting with pureed and mashed fruits, vegetables, rice, and potato (Table 13.6). If starting weaning from 6 months, babies will need to be exposed to a variety of foods from the outset, especially those containing iron. Foods can be offered 2–3 times a day, of either thick purees or mashed textures and finger foods (Box 13.5). Weaning from 6 months is simpler as there are only few foods that cannot be offered.

As breastfeeding is baby led, letting the baby choose when to wean is favoured by some. Emphasis is on the baby self-feeding and discourages

[1] ESPGHAN- Society for Paediatric Gastroenterology, Hepatology and Nutrition (2008). Complementary feeding: a commentary by the ESPGHAN committee on nutrition. *J. Paed. Gastro. Nutr.*; **46**, 99–110.

[2] European Food Safety Authority (2009) Scientific opinion on the appropriate age for introduction of complementary feeding of infants. *EFSA J.* **7**(12), 1423–61.

the use of spoon feeding. This approach may be extreme and many parents would find it difficult not to feed their baby. A compromise might be to provide a meal containing a combination of finger foods such as vegetables and new potatoes or pasta and some mashed foods, so that the baby can feed him/herself and can also be fed by the parent/carer.

Box 13.5 Healthier snack suggestions for 1–5-year-olds

- All fresh fruit
- Popcorn (unsweetened)
- Sticks of carrot, celery
- Plain biscuits
- Peppers, cucumber
- Cherry tomatoes
- Olives without stones
- Cubes or slices of cheese
- Yogurt/fromage frais (lower sugar varieties)
- Teacakes/scone
- Low sugar cereal and milk
- Fruit or malt loaf
- Pitta bread and hummus/cream cheese
- Oat cakes/crackers
- Sandwiches, tortilla wraps, toast
- Rice cakes, bread sticks
- Bagels

Which foods to avoid <12 months

The DH (2010) recommends that infants <12mths should avoid:

For food safety reasons

- Raw eggs (risk of salmonella).
- Some fish—swordfish, marlin and shark (mercury affects CNS growth).
- Honey (risk of infant botulism—11 cases between 1980 and 2010).
- Whole nuts (choking risk).
- Shellfish (risk of food poisoning).

For healthy diet reasons

- Salt (kidneys immature).
- Sugar (encourage sweet tooth).
- High fibre foods (risk of high bulk → low energy intake → faltering growth).
- Low fat/low energy foods (risk of → low energy intake → faltering growth).

Table 13.6 Summary of guidance for weaning and feeding for under-5s

	6 months*	6–9 months	9–12 months	1–5 years
Milk	Breastmilk or 1 pint of infant formula. Introduce cup or beaker from 6 months			2–3 servings/day from full fat varieties, moving on to lower fat varieties >2 years of: cheese (30 g), yogurt (pot), 1/3 pint milk.
Dairy foods	Yogurt/custard	Cubed/grated hard cheese, cheese spread. Full fat yogurt, fromage frais, custard, full fat cows' milk in cooking		
	Goat's and sheep's milk should be avoided <12 months			
Fruit and vegetables	Smooth puree of softly cooked	2 servings/day. Mashed with fork/ lightly cooked; soft peeled fruit & vegetables as finger foods	3–4 servings/day. No need to peel apple/pear. Raw/lightly cooked	≥4 servings/day. Same form as for adults
Meat, fish, and alternatives	After a few weeks: pureed meat, beans and lentils. No raw eggs, marlin, shark or swordfish, shellfish, nuts, or nut butter <6 months	1 serving/day. Mince/pure meat, beans & lentils; hard-boiled egg, fish, tofu	2 servings/day, e.g. mince/chopped red meat, chicken, fish, eggs, tofu	2 servings/day. As for 9–12 months. Aim to include oily fish (sardines, salmon, mackerel) up to twice weekly but no swordfish, shark or marlin. No whole nuts <5 y due to choking risk
Starchy foods	Baby rice/smooth potatoes Wheat/gluten-based cereal	2–3 servings/day. Include gluten-containing foods, e.g. bread, breakfast cereals, toast, pasta	2–3 servings/day, e.g. toast, breadsticks, rice cakes	≥4 servings/day of bread, pasta, potatoes, rice, chapatti

Sugary foods and drinks	No added sugar and no honey <12 months due to infant botulism risks	Drinks should be breast/formula milk or water. Fruit juices should be discouraged but if taken diluted 1 in 10. No sweet biscuits and rusks (including low sugar)		Limit sweet foods and drinks; especially between meals
	Herbal drinks, fizzy drinks, and squashes, including 'diet' drinks with artificial sweetener are not recommended. No tea/coffee			
Salty foods	Salt should not be added; kidneys not mature		Small amounts of gravy/ ketchup	Limit crisps and savoury snacks
Vitamin drops	✓If the infant is still breastfed >6 months ✓If a formula-fed infant is taking <1 pint of milk/day			✓ DH recommends for all 1–5-year-olds
Texture	***Smooth purees, mashed foods***	***Mashed, soft lumps, soft finger foods, liquid in lidded beaker***	***Hard finger foods, chopped, minced family foods***	***Family foods***

*But no earlier than 4 months (17 weeks) for parents deciding to wean their babies earlier.

Weaning babies with risk of allergy

- Infants at risk have either an atopic parent or sibling
- Ideally should breastfeed through weaning and introduce high allergen foods (see Box 13.6) one at a time to assess effect.

No clear evidence that delaying introduction of high allergen foods ↓ risk of allergy.

Box 13.6 High allergen foods

- Milk
- Mustard
- Eggs
- Peanuts, tree nuts
- Fish and shellfish
- Wheat
- Sesame seeds
- Lupins
- Celery.

Nuts and nut allergy

Peanut allergy appears to be increasing in children (see Chapter 37, 'Food hypersensitivity', p. 730). When introducing peanut into a child's diet, the FSA's revised advice (2009) states that if parents choose to start weaning <6 months of age, they should not introduce peanuts or other allergenic foods (such as other nuts, seeds, milk, eggs, wheat, fish, or shellfish) before this time, and when they do, these foods should be introduced one at a time so that they can spot any allergic reaction. However, there are studies currently underway looking at the effects of early introduction of peanuts or a range of food groups (LEAP—www.leapstudy.co.uk and EAT—www.eatstudy.co.uk), with the hypothesis that early introduction of allergens may reduce the risk of developing food allergy.

The FSA also advise that where a child already has another kind of allergy (e.g. diagnosed eczema or allergy to other foods), or if there is a history of allergy in the child's immediate family (parents, siblings), then parents/carers should talk to their GP, health visitor or medical allergy specialist before giving peanut to the child for the first time, because these children are at higher risk of developing peanut allergy. Other infants can take peanuts and other nuts from 6 months of age. Whole nuts should be avoided generally until 5 years of age due to the risk of choking.

Weaning preterm infants

Advice on appropriate weaning age should be sought from the specialist paediatric medical and dietetic team caring for the infant. It is usually recommended that complementary feeding commences between 5–7 months actual birth age. Further information on feeding preterm infants is available from www.bliss.org.uk.

Weaning Support literature for parents

Department of Health (2009). *Weaning—Starting solid food* leaflet.
Department of Health (2010). *Start4Life 'Introducing solid foods'* leaflet.
Department of Health (2009). *Birth to Five* book.
Department of Health publications web address: www.dh.gov.uk/publications.

Common feeding problems

Prolonged use of feeder bottle and delayed weaning

The DH recommends that infants should be introduced to drinking from a cup around 6 months age and actively discouraged from taking drinks in feeder bottles after 12 months. This is part of the natural progression for sipping and swallowing to replace sucking. Delayed weaning (>1 year) is more common in some deprived South Asian communities than for other ethnic groups.

Problems arising from prolonged use of the feeder bottle and delayed weaning include:

- Food refusal as the infant may be filling up on milk ∴↓ desire for food.
- Iron deficiency anaemia due to increased iron requirements not being met from a mixed diet.
- Faltering growth.
- Speech development as child's ability to chew and the swallowing reflex may ↓.
- Dental caries if sugary/acidic drinks are given in a bottle.
- Obesity if sugary drinks are given in a bottle.

❗ Risk of choking

All babies have a sensitive gag reflex and it is normal for them to gag on exposure to increasing textures of food. This should not be a reason for avoiding such textures, and they soon adapt. However, babies should be supervised whilst eating and given softer finger foods at first, such as banana, melon, or avocado. Once the child is able to chew well, s/he can be given non-dissolvable harder finger foods, e.g. apple. Infants do not need teeth to be able to chew solid foods. Whole nuts or olives containing stones should be avoided until 5 years of age due to risk of choking.

Iron deficiency anaemia in infancy

Common causes:

- Mother's diet was inadequate in iron during pregnancy;
- The baby is weaned late, i.e. >6 months;
- Slow in progressing from weaning foods to family meals;
- Early introduction of cows' milk as the main drink for children <1 year;
- Heavy reliance on sweet baby foods (high in sugar, low in iron and protein), as avoiding savoury products that may have non-*halal* ingredients in some Muslim families.

A varied diet with a regular intake of red meat, fruit, green vegetables, fortified breakfast cereals, and beans and pulses should be encouraged. This is especially important for breastfed babies because despite good absorption of iron from breastmilk, the iron content is insufficient after 6 months age as infants' endogenous stores are depleted. Infant formula is higher in iron, but it should not be relied upon as a sole source of iron in babies >6 months of age. 📖 Chapter 6, 'Iron,' for foods rich in iron.

Faltering growth

Weight faltering is defined as weight falling through centile spaces, low weight for height, or no catch-up from a low birth weight. Weight usually tracks within one centile, but an acute illness can cause a weight centile fall. <2% of infants will show a sustained drop through ≥2 weight centile spaces on the WHO charts, compared with 5% using the UK90 charts.

Growth faltering is defined as crossing down through length/height centile(s) as well as weight, a low height centile, or a height <expected from parental heights (Box 13.1).

Only 5% of young children whose weight or growth falters will have an organic root to the problem, see Box 13.7 for other causes. It is estimated that a further 5% will need the support of Child Protection Agencies. As a result, it is recommended that management for the majority of faltering growth should occur in primary care, rather than in hospitals.

Triggers for primary care assessment

- A weight or height below the 0.4th centile for the first time.
- A sustained fall through 2 centile spaces.

Managing faltering growth in primary care

In the UK, teams comprising of health visitors, community paediatric nurses and nursery nurses are best placed to identify and support infants because of their key responsibility for the health and well-being of children <5 years of age. It will depend on suspected cause as to whether a dietitian or pediatrician gets involved first; see Fig. 13.2.

Home visits to observe meal times are ideal and to collect data on:

- Feeding and symptom history since birth.
- Growth history since birth.
- Any relevant medical or domestic details.
- Food diary outlining food/drinks offered and taken and when.
- Details of mealtime routines, including observation of food preparation and mealtime interactions.
- Family's concerns/anxieties.
- Interaction between parents/carers and child, with description of any behavioural problems.

The Health Visitor will need to identify areas where there is potential for change and offer appropriate advice and ongoing support. This is likely to include advice on:

- Insufficient nutrient intake, e.g. faddy eating, excess drinking, poor parent–child interaction, strict adherence to a low fat-high fibre diet.
- Insufficient nutrient offered, e.g. poor parent/carer nutrition knowledge or food skills, stressful social situations, including neglect/abuse.

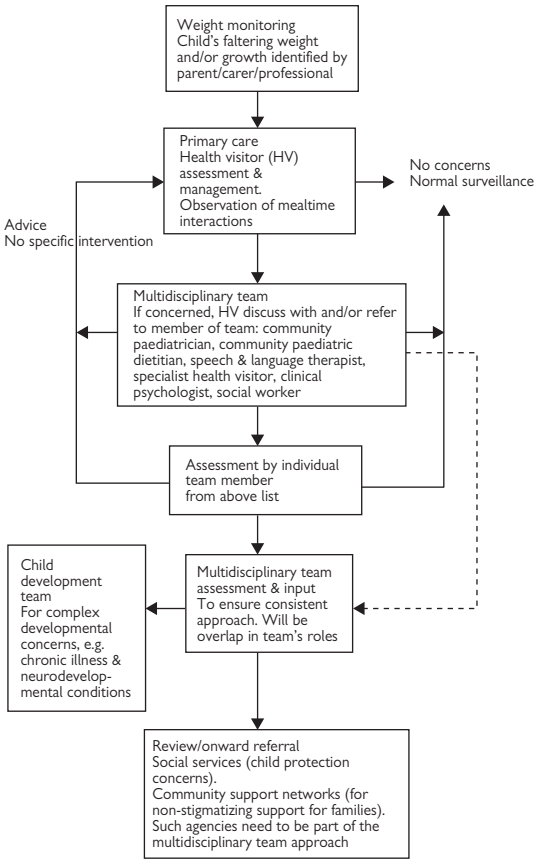

Fig. 13.2 Care pathway for young children's faltering growth/weight. Reproduced from the *Recommendations for best practice for growth faltering in young children* (Copyright 2002). With permission from the Children's Society.

When a health visitor becomes concerned, s/he should discuss or refer to the most relevant member of the multidisciplinary team (see Fig. 13.2).

The dietitian's role is to use motivational interviewing and counselling techniques to address the following:

- Positive food attitudes, value systems, and beliefs.
- Appropriate parent/child interactions, particularly related to food and drink.
- Drinking habits that discourage prolonged use of a bottle.
- Age-appropriate structured mealtimes, snacks, and drinks.
- Increasing nutrient density of meals using foods where possible.
- Identifying and correcting for any micronutrient deficiencies.
- Considering use of nutritional supplements if there is no improvement in growth as a result of the above interventions.

Also see 🕮 this Chapter, 'Fussy eaters', p. 276.

Box 13.7 Dietary and social causes of faltering growth

- Insufficient energy intake is major cause
- Formula milk too weak/too concentrated
- Late weaning >6 months
- Prolonged use of feeder bottle
- Fussy eating/behavioural problems at mealtimes
- Physical feeding problems, e.g. gastro-oesophageal reflux, oral motor dysfunction
- Over health conscious parent/carer → diet low in fat and high in fibre
- Inadequacy of the nutritional content or frequency of meals
- Poor inherent feeding drive
- Developmental difficulties
- Illness—although it is rare for serious organic disease to present with weight and/or growth faltering alone
- Abuse and/or neglect (minority of cases)
- Unhealthy parent/carer–child relationship.

Obesity prevention in infancy

The increase in number of children who are overweight or obese presents a major public health challenge. The Government's obesity strategy includes guidance on preventing obesity in pregnancy and the first years of life.[1] It intends to make breastfeeding the norm, which is believed to reduce the risk of excess weight in later life, delay weaning until around 6 months when healthy foods and portion size control should be emphasized, and encourage an active lifestyle. As a result, nutrition, active play and obesity prevention is one of the priority topics for the DH Healthy Child Programme, 2 year review.[2] Key messages for all families with young children are detailed in this document.

Age 2 years is the key age for identifying children who are overweight and for establishing life-long healthy eating and physical activity habits. Food that parents offer infants and toddlers influences taste preferences and eating habits and therefore later health. Factors influencing a child's weight include:

- parental attitudes to food, e.g. convenience foods;
- portion sizes;
- family eating behaviours, e.g. lack of family meal times;
- food choices, e.g. lack of healthy options;
- disincentives to physical activity;
- ease of sedentary entertainment, e.g. television and computers.

At 2 years, children at risk of obesity or causing concern should be measured for weight and height and a BMI centile obtained (see growth reference charts in 📖 this Chapter, p. 238 and 📖 Appendix 2, p. 764). A child with a BMI centile > 91st suggests overweight and BMI > 98th centile is very overweight (clinically obese).

Risk factors for obesity in infancy include:

- parental obesity;
- poverty;
- excessive weight gain in pregnancy;
- bottle feeding;
- rapid weight gain during infancy and early weaning.

If the child is confirmed as overweight or obese, the health visiting team should offer individual counselling and ongoing support of positive lifestyle changes and consider family-based as well as individual interventions, depending on the age and maturity of the child. HENRY (🖱 www.henry.org.uk), MEND 2–4 (🖱 www.mendprogramme.org/mendservices/minimend) and Trimtots are projects aiming to optimize eating behaviours and physical activity for under 5s and are currently being evaluated (2010).

[1] Cross-Government Obesity Unit, Department of Health and Department of Children, Schools and Families (2008). *Healthy weight, healthy lives: a cross-government strategy for England*. Available at: 🖱 www.dh.gov.uk/publications.

[2] Department of Health (2009). *Healthy Child Programme—the two year review*. Available at: 🖱 www.dh.gov.uk/publications.

Start4Life campaign

Start4Life (www.nhs.uk/start4life) is part of the UK government's social marketing Change4Life campaign, which is aimed at changing behaviour in relation to nutrition and physical activity in families with children aged 5–11 years. Start4Life was launched in 2009 and is aimed at pregnant women and families with children from birth to 2 years of age. It provides healthcare and childcare professionals with the most up-to-date advice on breastfeeding, introducing solid foods and active play. Start4Life communicates six key messages designed to build healthy habits from day one:

- Mother's milk.
- Every day counts (continuation of breastmilk).
- No rush to mush (waiting to wean).
- Taste for life (variety of solids).
- Sweet as they are (avoid sweet tooth).
- Baby moves (active play).

The campaign supports existing activities to increase the levels of initiation and continuation of breastfeeding and educate on introducing solid foods, with the aim of reducing obesity levels (and related illnesses) in later life. Parent support literature has been developed on breastfeeding and weaning (see relevant sections in this Chapter).

Constipation, toddler diarrhoea, and milk hypersensitivity

Constipation in infants

Constipation is more common in babies fed infant formula compared with breastfed babies. Potential contributing factors include absence of structured fatty acids, LCP's, alpha-lactalbumin and prebiotics in formula (see Box 13.2). The energy content of breastmilk may also be lower than originally thought, and hence fluid intake may differ.

In young infants who have not yet been weaned (<6 months of age) constipated bottle-fed infants should be given extra water in between feeds and correct preparation of formula should be established. Bicycling the infant's legs, or an abdominal massage may also help.

Formula-fed infants may have constipation caused by an allergy to cow's milk. A trial of an extensively hydrolyzed milk formula could be considered, particularly in those not responding to first line medical management. Constipation in an exclusively breastfed child is unusual, and it may be appropriate for the mother to undertake a cow's milk free diet. Breastfeeding should be continued as long as practical.

In infants who have begun weaning or who are now taking solids, extra water or diluted juices should be offered that have high sorbitol content (e.g. apples, grapes, pears, prunes). Giving a high-fibre cereal such as Weetabix® or Shredded Wheat® may also help infants >6months age.

Constipation due to low fibre intake and sometimes low fluid intake is relatively common in UK infants. A higher fibre diet should be encouraged, containing foods that are acceptable to the child. Encouraging fresh and dried fruit and vegetables for pectins, and bread and high fibre 'brown' breakfast cereals for insoluble fibre will relieve symptoms along with plenty of fluid, preferably as water.

All constipated infants (weaned or not weaned) should be prescribed an oral laxative[1] as first line treatment. An osmotic laxative (Lactulose®) is usually used in young infants but polyethylene glycol (Movicol®) is more favoured in children >2 years.

Toddler's diarrhoea

Symptoms: frequent, loose stools, containing undigested foodstuffs.

Usually a self-limiting problem, occurring in otherwise well infants <3 years, who are gaining weight and growing satisfactorily; commonly due to immaturity of the gastrointestinal tract. As well as reassuring parents that the condition will cease spontaneously, dietary treatment is to:

- avoid large quantities of sucrose, fruit and fruit juice;
- reduce dietary fibre intake (choose white bread and avoid high fibre cereals, fibre-dense fruit and vegetables, such as peas and sweetcorn, and temporarily reduce consumption of fruit and vegetables in general);
- ensure sufficient fat in the diet. Where this is not possible, a fat-based nutritional supplement can be a useful addition.

[1] NICE (2010) *Diagnosis and management of idiopathic childhood constipation in primary and secondary care*. NICE clinical guideline 99.

If foodstuffs are observed in the stools within 12 h of ingestion however, this would be considered to be rapid gut transit, indicative of possible food allergy.

Milk hypersensitivity (See 🕮 Chapter 37, p. 732)

Cow's milk hypersensitivity comprises of immediate (immunoglobulin E (IgE)-mediated) allergy, delayed symptoms (non-IgE mediated allergy) and lactose intolerance. IgE mediated reactions are relatively easy to diagnose due to immediate response (<2h) following ingestion of cow's milk protein and can be confirmed by skin prick tests or specific IgE antibodies. These can subsequently be used to determine when to re-challenge with milk.

Often the diagnosis between lactose intolerance and non-IgE mediated cow's milk protein allergy (for which there are no tests) is confused and it is important to differentiate between them. This can be done by taking a detailed food allergy clinical history focusing on feeding history from birth, related symptoms pertaining to gastrointestinal, cutaneous and respiratory systems and family history of allergy.

Lactose intolerance

Symptoms of lactose intolerance include diarrhoea, bloating and cramping

- Transient lactose intolerance is implicated in some cases of infantile colic (resolves around 4 months age), associated with delayed production of lactase.
- Secondary lactose intolerance is associated with loss of lactase as a result of damage to gut, e.g. delayed recovery post-gastroenteritis (resolves in approx 6 weeks).
- Primary lactose intolerance is associated with a gradual reduction in lactase activity over time, and generally does not affect infants and young children.

Treatment involves lactase drops (Colief®) or a reduced or low lactose formula alongside a low/reduced lactose diet for as long as required.

Cow's milk protein allergy

Conditions associated with non-IgE mediated cow's milk protein allergy in infants, especially those unresponsive to first line medication include gastro-oesophageal reflux (GOR), constipation, diarrhoea, faltering growth, eczema and aversive feeding behaviour.

Treatment involves use of an extensively hydrolysed or elemental infant formula alongside a strict cow's milk protein free diet. The allergy is likely to be outgrown during childhood, and re-challenging at 6–12 monthly intervals will be required. For non-IgE mediated reactions, this can usually be done at home.

Nutritionally vulnerable infants

Low income families

Children from families living on a low income are at a greater risk of having an unvaried, unbalanced diet, developing micronutrient deficiency and faltering growth. See 🕮 Chapter 16, 'Eating on a low income', p. 318.

In 2003, the UK Government published a Green Paper called *Every Child Matters* alongside the formal response to the report into the death of Victoria Climbie. After a thorough consultation process, the *Children Act 2004* became law. This legislation is the legal underpinning for *Every Child Matters*, which sets out the Government's approach to the well-being of children and young people from birth to age 19 years.

The aim of the *Every Child Matters* programme is to give all children the support they need to:

- be healthy;
- stay safe;
- enjoy and achieve;
- make a positive contribution;
- achieve economic well-being.

The *Every Child Matters* agenda has been developed further through publication of the *Children's Plan* in December 2007. *The Children's Plan* is a ten-year strategy to 'make England the best place in the world for children and young people to grow up'.

Sure start

Sure Start brings together childcare, early education, health and family-support services for families with children under 5 years old. It aims to tackle child poverty and social exclusion working with parents-to-be, parents, carers and children to promote the physical, intellectual and social development of babies and young children at home and when they get to school.

Children's centres are where children <5 years and their families can receive integrated services and information. Every community is served by a Sure Start Children's Centre, aiming to offer universal provision across the UK. For more information see 🖱 http://www.education.gov.uk/.

South Asian families

There is evidence that certain infant feeding practices in some South Asian families increase the risk of iron deficiency anaemia, faltering growth, and constipation. These include:

- late weaning, slow to move on to family foods;
- late progression from a bottle on to a feeder cup;
- adding honey/sugar to sweeten milk;
- adding solids to formula milk;
- choosing sweet commercially prepared baby foods so as to avoid running the risk of using non-*halal* meat products; ∴ lack of iron and protein in weaning foods;
- use of cows' milk as the main drink with infants <1 year.

The vegetarian baby

If infants consume a well-balanced vegetarian diet, there is no reason that they should not meet all requirements for growth. See 🕮 Chapter 16, 'Vegetarians', p. 312.

❗ Diets that are unbalanced or more restrictive than this, e.g. vegan, strict macrobiotic or fruitarian, are likely to result in nutrient deficiencies and need particular attention, especially in infants. The FSA state that vegan diets do not easily give babies all the energy and nutrients they need and therefore they do not recommend them for young babies. In all cases of highly restrictive diets, referral to a dietitian is essential.

Fussy eaters

Box 13.8 Parents' FAQs for fussy eaters for mealtimes

Every meal time is like a battle of wills; how can I break the cycle?
Encourage parents to avoid arguments and try to keep calm. Mealtimes should actually be fun! The child is probably trying to either gain attention or show that s/he has control over parents. It is important not to give in and to ignore the behaviour. The child should not detect that his/her behaviour causes anxiety. Reassure parents that as long as a child is gaining weight overall there isn't too much cause for concern. Never force feed children.

Should I let him have his dessert if he doesn't finish his main course?
Advise parents to put the food on the table and if it is not eaten after 20–30 min, simply remove with no comment. Suggest not giving sweet foods if the savoury meal is rejected completely. Trying the savoury meal may be acceptable and deserve a dessert. This will help change behaviour long term. Cooking an alternative meal should be avoided, as this is just as likely to be refused.

I am worried about my 2-year-old daughter going hungry if she doesn't eat her meal, is it okay to let her have in-between snacks?
Children can go for days without eating, and if they are hungry their behaviour deteriorates. If they refuse their meal, having a small snack (see Box 13.6) a couple of hours later will not impact on mealtimes, and it is far enough off for them not to associate snacks with not eating meals. This helps relieve parental anxiety too, as their child is getting something to eat. Mealtimes are approximately 4 hours apart, and snacks (mid am, mid pm, and supper) fall between these, but should not be given any closer than 1½ h before a meal. This applies to drinks too.

Is it okay to reward my child with sweets for eating his dinner?
Parents should avoid using sweet foods as a bribe to encourage children to eat their meals. Other non-food rewards could be used, like a cuddle, playing a game, or reading a story.

Could it help if he eats on his own, so that we can eat in peace later?
A young child should never be left alone whilst eating as there is a risk that s/he might choke. It is preferable if the whole family tries to sit down and eat together, ideally the same food, so that they are acting as role models and can share food. Inviting friends to eat can help as children often copy each other (as long as the guest is not a fussy eater too!). Parents need to try and create an enjoyable environment.

Box 13.8 (*Contd.*)

Is it okay to feed him myself whilst he watches TV, because at least that way he eats his dinner?

Parents should not feed the child (when s/he is capable of doing so) as the attention will be enjoyed and there will be little incentive to self-feed. It is normal for children to make a mess when they feed, and they like to eat with their fingers. This should be encouraged and avoid wiping their hands and mouth until the end of the meal, as it can upset the child. The TV should be switched off during eating as this is distracting and the meal is a good opportunity for parent-child interaction.

Promoting healthy infant feeding

See 'Model of infant feeding policy' (📖 Chapter 18, p. 373).

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Chapter 14

School-aged children and adolescents

Nutrition and growth in childhood and adolescence

- Children need a balanced diet to meet requirements for growth and development.
- Health-related behaviour and attitudes towards food are formed in childhood.
- The processes for some adult diseases may start early in life.

Growth and development

Each year of life from 1 year to adolescence, a child grows taller by 5–8 cm. Girls' growth spurt begins at 10–11 years. Boys' growth spurt begins at 12–13 years. About 25% of height is gained in adolescence. This requires increases of energy, protein, and several vitamins and minerals (see Table 14.1). If energy needs are not met this can result in stunting or delayed growth. Once the growth spurt is over, nutrient requirements become those of adults. During this period there is ↑ muscle growth in boys and adipose fat in girls. Genes have the strongest influence on onset of menarche.

See girls' and boys' growth charts in 📖 Appendix 2, p. 764. These have been endorsed by the Royal College of Paediatrics and Child Health and the Department of Health (DH). Also see discussion in 📖 Chapter 13, 'Infant growth and development', p. 238. BMI identification charts are on the back of growth charts and each centile represents 0.67 SD.

The charts have 9 reference centiles of 0.4th, 2nd, 9th, 25th, 50th, 75th 91st, 98th, and 99.9th, e.g.

- 98th centile curve, below which 98% of UK children lie (2 in 100 children will be as tall/ heavy as this);
- 50th centile curve, below which 50% of UK children lie (average weight and height for a child of that age);
- 2nd centile curve, below which only 2% of UK children lie (2 in 100 children will be as small/ light as this).

These are available from Harlow Healthcare (🖱 http://www.healthforallchildren.co.uk).

Dietary recommendations for children and adolescents

The 'Eatwell Plate' eating model (in 📖 Chapter 2, p. 27) applies to older children (≥5 years) and adolescents, as there is evidence that early atherosclerotic plaques can develop from adolescence → CVD in later life.

In addition to a balanced diet, advise:

- Extra calcium requirements gained from drinking the equivalent of 1 pint of milk a day (see 📖 Chapter 6, 'Calcium', p. 122 for equivalents).
- Regular meals if possible, healthy snack meals if not (see Box 14.1).
- Offering praise when a healthy food is eaten as this leads to ↑ consumption of the food in younger children.
- Parents to make healthy foods easily available and serve these foods in positive mealtime situations.
- As children prefer familiar foods, repeated exposure to new foods can alter the response from rejection to acceptance.
- Interventions promoting familiarity with foods, e.g. fruit and vegetable tasting, can increase consumption.
- Eating with peers can have a positive effect on eating behaviour.
- Eating together as a family is valued by children of all ages and it has been associated with lower levels of unhealthy weight-control behaviour and chronic dieting.

RNIs vary for age and gender and are related to growth needs; ∴ they reflect differences in growth rates and body composition (Table 14.1).

Box 14.1 Healthy snack suggestions

- Fruit
- Vegetables: sticks of carrot, celery, cucumber; cherry tomatoes
- Toast, teacakes, scone
- Fruit or malt loaf
- Oat cakes
- Crackers
- Rice cakes
- Bread sticks
- Bagels
- Mixed nuts and raisins
- Popcorn (unsweetened)
- Plain biscuits
- Glass of milk
- Cubes or slices of cheese
- Yogurt/fromage frais (lower sugar varieties)
- Low sugar cereal and milk
- Sandwich.

Table 14.1 RNI for school-aged children and adolescents*

Nutrient†	7–10 years		11–14 years		15–18 years	
	Boys	Girls	Boys	Girls	Boys	Girls
Energy (kcal)	1970	1740	2220	1845	2755	2110
Protein (g)	28.3	28.3	42.1	41.2	55.2	45.0
Fluid (ml/kg)	75	75	55	55	50	50
Vitamin C (mg)	30	30	35	35	40	40
Vitamin A (μg)	500	500	600	600	700	600
Folic acid (μg)	150	150	200	200	200	200
Thiamine (mg)	0.7	0.7	0.9	0.7	1.1	0.8
Riboflavin (mg)	1.0	1.0	1.2	1.1	1.3	1.1
Niacin (mg)	12	12	15	12	19	14
Vitamin B12 (μg)	1.0	1.0	1.2	1.2	1.5	1.5
Iron (mg)	8.7	8.7	11.3	14.8	11.3	14.8
Calcium (mg)	550	550	1000	800	1000	800
Phosphorus (mg)	450	450	775	625	775	625
Magnesium (mg)	200	200	280	280	300	300
Zinc (mg)	7.0	7.0	9.0	9.0	9.5	9.5
Selenium (μg)	30	30	45	45	70	60
Copper (mg)	0.7	0.7	0.8	0.8	1.0	1.0

* Department of Health (1991) *Dietary reference values for food and nutrients for the United Kingdom*. HMSO, London.

† In the UK there is not much evidence of difference in requirements for different ethnic groups except for vitamin D supplements for those Asian schoolchildren who have a lack of sunlight exposure.

Children and adolescents' food habits

The UK National Diet and Nutrition Survey of 2008/9 included data for 4- to 18-year-olds (DH, 2010)[1] and found the following:

- *Fruit and vegetable* intakes are low (~3 portions/day for boys and girls aged 11–18 years). Only 7.2% of girls and 22.1% of boys aged 11–18 years achieve the recommendation of ≥5-a-day. ►In the UK, portions of fruit and vegetables for the 'five a day' recommendation can include up to 150 ml fruit juice and a portion of pulses, including baked beans.
- *Total fat* intake of 4–18-year-olds is at the top end of the recommendation (≤35% food energy from fat), with girls (mean of 35.7% energy from fat) reporting higher intakes than boys (mean of 34.3% energy from fat). Similar for 4–10 years and 11–18 years age groups.
- *Saturated fat* intake of 4–18-year-olds is higher than the recommendation (≤11% food energy from saturated fat), with girls (mean of 13.7% energy from saturated fat) reporting slightly higher intakes than boys (mean of 13.0% energy from saturated fat). Intakes higher for younger (4–10 years) than older (11–18 years) children.
- *Non-milk extrinsic sugars intake* of 4–18-year-olds is higher than the recommendation (≤11% food energy from non-milk extrinsic sugars), with girls (mean of 14.8% energy from non-milk extrinsic sugars) reporting lower intakes than boys (mean of 15.5% energy from non-milk extrinsic sugars). Children's intakes are higher than those of adults in the National Diet and Nutrition Survey (NDNS; mean 12.5% from non-milk extrinsic sugars). The SACN[2] report of nutritional wellbeing of the British population reported even higher intakes of 19% food energy from Non-milk extrinsic sugars, mainly from soft drinks (mean of 3 l/week in 7–10-year-olds).
- *NSP* (*aka* dietary fibre) intakes are lower than the recommendation (mean intake of 18 g/day), with girls (mean of 10.6 g/day) reporting lower intakes than boys (mean of 12.2 g/day). Intakes are higher for older (11–18 years) than younger (4–10 years) children. Children's intakes are lower than those of adults in the NDNS (mean of 14.0 g/day).

Other features of children's habits

Missing breakfast

In the UK ~1 in 3 children[3] aged 10–16 years miss breakfast some or most days. More girls (38.6%) than boys (26.6%) skip breakfast some or all of the time, with girls motivated by controlling their weight and boys by lack of time. This has been linked to poor concentration and ↓ cognitive performance late morning.

[1] D.H/F.S.A (2010). *National Diet and Nutrition Survey of 2008*. TSO, London.
[2] SACN (2008). *The Nutritional Wellbeing of the British Population*. TSO, London.
[3] Sandercock, G.J.M., Voss, C., and Dye, L., *et al.* (2010). Associations between habitual school-day breakfast consumption, body mass index, physical activity and cardiorespiratory fitness in English schoolchildren. *Eur. J. Clin. Nutr.* 64, 1086–1092.

Drinking alcohol

Especially of concern is binge drinking in adolescence. Weekly alcohol consumption amongst Welsh adolescents is higher than for any other European country, with England and Northern Ireland not far behind. The WHO[4] estimated that 55.9% of boys and 48.6% girls aged 15 years drink alcohol ≥once/week. In the UK, alcohol consumption and smoking are increasing, particularly in girls. Alcohol provides extra calories (could lead to weight gain) and could displace nutrient rich foods in the diet.

The SACN report (2008) found that boys and girls aged 15–18 years reported a mean alcohol consumption of 9 and 7 units of alcohol/week, respectively.

Sedentary behaviour

The Chief Medical Officer recommends that all young people aged 5–18 years should participate in physical activity of at least moderate intensity *for 1 h a day*. This can be accumulated throughout the day, e.g. 4 times 15 min, and can be through structured exercise, sport, or everyday physical activity as part of habits.

- Only about 32% of boys and 24% of girls (2–15 years old) meet this recommendation.[5]
- Among girls, physical activity levels tend to fall with age and by age 14, only 12% achieve the recommended levels of activity. Boys' activity levels are less linked to their age.[5]
- A sedentary way of life is similar for boys and girls and increases with age, e.g. television and computer viewing of 3–4 h/day. High number of hours of television viewing is associated with ↑ risk of obesity.

Snacking

Adolescents eat at least 3 snacks/day, contributing ~25% of daily dietary energy. Snacking can have a negative effect on the nutritional value of the diet as snacks are often low in calcium, iron and high in saturated fat and sugar. Most popular are crisps, sweets, biscuits, sandwiches, fruit, and milk chocolate. Box 14.1 lists some healthy alternatives.

[4] WHO (2004). *Global status report on alcohol 2004*. WHO, Geneva.
[5] Department of Health (2008). *Health Survey for England 2008*. The Health and Social Care Information Centre. TSO, London.

Nutrient deficiencies in children

The UK National Diet and Nutrition Survey of 2008/9 included data for 4–18-year-olds (DH, 2010)[1] on nutrient intake and found that most children meet micronutrient recommendations, with some exceptions.

Iron

- Iron is the most prevalent nutrient deficiency in UK girls: 27% of 11–18-year-olds do not meet the LRNI, compared with only 4% of boys.
- Mean intakes of iron are <RNI (see Table 14.1) particularly for older girls (11–18 years), i.e. mean intake is 8.5 mg/day for girls and 11.1 mg/day for boys. See Box 14.2 for foods rich in iron.
- Failure to meet iron requirement in girls is mainly due to iron requirement ↑ with onset of menstruation (from 8.7 to 14.8 mg/day at age 11 years).
- 27% of 15–18-year-old girls have low serum ferritin levels and 9% are anaemic (SACN, 2008).
- Low iron intakes are associated with ↓ physical activity → ↓ peak bone mass.

Box 14.2 Foods rich in iron (see 🕮 'Iron' in Chapter 6, p. 128 for full list)

- Peanut butter sandwiches
- Fortified breakfast cereals
- Dried apricots and raisins
- Red meat, e.g. beef, pork, lamb, lean minced meat, ham
- Egg yolk
- Leafy green vegetables
- Peas, beans, and lentils
- Tinned tuna and salmon.

Magnesium

Over a quarter of girls (27%) and 14% of boys did not meet the LRNI for magnesium. Mg is an integral part of bones and teeth and is a component of muscle and nerve cell function (see 🕮 Chapter 6, 'Magnesium', p. 144 for further information; see also Box 14.3).

Box 14.3 Good food sources of Mg

- Green vegetables
- Pulses and whole grain cereals
- Meat.

Calcium

Adolescence is a critical period with ~25–40% of peak bone mass laid down at this time in ♀, whilst ~90–95% of peak bone mass is attained by

[1] Department of Health/Food Standards Agency (2008). *National Diet and Nutrition Survey of 2008*. TSO, London.

30 years of age. Adequate calcium and phosphate intake is necessary combined with weight-bearing physical activity to maximize peak bone mass.

In the UK, more girls than boys do not achieve the LRNI for calcium, as 7% of 11–18-year-old girls and 4% of 11–18-year-old boys do not meet requirements. These estimates are lower than previously, suggesting that calcium content of diets is improving. However mean intakes of 702 mg/day in 11–18-year-old old girls are below RNI of 800–1000 mg/day. Boys fare better with mean intake of 919 mg/day.

Chronic dieting to lose weight in girls is also likely to contribute to suboptimal peak bone mass and osteoporosis in later life.

►Vitamin D also plays an important role in bone health as it is vital for calcium absorption the use of sun creams that reduce access to vitamin D via sunlight may be one explanation for why requirements are not met.

Dietary fibre

Constipation due to low fibre intake and sometimes low fluid intake is relatively common in UK children. Encouraging fresh and dried fruit and vegetables for pectins, bread and breakfast cereals for cereal fibre, will relieve symptoms along with plenty of fluid, preferably as water. Children's access to fluid may be restricted during the school day; if so, they should be encouraged to carry bottled water/sugar-free fluids with them. They may intentionally restrict their fluid intake to reduce frequency of micturition.

► Children from low income households

- Children living in households in receipt of benefits are more likely to have intakes of vitamins and minerals below LRNI and eat less fruit and vegetables than other children (<2 portions/day; boys < girls).[1,2]
- Children from low income households are more likely to eat sausages, coated chicken and turkey, burgers and kebabs than adults.
- Sweetened soft drinks contribute more to non-milk extrinsic sugar intakes of children from low income households compared with the general population of children.

Also see 🕮 Chapter 16, 'Eating on a low income', p. 318.

[1] SACN (2008). *The Nutritional Wellbeing of the British Population*. London, TSO.
[2] Nelson, M., Erens, B., Bates, B., *et al.* (2007). *Low income diet and nutrition survey*. FSA. London, TSO.

Childhood obesity and weight problems

Obesity/overweight

↑ in childhood obesity worldwide due to widespread transition to energy dense diet and ↓ in physical activity. This is also the case in the UK, where an estimated sixth of boys (17%) and girls (15%) aged 2–15 years are obese and almost a third (31% boys and 29% girls) are either overweight or obese.[1] Prevalence of obesity increases with age, i.e. 21% of boys and 18% of girls aged 11–15 years are obese and is higher in children from lower income families (Low income diet and nutrition survey, 2007).

►Trends for obesity appear to be levelling off since 2006 in the UK.

Classification of childhood obesity

Assigning cut-off points for childhood obesity is more complex than for adults. BMI percentile chart should be used to identify obesity and the UK 1990 chart is recommended for routine clinical diagnosis. Overweight is classified as ≥91st centile; and obesity ≥98th centile of the UK 1990 data. For epidemiological studies, an internationally acceptable definition[2] to classify prevalence of child overweight (≥85th centile) and obesity (≥95th centile) of the 1990 data is recommended.[3]

Immediate effects on health

In extreme cases of childhood obesity, children can develop cardiomyopathy, pancreatitis, orthopaedic disorders, upper airway obstruction, or chest wall restriction.

Effects on well-being

Besides physical aspects, children also suffer from ↓ self-esteem, ↓ social interaction, and poorer academic achievement. Earlier puberty may also → emotional problems as a mismatch between physical and emotional development can → higher expectations from adults. However, obesity limited to childhood has little impact on social, psychological, economic, and educational outcomes in adult life. Persistent child to adult obesity is associated with poorer employment and relationship outcomes in females only.

Long-term effect on health

The ↑ prevalence of obesity and overweight combined with a diet low in fruit and vegetables, high in saturated fat, and low in calcium and low physical activity means that it is likely that there will also be an ↑ risk of type II diabetes, CVD, suboptimal peak bone mass, osteoporosis, gallstones, and diet-related cancers in later life, especially if the increase in obesity is sustained in adult life. Older obese children are at a higher risk of becoming obese adults, but not all obese children become obese adults.

Unnecessary dieting

Around a fifth of UK female adolescents are dieting to lose weight at any time with little evidence of a structured weight-reducing plan.

[1] Department of Health (2009). *Health survey for England 2008*. TSO, London.
[2] Cole, T.J., *et al.* (2000). Establishing a standard definition for child overweight and obesity worldwide: international survey. *Br. Med. J.* **320**,1240–3.
[3] For further information: Management of obesity in children and young people report. A clinical national guidelines. Available at: www.sign ac.uk.

Inappropriate approaches are common, such as crash diets, binge eating, chaotic eating plans, missing meals, eating slimming products (alongside energy dense foods), replacing meals with high sugar and fat snacks. This can lead to low intakes of several nutrients, especially iron, calcium, vitamin B_6, and riboflavin, as well as ↑ risk of developing eating disorders. See 🕮 Chapter 32, 'Eating disorders', p. 680.

Underweight

Prevalence of underweight in UK children is very low (<2%). Undernutrition in childhood and adolescence can result in stunting, i.e. inability to achieve inherited potential. Undernutrition may also impact on school achievement.

- *Diagnosis of stunting* in adolescents: height for age <2nd centile of WHO reference data or <−2 SD.
- *Diagnosis of thinness in adolescence*: BMI <2nd centile (UK National Child Measurement Programme). See 🕮 Chapter 25, 'Malnutrition universal screening tool', p. 504 and 'Undernutrition', p. 508.

National child measurement programme

This is part of the UK Government's *Healthy Weight, Healthy Lives* strategy. This cross government strategy, developed in England in 2005, operated jointly by the DH and the Department for Education. Every year, children in Reception and Year 6 are weighed and measured during the school year to inform local planning and delivery of services for children; and gather population-level surveillance data to allow analysis of trends in growth patterns and obesity. There has been a gradual shift from this being used to provide prevalence data to being used as a screening tool for childhood obesity, with each family receiving a letter informing them of the results of their child's BMI.

Further information see: 🖰 www.noo.org.uk/NCMP.

Preventing childhood obesity

See 🕮 this Chapter, 'Promoting healthy eating in children', p. 294, and Chapter 18 🕮 'Initiatives targeting children', p. 354 and 'Policy options for preventing obesity', p. 360.

Vegetarian children

Only 2% of children aged 4–10 years and 3% aged 11–18 years report being vegetarian in the UK's NDNS (2010). Vegetarian adolescents are at particular risk of inadequate diet if they are the only vegetarian in the house, as they may tend to eat the same as the rest of the family except 'remove' the animal protein aspect of the meal and replace it with too much cheese or vegetarian ready meals such as burgers, sausages, or pizza. They should replace it with suitable alternatives (See 🕮 Chapter 16, 'Vegetarians', p. 312). They may have less knowledge of a balanced vegetarian diet than adults resulting in low intakes of iron, zinc, protein, and vitamin B_{12}.

❗ Vegan children should be referred to see a dietitian as careful planning is needed to meet nutrient requirements.

Acne and diet

Young people and their parents often link acne with a diet high in fat and sugar, but there is little evidence that diet is a factor in causing acne. Reassure children that their eating pattern is not responsible for their acne; genes are an important determinant and this is one thing they can blame on their parents!

There has been recent debate regarding the possible link between acne and milk consumption as there appears to be some evidence that higher milk consumption is linked with ↑ risk of acne;[1] the authors hypothesize that this could be due to androgens, progesterone and insulin growth factor 1 present in milk that may stimulate acne.

[1] Spencer, E.H., Ferdowsian, H.R., Barnard, N.D., *et al.* (2009). Diet and acne a review of the evidence. *Int J Dermatol.* **48**, 339–47.

Dental health and children

In the UK, dental health in young people is improving, due to the introduction of fluoride toothpaste in the 1970s. However, it's still a public health concern, particularly in socially deprived groups, possibly because less preventive dentistry is practised; frequent consumption of sugary foods and ↓ regular brushing → ↓ fluoride intake.

Targeting for dental caries prevention is recommended as decay is unevenly distributed in the UK population: 9% of 5-year-olds and 6% of 14-year-olds have 50% of the disease (🖱 www.sign.ac.uk/pdf/sign47.pdf).[1]

There is ongoing debate in the UK and the EU concerning introduction of fluoride in non-water-fluoridated areas.

Causes of dental caries

- Frequent consumption of non-milk extrinsic sugars (glucose, sucrose, fructose) plays an unequivocal role in dental caries development as NMES act as a substrate for oral bacteria.
- Both quantity and stickiness of sugar affect length of time it takes for surface pH to ↑ to normal. Sticky foods may become stuck in-between teeth and are in contact with teeth for longer.

For guidelines for good dental health see Box 14.4.

Box 14.4 Guidelines for good dental health

Advise children to:

- Decrease both quantity and frequency of sugar intake
- Avoid sugary foods in-between meals and just before bedtime
- Avoid drinks that are carbonated, acidic, or high in sugar in-between meals (→ dental erosion); water and milk are better choices
- Eat sweet foods (including dried fruit) with meals rather than in-between (↑ saliva at end of meal → buffer of low pH)
- Avoid eating sticky and chewy foods
- Choose sugar-free medicines
- Regular flossing and brushing with fluoride toothpaste, but not immediately after consuming sweet foods/acidic drinks as ↓ mineralization
- Regular dental visits.

[1] Further information is available from SIGN guideline '*Preventing dental caries in children at high caries risk*' 2000. 🖱 www.sign.ac.uk.

Influences on children's food choices

The key factors influencing the eating habits of children and adolescents are shown in Fig. 14.1.

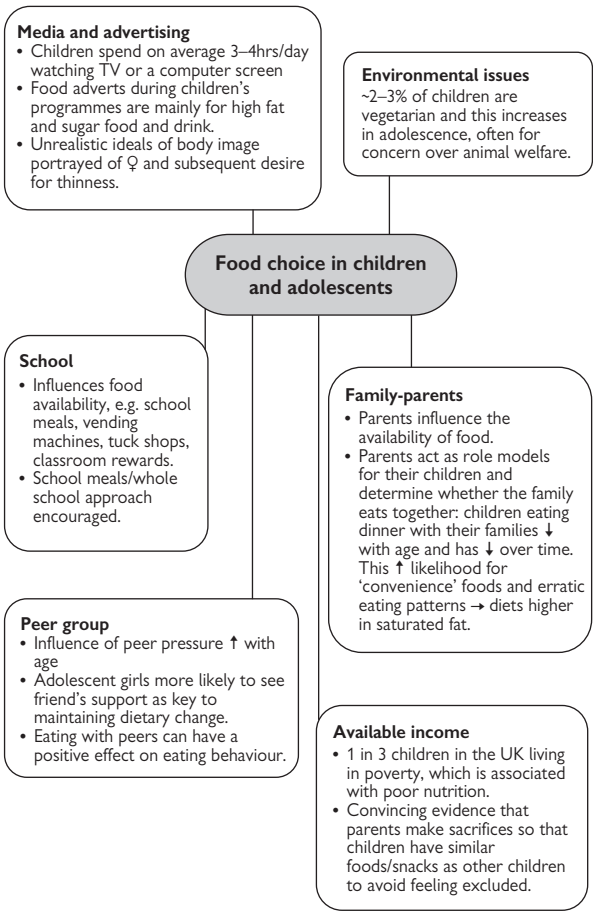

Fig. 14.1 Food choice in children and adolescents.

Promoting healthy eating in children

Implications from the various influences on children's food choice for nutrition education/health promotion are that a holistic approach needs to be used to tackle the multitude of influences on food choice. See 🕮 Chapter 17, 'Influences on food choice', p. 330.

Also see 🕮 Chapter 18, 'Model of a local school food policy', p. 374 (in examples of nutrition policy in different settings) and see 🕮 'Initiatives targeting children', p. 354 for information on school meals and national programmes.

Useful children's food photograph resources at CHEW (Children Eating Well) (🖰 www.cwt-chew.org.uk) for ages 5–11 and 12–18 years.

See 🕮 Chapter 10, 'Schools', p. 206 for detailed information on catering standards for school meal provision.

Chapter 15

Older people

An ageing population

Worldwide 10% of the population is now ≥60years, and this is expected to double by 2050.[1] The UK also has an ageing population; the number of older people (>65 years) has increased by 1.5 million over the last 25years to represent 16% of the population (Smith, 2010).[1] Life expectancy at birth is increasing steadily in the UK: a newborn baby boy could expect to live 76.6 years and a newborn baby girl 81.0 years (Hudson and Kyte, 2010)[2] if mortality rates don't change.

The vast majority of older people live in the community, either with family or in their own homes, but around 5% live in nursing and residential care homes, although this increases to over 1/4 of those aged >85 years.

The ageing process The ageing process involves a range of physiological and biological changes that vary between individuals. These influence digestive capacity, ↓ taste and nasal sensitivity and ↓ LBM, ↑ the relative proportion of adipose tissue and subsequently ↓ BMR.

What older people are eating Older people are not a homogeneous group and the majority are adequately nourished and meet the RNI for most vitamins and minerals.[3] However, those living in institutions and in lower socio-economic groups tend to have poorer diets.

- Intakes fail to meet recommendations for oily fish (mean intake of 0.1 portion/week) and NSP (only 12 g/day in free-living older people and even less in those living in institutions at 10 g/day).[4]
- Housebound and 'institutionalized' older people have lower intakes of fruit and vegetables (Mean intake of ~2 portions/day versus 3 portions/day in free living older people).
- Both free-living and institutionalized older people have Mean vitamin D intakes at 34% of the RNI, which is needed for the absorption of calcium and for bone health. 38% of the institutionalized group had low biochemical vitamin D status (vs. 8% of free-living older people).
- 46% of the institutionalized group was anaemic (vs. 10% of free-living older people).
- In some older people (>85 years), B vitamin status is poor, particularly for folic acid. (See 🕮 Chapter 6, 'Folate (folic acid)', p. 112 for good sources).
- A traditional diet is common, especially amongst older people in institutions, and the most commonly consumed foods are potatoes, white bread, biscuits, and tea.

There are categories of older people who are 'at risk' of malnutrition (see 🕮 this Chapter 'Undernutrition in older people', p. 298) and some of these depend on whether the individual is 'fit' or 'frail'.

[1] www.un.org/esa/socdev/ageing/popageing
[2] SACN (2008). The nutritional wellbeing of the British population. TSO, London
[3] Smith, C.W. (ed.) (2010). Population Trends, edition no. 142. ONS. www.statistics.gov.uk/populationtrends/ptissue/
[4] Hudson, M. and Kyte, L. (2010). Life expectancy at birth and at age 65 by local areas in the United Kingdom, 2007–09. *Statist. Bull.* ONS. http://www.statistics.gov.uk/pdfdir/liex1010.pdf

Dietary recommendations for older people

Some of the RNIs vary with age; ∴ they reflect differences in body composition and needs. In adulthood, requirements fall for calcium, phosphorus, niacin, thiamin (♂ only) and iron (but not before 50 years in ♀). Requirements for copper and selenium increase in adult males. See 🕮 Appendix 6, p. 777 for full dietary reference values for adults and older adults aged >50 years. Vitamin D is the only nutrient where dietary requirement increases (from zero to 10 µg from age 50 years) because some older people will have less exposure to the sun and therefore their vitamin D requirement will not necessarily be met by sunlight alone.

General healthy eating guidance (see 🕮 Chapter 2, 'The Eatwell Plate', p. 27) applies to well older people (≥65 years), as a balanced, nutrient rich diet to meet requirements. In addition to a balanced diet based on the 'The Eatwell Plate', older people who are housebound will need to eat foods rich in vitamin D, e.g. milk (doorstep deliveries if available), eggs, fortified margarines, oily fish. The DH recommends regular sunlight exposure during May–September and vitamin D supplements during winter months, if housebound. If fresh fruit and vegetable consumption is difficult due to preparation or eating difficulties, juices and frozen and canned produce should be encouraged, as the nutrient value is just as rich.

Older people who are 'frail' are at higher risk of malnutrition, so the aspects of healthy eating guidelines related to reducing consumption of energy dense foods (from fat and sugar) are inappropriate. In practical terms this means that full-fat milk and full-fat margarines will be preferable, and sugar in the diet may provide useful calories and may help where taste sensitivity is ↓. However, sweet foods need to be part of a nutrient-rich diet and ideally eaten with meals.

Undernutrition in older people

There are categories of older people who are 'at risk' of malnutrition:

- *People who depend on others*, e.g. depending on others to shop for food (around half of older people rely on someone else to do some of their food shopping); living in institutions (hospitals, nursing, or residential care homes and sheltered housing). Nutritional problems are common among tenants of sheltered housing in England, data from 2009 found 14% of tenants were 'malnourished' (medium + high risk) using the MUST tool (Elia M & Russell CA, 2009).
- *People with difficulty eating*, e.g. poor dentition or sore mouth, masticating or swallowing disorders (see 🕮 Chapter 23 'Dysphagia', p. 480), sensory loss, disorders of the upper limb, difficulty manipulating cutlery (could be from arthritis).
- *Older age groups* (>75 years).
- *Lower socio-economic groups*: there is evidence that intakes of a range of nutrients are less in older lower socio-economic groups, especially magnesium and potassium in women. Access to shops may be worse.
- *People with illness-related malnutrition*, e.g. malignancy, Parkinson's disease, pressure sores (↑ requirements).
- *People in a vulnerable psychosocial situation*, e.g. loneliness, depression, dementia, recent bereavement → ↓ appetite.
- *People with physical difficulty in preparing food*, e.g. painful/frail hands from arthritis or reduced muscle strength, could → ↓ intake of fresh fruit and vegetables.
- *People on certain medication* affecting appetite, taste, GI tract (see 🕮 Chapter 38, 'Drug–nutrient interactions', p. 738).

However, in most cases, the causes of undernutrition are multifactorial but an awareness of some specific contributory factors is a valuable first step in prevention. (see 🕮 Chapter 25, 'Undernutrition', p. 508).

Nutrition screening for malnutrition risk factors in older people

Classifying undernutrition in older people is concerned with establishing risk. The consequences of failing to identify and treat undernutrition are potentially serious and therefore caution should be used when interpreting results. Vulnerable older people living in the community need regular nutritional assessments by a member of the PHCT using a routine nutritional screening tool (see 🕮 Chapter 25, 'Malnutrition universal screening tool', p. 504). Older people in residential homes should be assessed on entry (dietary intake and weight) and then weighed monthly.

Consequences of malnutrition in older people

The effects of undernutrition vary from subclinical, with no apparent clinical impairment to death, and are dependent on the type, length, and degree of nutritional inadequacy and the nutritional and health status of the individual.

In addition to a significant increased risk of mortality, undernutrition is associated with greater morbidity:

- Weight loss (predominantly fat and muscle), e.g. poorer mobility, ↑ risk of falls, ↑ risk of chest infection.

- Reduced immune function, e.g. ↑ rates of infection.
- Impaired synthesis of new protein, e.g. poor wound healing.
- Prolonged recovery from illness and hospital stays.
- Psychological, e.g. depression, anorexia, ↓ motivation.

See 🕮 Chapter 25, 'Undernutrition', p. 508.

Treatment of malnutrition

There is good evidence that nutritional support in older people can ↑ energy and protein intake, ↓ weight loss, ↑ functional outcomes (muscle strength, walking distances, activity levels, mental health) and clinical outcomes (mortality, complications) in hospital and community settings.

Treatment options could include:

- *Improving food access*, e.g. arranging support through appropriate carers, e.g. shopping, cooking, company whilst eating;
- *Supplementation of food and/or drink using ordinary food items*, e.g. increasing energy/protein density of meals by fortifying, e.g. adding butter and grated cheese to mash potato;
- *Supplementation using proprietary products*; some are available on prescription or over the counter, e.g. milk or juice-based drinks, soups, desserts.

Also see 🕮 Chapter 25, 'Enteral feeding', p. 516; see also Box 15.1.

Box 15.1 Tips for overcoming institutional factors contributing to malnutrition in care homes and hospitals

- A named individual needs to be responsible for nutritional care
- Staff breaks do not coincide with mealtimes
- Adequate staff to assist patients/residents with eating
- Avoid medical rounds or investigations at mealtimes
- Food served needs to meet nutritional recommendations (catering staff may need extra training), i.e. average day's food intake, estimated over a 1 week period should meet EAR for energy and RNI for other nutrients (Appendix 6)
- Food needs to be at the right temperature for eating
- Offer patients adequate choice with a varied meal cycle
- Provide facilities for patients/residents (who are able) to make drinks and snacks
- Provide storage facilities for patients'/residents' own food
- Evening meal should not be so early that patients/residents are not yet hungry
- A bedtime snack should be available for those who are hungry later in the evening
- Residents/patients should be consulted when devising new menus
- Portion size should be flexible to allow for smaller and larger appetites and needs
- Environment should be conducive to eating, i.e. clean, light, relaxing, as the social aspects of eating are important for well-being

Other nutritional problems in older people

Obesity

Obesity affects all age groups, and older people are no exception. For example, almost ¼ (24%) of tenants of sheltered housing in England were obese using the MUST tool (Elia M & Russell CA, 2009). Subsequent diet related chronic disease follows, such as type 2 diabetes, CVD (see 📖 Chapter 21, 'Obesity', p. 411).

Constipation

Constipation is relatively common in older people. Fibre and fluid intake need to be assessed and increased consumption encouraged. (see 📖 Chapter 26, 'Disorders of the colon', p. 602).

Iron deficiency anaemia

Around 10% of older people aged ≥65 years were found to have low iron stores in a population survey reanalyzed by SACN.[1] Low Hb levels were found in 37% men and 16% of women in the 85+ age group. May be due to poor intake or ↓ absorption from GI disorders, GI blood loss. Encourage iron-rich foods and vitamin C consumption (see 📖 Chapter 6, 'Iron', p. 128).

Arthritis

See 📖 Chapter 34 'Osteoarthritis', p. 702 and 📖 'Rheumatoid arthritis', p. 704.

Osteoporosis

Osteoporosis and the subsequent fractures (hip, wrist, spine) are an important cause of morbidity and mortality in older people. Calcium and vitamin D intake, along with sunlight exposure, are important means of maintaining bone health in older people. The DH recommends regular sunlight exposure during May to September and vitamin D supplements during winter months if housebound.[1] Being under and overweight can influence risk of osteoporosis. See 📖 Chapter 34, 'Osteoporosis', p. 712.

Dehydration

The risk of dehydration is more common in older people, especially those dependent on others or where there is mental impairment. A daily intake of 1500–2000 ml is recommended, around 6–8 mugs (1 mug = 250 ml).

Dementia

See 📖 Chapter 32, 'Dementia', p. 684.

[1] SACN (2008). *The nutritional wellbeing of the British population*. TSO, London.

Community strategies to promote a healthy diet for older people

Possible strategies include:

- *Community shopping facilities/home delivery:* serving isolated shoppers, usually grocery produce.
- *Community cafés:* where older people can eat a cheap meal in a sociable setting, e.g. at pensioners clubs, community centre.
- *Community transport:* to help bring older people that are isolated from shops nearer to them. Could be run by a local supermarket or local authority funded.
- *Cooking club:* practical group cooking sessions to improve food skills working with a health professional. Members of the group will cook and taste different recipes.
- *Box schemes:* customers receive a weekly box of fresh fruit and vegetables from a farmer that is distributed to a central place in the community. A group of people have to buy food regularly. Prices are more affordable as bought directly from the farmer.
- *Lunch clubs and meals on wheels:* provide hot meals for older and disabled people; may be run by the local authority or a voluntary organization such as Age UK or the WRVS (largest provider). The average meal over a 1–2-week period should provide ≥33% of RNI, except that energy, folate, vitamin C, calcium, and iron should be higher.
- *Grow your own:* e.g. growing food in allotments and back gardens.
- Developing a food policy for older people including guidelines for care homes (see Fig. 15.1 and Box 15.1).

Model of a food policy for older people

See Fig. 15.1. The composition of working party could be as follows.

- Dietitians working with older people.
- GPs, hospital doctors.
- Social services; social worker.
- Community dental health service.
- Community nurses.
- Older people's hospital nurses.
- Age Concern representative.
- Representative of residents/patients.
- Residential care home representative.
- Occupational therapist.

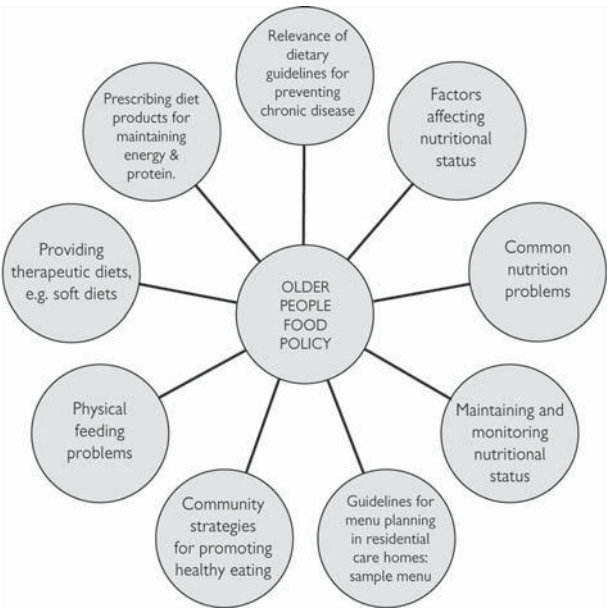

Fig. 15.1 A suggested format for a joint community–hospital-based food policy for older people.

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Chapter 16

Nutrition in vulnerable population groups

Minority ethnic communities

Traditional dietary habits and food restrictions

Traditional food restrictions for ethnic minority communities that are predominant in the UK are shown in Tables 16.1–16.3. There is a great deal of variety in dietary habits within all communities, including those of minority ethnic and religious groups and this has implications for nutrition education (Box 16.1); it is essential to find out the nature of the individual's restrictions; ∴ one cannot assume anything.

However, it is important to be aware of these orthodox food restrictions, even though there is great diversity in following them within UK minority ethnic communities. Younger generations of some ethnic groups are more likely to adopt a mixed diet incorporating that of the mainstream food culture.

Box 16.1 Implications for nutrition education

- Promote positive aspects of traditional diets and eating patterns
- Encourage use of readily available fruit and vegetables to incorporate into traditional eating patterns
- Encourage cooking and food preparation methods that reduce fat and sugar consumption of some traditional practices, e.g. oil/ ghee/ butter in curry or spread on chapattis
- Respect and take into account religious and cultural food restrictions (see Tables 16.1–16.3)
- Take account of the diverse patterns of responsibility for food shopping and cooking, e.g. men in some communities may be responsible for buying food
- Promote healthy eating within the whole family, not just centred on individuals
- Do not assume anything: each individual is different. There is a need to acknowledge this heterogeneity in nutrition education interventions
- Identify target groups that may be nutritionally at risk—low income groups, pregnant and lactating women, young children, older people
- There is a need to have good knowledge of health issues for each community to conduct evidence-based interventions
- Members of the local minority ethnic community should be employed as community or health workers where possible
- Information as leaflets, video, and audio should be available in the mother tongue, and recognize the diversity of food consumption in a given community, wherever possible
- For interventions using one-to-one communication, use interpreters where possible and necessary.

Table 16.1 Traditional food restrictions of Chinese and Jewish communities living in Great Britain (census 2001, Office National Statistics)*

	Chinese	Jewish
Religion	Include Taoism, Confucianism, Buddhism, Christianity, Islam	Judaism
Origin	Hong Kong, Malaysia, Singapore, China, Taiwan, Vietnam	Europe, Middle East
Language (besides English)	Written Chinese is common to all. Cantonese or Hakka are often spoken	Hebrew, Yiddish
Fasting	×	✓ 1 day for Yom Kippur and Tish'ah B'av
Staple cereals	Rice	–
Eggs	✓	✓
Dairy	✓ But not frequently Warm milk may be taken	✓ Some, but no cheese with rennet. Dairy foods not consumed at a meal with meat. Separate dishes and pans may be used for meat and dairy foods
Fish and shellfish	✓ Also salted fish	✓ If fins and scales. No shellfish
Poultry	✓	✓ Chicken, turkey, goose, and duck. No birds of prey. Don't eat fish and meat at the same meal
Red meat	✓ Mostly; except some religions	✓ Kosher† only meat, but pork and its products prohibited
Alcohol	✓ Mainly for celebrations	✓ Wine (ideally approved by the rabbi)
Caffeine	✓ But coffee not commonly drunk	✓
Nutritional implications	Low calcium intake could result from low consumption of dairy foods. High sodium intake could be a problem (from mono-sodium glutamate in soy sauce), NB. Food is seen as contributing to the body's balance, i.e. yang (foods that have a hot effect on body) and yin (cold affect). Cold food is often avoided	Traditional diet is high in total fat, saturated fat, and salt. ↑ Risk of obesity, type 2 diabetes and cardiovascular disease

* There is great diversity in following food restrictions as they should be used as a guide and not; a substitute for discussing individual dietary patterns.

† Kosher, slaughtered in a prescribed way.

Table 16.2 Traditional food restrictions of Asians living in Great Britain (4% of population census 2001, Office National Statistics)*

	Indian	Indian	Pakistani	Bangladeshi
Religion	Hindu	Sikh	Muslim	Muslim
Origin	Gujarat	Punjab	Pakistan	Bangladesh
Language (besides English)	Hindi/Kutchi	Punjabi, Hindi	Urdu, Punjabi	Bengali/ Sylheti
Fasting	✓ Certain holy days, especially month of Shravan	No religious obligation to fast	Especially month of Ramadan: no food during daylight. Pregnant, lactating and menstruating women, prepubescent children, diabetics and those needing regular medication are exempt	
Staple cereals	Chapatti, rice	Chapatti, rice	Chapatti, rice	Rice
Eggs	× If strict	✓ Probably; no if strict.	✓	✓
Dairy	✓ Milk, yogurt (may be home-made), but no cheese with rennet.			
Fish and shellfish	× If strict	✓ Possibly	✓ Possibly	✓ Often eaten
Poultry	×	✓ But no if strict.	✓ Halal†	✓ halal†

Red meat	✖ Most often lacto-vegetarian. Beef is prohibited	✓ Except no beef. May be lacto-vegetarian. No if strict.	✓ Halal† only, but pork and pork products prohibited	
Alcohol	✖ If strict	✓ If strict	✓	✓
Caffeine	✖ If strict	✓	✓	✓
Nutritional implications	If unbalanced vegetarian diet, possible deficiencies in protein and energy (faltering growth in infants), B_{12} and iron (anaemia), Calcium and vitamin D (rickets/osteomalacia). See 🕮 'vegetarians', this chapter, p. 312 and 🕮 'nutritionally vulnerable infants', Chapter 13, p. 274		Attention for Infant feeding: commercially prepared baby foods with non-halal meat may be replaced with sweet baby foods, low in iron and protein. See 🕮 'Nutritionally vulnerable infants', Chapter 13, p. 274	
	If curry cooked for a long time → ↓ folic acid, vitamins B_{12} and C.			
	↑ Risk of developing obesity, type 2 diabetes and CHD in later life; ∴ advise ↓ in fried foods (e.g. samosa, sev, bhaji, ganthia, puri, chevda, chips, crisps) and sweets (e.g. Indian sweets, including jelabi, burfi, gulab jamen, kulfi and laddo, and gur, jaggery, honey, chocolate, cakes and biscuits)			

*There is great diversity in following food restrictions; they should be used as a guide and not a substitute for discussing individual dietary patterns.

†Halal, slaughtered in a prescribed way.

Table 16.3 Traditional food restrictions of African Caribbeans and Black Africans living in Great Britain (2% of population census 2001, Office National Statistics)*

	African Caribbean			Black African
Religion	Christian	Rastafarian	Seventh Day Adventist	Muslim, Christian
Origin	West Indian Islands, especially Jamaica (60%), Dominica, Barbados, Trinidad.			Mainly Nigeria, Ghana, Somalia
Language (besides English)	Patois			Nigeria: Hausa, Yoruba, Ibo. Ghana:wi. Somalia: Somali
Fasting	–	–	×	✓ Especially month of Ramadan: no food during daylight. Pregnant, lactating and menstruating women, prepubescent children, diabetics and those needing regular medication are exempt
Staple cereals	Rice, plantain, yam, potato, pasta			Cassava, yam, plantain
Eggs	✓	×	✓	✓
Dairy	✓ But condensed and evaporated milk may be used instead of fresh milk	✓ Unless vegan	✓	✓ Milk, yogurt but possibly no cheese with rennet
Fish and shellfish	✓ Including salt fish	✓ If fins and scales. No shellfish	✓ If fins and scales. No shellfish	✓ Possibly
Poultry	✓	×	✓	✓ Halal† if Muslim

Red meat	✓	× Mostly vegetarian; some are vegans	✓ Mostly vegetarian, but some may eat meat but no pork	✓ If Muslim, possibly halal† only, but pork and pork products prohibited
Alcohol	✓	× If strict	×	× If strict Muslim
Caffeine	✓	× If strict	×	✓
Nutritional implications	Can be high in fat, sugar and salt. ↑ Risk of developing hypertension, CVD, and type 2 diabetes later	B_{12} deficiency seen in strict adherent to Ital diet		Attention for Infant feeding: commercially prepared baby foods with non-halal meat may be replaced with sweet baby foods, low in iron and protein. See 🕮 'Nutritionally vulnerable infants', Chapter 13, p. 274
	Possibly higher prevalence of lactose intolerance (see 🕮 Chapter 26, p. 584)			

*There is great diversity in following food restrictions; they should be used as a guide and not a substitute for discussing individual dietary patterns.

†Halal, slaughtered in a prescribed way.

Vegetarians

Trends in vegetarianism

In the UK, the number of vegetarians is estimated at 2–3% of the population in national surveys by the Food Standards Agency (FSA, 2008/9)[1] and Department for Environment, Food and Rural Affairs (DEFRA, 2007).[2] It is most prevalent in ♀, young people and adolescents, black and minority ethnic groups, and higher socio-economic groups. The same surveys report that a further 5–7% of adults describe themselves as partly vegetarian (avoiding red meat or fish). It is important to respect the individual's choice when giving dietary advice.

Common reasons for choosing a vegetarian diet include:

- religion (e.g. strict Hindus, Buddhists, and 7th Day Adventists);
- cultural;
- ethical, moral, or political beliefs;
- environmental concerns for use of world resources;
- animal welfare;
- perceived health benefits;
- food safety scares;
- limited availability of halal or kosher meat;
- financial constraints.

Types of vegetarian diets

One cannot always categorize individuals along these lines, as there is a large variation, so health professionals should avoid making assumptions about that foods are acceptable, but generally vegetarians fall into the groups shown in Table 16.4.

Is a vegetarian diet a risk for health?

A well balanced vegetarian diet can be nutritionally adequate for all age groups, ***but*** times of extra nutritional requirements need specific attention, i.e. pregnancy, lactation, infancy, childhood, and adolescence. Children consuming a well-balanced vegetarian diet should meet all requirements for growth. A well-planned vegetarian diet is more likely to comply with food-based dietary guidelines for reducing long-term risk of certain nutrition-related chronic diseases (NCD). There is evidence that vegetarians suffer less NCD than non-vegetarians, but this may be due to vegetarians adopting other health-promoting behavior patterns, e.g. being physically active, avoiding smoking, or drinking less alcohol. However, those who rely heavily on full-fat cheese and dairy foods could have a high saturated fat diet.

❗ Diets that are unbalanced or more restrictive, e.g. strict macrobiotic or fruitarian, are likely to result in nutrient deficiencies and need particular attention, especially in infants, children, and pregnant and lactating women. Referral to a dietitian for assessment is essential (Box 16.2).

[1] FSA/DH (2009) National Diet and Nutrition Survey. Headline results from Year 1 of the Rolling Programme (2008/2009).
[2] DEFRA (2005) Expenditure and food survey.

Table 16.4 Types of vegetarian diet

Type of vegetarian	Eggs*	Dairy	Fish and shellfish	Poultry	Red meat†
Vegan	×	×	×	×	×
Laco-vegetarian	×	✓	×	×	×
Lacto-ovo-vegetarian	✓	✓	×	×	×
Demi-vegetarian	✓	✓	✓	✓	×
Piscatarian	✓	✓	✓	×	×
Macrobiotic	×	×	✓‡	×	×
Fruitarian	×	×	×	×	×

*Possibly free-range only.

†Beef, lamb, pork; also sometimes their derivatives, e.g. gelatine, rennet.

‡Eaten at certain lower 'levels' of macrobiotic diet. Highest level eliminates everything except brown rice and water.

Box 16.2 Vegetarian groups at risk of an unbalanced diet

- Vegans
- Macrobiotics
- Fruitarians
- Strict Asian vegetarians
- Pregnant and lactating vegans
- Vegan infants and children
- Adolescent vegetarians
- 'New' vegetarians
- Vegetarians with an erratic eating pattern.

Possible nutrients needing special attention for vegetarians

The main nutrients to keep an eye on are: protein, energy, vitamin B_{12}, vitamin D, calcium, and iron. Iodine (vegans) and zinc intakes should also be verified.

Energy

Energy intake is only usually of concern for vegans, and those with more restrictive macrobiotic and fruitarian diets. They need to avoid a low energy diet that is too bulky and rich in fibre for infants and children as this could restrict growth; vegan children tend to be leaner than omnivores. See 🕮 Chapter 13, 'Nutritionally vulnerable infants', p. 274.

Protein

Plant protein can meet protein requirements if a wide variety of plant foods are consumed and energy needs are met, i.e. in well-balanced vegetarian diets. Protein is usually adequate if the diet contains a variety of the following (2–3 serving/day):

- Nuts and seeds, peanut butter.
- Beans and pulses, e.g. baked beans, red kidney beans, soya beans, chick peas, lentils, hummus.
- Soya products, e.g. bean curd (tofu), textured vegetable protein (TVP).
- Eggs.
- Dairy products: milk, cheese, yogurt, fromage frais.

For vegans, high quality protein (see 📖 Chapter 5, 'Protein', p. 58) can be achieved by 'protein complementing,' but energy intakes need to be adequate; otherwise protein is used for energy. Protein complementing foods must be consumed on the same day, but not necessarily at the same meal.

High quality protein =
grain (insufficient lysine) + pulse (insufficient methionine)

e.g. rice and dhal, baked beans on toast, or rice and peas.

Vitamin B_{12}

Animal foods are the main source of vitamin B_{12} (see 📖 Chapter 6, 'Cobalamin B_{12}', p. 116). Deficiency is rare, but vegans and those following stricter macrobiotic and fruitarian diets need to be advised to consume suitably fortified foods:

- yeast extracts/fortified vegetable stocks;
- fortified rice and soya milks;
- breakfast cereal fortified with B_{12};
- fortified blackcurrant cordial;
- fortified tinned spaghetti;
- almonds.

If vitamin B_{12} supplements are recommended these should not exceed the RNI. See DRV tables (📖 Appendix 6, p. 777).

NB. Vitamin B_{12} analogues in seaweed and algae are not well absorbed.

Vitamin D

Vegetarians are no exception to the UK RNI for vitamin D that adults <65 years and children >3 years should meet their vitamin D requirement by solar UV radiation if living a normal lifestyle.

At risk groups that should take a daily supplement of Vitamin D are:

- Asian vegetarian children, adolescents, and women (see 📖 Chapter 12, 'DRVs and dietary guidelines during pregnancy', p. 222).
- Children on strict vegan diets, especially African-Caribbean infants.
- Older vegetarians who are housebound or live in residential care.

Calcium

Vegetarians who consume dairy products regularly are not at risk of calcium deficiency. However, vegans, fruitarians, and macrobiotics may be at risk. Three servings should be eaten daily from a variety of sources:

- dairy products: milk, cheese, yogurt (if lacto-vegetarian);
- tofu;
- nuts: almonds, brazil, hazelnuts;
- fortified soya or rice milks;
- fortified bread;
- green leafy vegetables, e.g. broccoli, spinach, rocket, watercress;
- peas, beans and pulses, e.g. baked beans, red kidney beans, soya beans, chickpeas, broad beans;
- sesame seeds, tahini;
- dried fruit, e.g. apricots, figs;
- white bread and white flour products.

As vitamin D enhances calcium absorption, vegetarians at risk of poor vitamin D status in particular need to be encouraged to eat a variety of the above foods regularly. Vegan children and pregnant women should be referred to a dietitian who may recommend calcium supplements if dietary sources are insufficient.

Iron

UK vegetarians generally consume similar intakes of dietary iron to UK non-vegetarians. However, non-haem iron (plant sources) is absorbed less readily than haem iron (animal sources). Vegetarians should be encouraged to consume a good source of vitamin C to help absorption, e.g. citrus fruits and juices, and avoid drinking tea at the same meal (↓ absorption). See 🕮 Chapter 6, 'Iron', p. 128 and 'Vitamin C (ascorbic acid)', p. 104.

Good vegetarian sources of iron:

- eggs;
- wholemeal flour and bread;
- breakfast cereals fortified with iron;
- dark green leafy vegetables;
- beans and pulses;
- dried prunes, figs, and apricots;
- yeast extract.

Iodine

As milk is an important source of iodine in the UK, vegans, fruitarians, and macrobiotics are at risk of low intakes → ↑ levels of thyroid-stimulating hormone. Encourage vegans to use iodized salt, sea vegetables or take iodine supplements.

n-3 fatty acids

Vegetarian diets are often low in n-3 fatty acids. Diets that do not include fish, eggs, or large amounts of algae are low in n-3 fatty acids, EPA and DHA. n-3 fatty acids are important for cardiovascular health, eye, and brain development. Encourage vegetarians to include good sources of α linolenic acid in their diets, e.g. flaxseed, walnuts, canola oil and soya. Linolenic acid converts to eicosapentaenoic acid (EPA), but bioconversion is only about 10%.

Zinc

Intakes of zinc by vegetarians and vegans are not lower than for omnivores. However, there is low bioavailability from plant sources due to phytates inhibiting zinc absorption; ∴ intakes of at least the RNI should be encouraged (7–9.5 mg/day in adults, depending on age and gender; see dietary reference values (DRV) tables, 📖 Appendix 6, p. 777).

Dietary guidelines for a balanced vegetarian diet

See 📖 Chapter 2, 'The Eatwell Plate', p. 27.

Vegetarians and pregnancy

Being vegetarian should pose no problem in pregnancy if the woman is well informed and eating a balanced lacto-ovo and lacto-vegetarian diet. Pregnant vegan, fruitarian, and macrobiotic women should be seen by a dietitian to assess the overall nutrient adequacy of their diets. They may require supplementation of vitamin B_{12}, iron, vitamin D, or calcium (if <600 mg/day consumed). Some fortified soya milks contain these nutrients. DHA-rich microalgae may be needed by pregnant and lactating vegetarian women to meet n-3 fatty acid requirements.

The DH recommends vitamin D supplements (10 μg/day) during pregnancy and breastfeeding in addition to sunlight between April and September (see 📖 Chapter 6, 'Vitamin D (calciferols)', p. 100) and that only those on a restricted diet need extra vitamin D.

Asian vegetarian women could be at risk of vitamin D deficiency if there is insufficient skin exposure → neonatal hypocalcaemia and rickets, ∴ may need vitamin D supplements (see 📖 this Chapter, 'Minority ethnic communities', p. 306).

Vegetarianism in childhood and adolescence

See 📖 Chapter 14, 'Vegetarian children', p. 290.

The vegetarian baby

See 📖 Chapter 13, 'Nutritionally vulnerable infants', p. 274.

Further information

Further information on vegetarianism at the Vegetarian Society see: www.vegsoc.org/index.html.

Position of the American Dietetic Association. Vegetarian diets (2009) J Am Diet Ass **109**(7), 1266–82.

Eating on a low income

Scale of poverty

Some UK families and children are at much higher risk of poverty than others, particularly larger families, families of Pakistani and Bangladeshi origin, and families with one or more disabled adults and/or one or more disabled children. Within the UK and wider Europe the most common definition is that low household income is ≤60% of the median household income in a given year.

An estimated 21% of children and of older people receiving a pension are still living below the poverty line, although the proportion is declining.[1] However, the poor are not a homogeneous group and people can move in and out of poverty with changing employment, relationships, or other circumstances. This impacts on financial resources and therefore reduces opportunities for eating a healthy diet.

Causes of food poverty

Causes of food poverty are multifactorial, but poverty is strongly related to income, social exclusion, and physical access to food (proximity of shops selling healthy foods of good quality at affordable prices). Studies have shown that the poorest 10% of UK households spend almost a third of their income on food compared with a fifth in the richest. Mean weekly spending on food and drink in the general UK population has been estimated at £28 per person/per week,[2] and this is roughly the same for low income households.[3]

A typical basket of food purchased in local shops can cost around 25% more than from a large supermarket; this difference can rise up to 60% if purchases are compared with supermarket economy lines. However, 80% of low income households do their main shopping at a large supermarket (< the general population) and 50% have access to a car for shopping.[3]

Nutritional problems that are associated with food poverty do not arise because money is spent poorly on food, but because there is not enough money to spend. Several studies have shown that spending is often based on maximizing value for money in terms of energy intake and ∴ compromising micronutrient intake. Several key influences in food choice take prominence to influence behaviour: particularly access to shops, cost of food, budgeting strategies, and the need for cultural and social acceptability (see 🕮 Chapter 17, 'Influences on food choice', p. 330).

It has been proposed that 'food deserts', i.e. poor communities where residents cannot buy affordable healthy food, are an important contributor to poor diet. Others have questioned the lack of empirical evidence for this.

[1] Tomlinson, M. and Walker, R. (2009). *Coping with complexity: child and adult poverty*. Child Poverty Action Group, London.
[2] DEFRA (2005) *Expenditure and food survey*. DEFRA, London.
[3] Nelson, M., Erens, B., *et al*., (2007). Low income diet and nutrition Survey. Food Standards Agency TSO, London. 🖱 www.food.gov.uk.

Dietary and nutritional consequences of poverty

The UK Low income Diet and Nutrition Survey (LIDNS) of almost 2500 households[4] (representing the poorest 15% of the population) found that lower-income households eat a diet that fails to meet recommended intakes (Box 16.3), but it is only marginally worse than the diet of the rest of the population, i.e. participants were:

- more likely to consume soft drinks (not diet versions), processed meats, whole milk and sugar, saturated fat spreads and have non-starch polysaccharides (NSP) intakes <12 g/day;
- less likely to eat healthier foods, such as unsaturated-fat spreads and lower-fat milk, wholemeal products, vegetables;
- consuming average daily intakes of most vitamins and minerals from food sources above or close to the reference nutrient intake (RNI), but there was a proportion of the population whose intakes <RNI for Fe, Mg, K and Zn, and some were < lower reference nutrient intake (LRNI), e.g. for iron, around half of women and approximately 40% of girls aged 11–18 years were found to have Fe intakes <LRNI;
- 30% of households within this low-income sample lived in food-insecure households, i.e. they reported that during the previous year their access to enough food that is both sufficiently varied and culturally appropriate to sustain an active and healthy life had been limited by lack of money or other resources;
- 1/5th reported that they reduced or skipped meals regularly because of lack of money.

Children <5 years from low income families are also at greater risk of having an unbalanced diet, developing micronutrient deficiency, such as iron deficiency anaemia and faltering growth.

There is convincing evidence that parents make sacrifices so that children have similar foods/snacks as other children, to avoid being teased or ostracized by their peers.

The short- and long-term consequences of poor diet in childhood include: reduced immune status, increased dental caries, reduced cognitive function and learning ability, and increased risk of developing obesity and overweight. The long-term consequences for adults include increased prevalence of central obesity and of overweight/obesity in lower social economic groups (62% of men and 63% of women were obese or overweight in LIDNS), and reduced life expectancy for men and women.

Older adults and low income

There is evidence that intakes of a range of nutrients are less in older people living on a low income. Poor access to shops may be one contributing factor and more than half of poorer older adults >65 years do not have natural teeth (>general population of >65-year-olds), which may → ↓ fruit and vegetable intake.

[4] Nelson, M., Erens, B., *et al*., (2007). *Low income diet and nutrition survey*. Food Standards Agency TSO, London. Available at: www.food.gov.uk.

Older people are not a homogeneous group and, although the majority are adequately nourished and meet the RNI for most vitamins and minerals, those living in institutions and in lower socio-economic groups may be at nutritional risk. See 🕮 Chapter 15, 'An ageing population', p. 296.

Box 16.3 Terminology

Food poverty
Is widely defined as 'the inability to acquire or consume an adequate quality or quantity of food in socially acceptable ways, or the uncertainty that one will be able to do so'. This may be because people lack shops in their area or have trouble reaching them. Other factors influencing food access are the availability of a range of healthy goods in local shops, income, transport, fear of crime, knowledge about what constitutes a healthy diet, and the skills to create healthy meals.

Food security
Is widely defined by the Food & Agriculture Organization (FAO) as meaning that 'people at all times should have physical and economic access to sufficient, affordable, safe and nutritious food necessary and appropriate for a healthy life, and the security of knowing that this access is sustainable in the future'. See 🕮 Chapter 19, 'Food security', p. 383.

Refugee
A refugee is a person who 'owing to a well-founded fear of being persecuted for reasons of race, religion, nationality, membership of a particular social group or political opinion is outside the country of his nationality and is unable, or owing to such fear, is unwilling to avail himself of the protection of that country; or who, not having a nationality and being outside the country of his former habitual residence, as a result of such events, is unable to or, owing to such fear, is unwilling to return to it'.[1]

Asylum seeker
An asylum seeker is someone who has applied for refugee status in order to be recognized as a refugee in the UK.

Statutorily homeless
After assessment by the local authority, the statutorily homeless qualify for permanent council/housing association housing. Includes people with dependent children, pregnant women, and vulnerable single people. They often wait in temporary accommodation such as bed and breakfast, and hostels that have limited cooking and storage facilities.

Non-statutorily homeless
The non-statutorily homeless do not qualify for permanent housing; they are usually single men. They often live in temporary accommodation such as bed and breakfast, and hostels that have limited cooking and storage facilities, but many 'sleep rough' on the streets, especially middle-aged, white men.

[1] United Nations (1951) *Convention relating to the status of refugees.* Available at: 🖱 http://www.unhcr.org/pages/49da0e466.html.

Refugees and asylum seekers

Refugees' health and nutritional status varies widely, and they should be treated on an individual basis as they are not a homogeneous group. Children may be well nourished or they may suffer from chronic undernutrition with growth stunting. The UK centiles have been complied using data from Caucasian children (see 📖 Chapter 13, 'Infant growth and development', p. 238) and therefore some ethnic groups, e.g. Ethiopians, may appear unusually tall whereas others may appear unusually short. It is important to refer children where there is more than a two centile discrepancy between height and weight, or where serial measurements of growth fail to show adequate weight or height gain.

However, there are a number of factors indicating nutritional risk.

- *Nutritional status on arrival in UK*: depends on the nature of their departure from their country of origin, whether time was spent in refugee camps, and exposure to communicable disease, which could all ↓ nutritional status and weight.
- *Poverty*: after arrival in the UK, many refugees live in poor housing, receive limited financial support, and have difficulty obtaining paid work → low income and poor diet. Professional skills may not be recognized.
- *Cultural factors*: foods eaten, preparation methods, and the social context of eating help define cultural identity. For refugees displaced from their own culture, friends, and family, the symbolic value of food can grow. Certain foods may not be available locally and familiar cooking implements or facilities unavailable. Whilst many refugees adapt, food intake will change by necessity, which may ↑ risk of a nutritionally inadequate diet compared with a traditional diet. Some refugees may have poor cooking skills as they are not used to preparing food, e.g. young men.
- *Communication*: refugees who do not speak English may have difficulty shopping for food, which is compounded when foods are unfamiliar and ingredients or cooking instructions cannot be read.
- *Psychological issues*: many have experienced violence, loss, or separation in their country of origin and two-thirds of adult refugees in the UK report anxiety or depression. This could ↓ appetite and interest in eating. Cooking and sharing food has been used as therapy with some refugees.

Homeless people

There are two main groups of homeless people: statutory and non-statutory homeless (see Box 16.3); these include those living in hostels and bed and breakfast accommodation, and those sleeping rough on the streets. Most homeless people have few or no means of buying, storing or preparing fresh food. A number of studies have shown that the health of homeless people is severely compromised due to inadequate diets, and those who do have usually received a free meal at a day centre. There is evidence of a high level of alcohol use among people sleeping rough, often co-existing with a mental health problem.

Some of the short-term consequences of a poor diet are:

- iron deficiency anaemia due to low intakes of meat, fruit, and vegetables;
- ↑ susceptibility to infection due to micro-and macronutrient deficiencies;
- constipation due to low dietary fibre intake from fruit and vegetables, and higher fibre cereals.

Some of the long-term consequences are:

- An increased risk of premature mortality from CVD and cancer. The average mortality rate of a homeless man is at ~42 years compared with the national average of 76 years.
- Dental caries and gum disease due to poor oral hygiene and eating patterns. If untreated could → difficulty eating certain 'hard' foods; ∴↑ chance of malnutrition.

Strategies to improve nutritional status of the homeless

Partnership working between staff and residents at hostels/bed and breakfasts, dietitians, city and borough councils, and health improvement officers can lead to improvements in the nutritional quality of meals provided by:

- developing nutrition education, e.g. practical nutrition resources and running cooking classes for residents on preparing healthy, affordable meals with limited cooking and storage facilities;
- lobbying for funding for 'mini-fridges' and microwaves in residents' rooms, locked food storage facilities, and better communal cooking facilities;
- producing a nutrition information pack for catering staff and home leaders that includes nutritional standards for meals provided.

See Box 16.4.

Box 16.4 Nutritional standards for meals provided in hostels, bed and breakfast accommodation, and day centres for homeless people

- Involve residents in menu planning
- Encourage the social and pleasurable aspects of eating
- Staff and volunteers need to be fully trained in food hygiene procedures
- Plan meals around a healthy eating food model (see 🕮 Chapter 2, 'The Eatwell Plate', p. 27)
- Use less salt and saturated fat in foods provided, e.g. ↓ processed foods used with low nutritional value, such as processed meat products, soups, and sauces
- Provide fortified breakfast cereals with a choice of full and lower fat milk
- Provide fruit and vegetables with every meal
- Provide plenty of foods rich in non-starch polysaccharides
- Provide water with meals
- Offer special diets where appropriate, i.e. therapeutic diets, and take into account dietary needs of people from different cultural groups (see 🕮 this Chapter, 'Minority ethnic communities', p. 306).

Policy options for reducing food poverty

The UK Low income Diet and Nutrition Survey of almost 2500 households[1] found that lower-income households eat a diet that fails to meet recommended intakes, but it is only marginally worse than the diet of the rest of the population. It has led to recommendations that health promotion campaigns on a national level should target the whole population, rather than low income groups. Cross-cutting policy initiatives that move beyond simple targeting and local action should be encouraged, that encompass a life-course approach and also recognize the diversity of 'low income' households.[2]

Ideas for local food projects

- *Community shops:* not-for-profit shop serving isolated shoppers, usually grocery produce. Also adds a social focus to communities.
- *Community cafés:* where people can eat a cheap meal in a sociable setting, e.g. at pensioners clubs, community centre.
- *Food co-operatives:* a group of people organizing to buy food in bulk direct from wholesalers or farmers to save money.
- *Community transport:* to help bring shops nearer to the isolated. Could be run by a local supermarket or local authority funded. Especially useful for older people and people with disabilities.
- *Links with local shops:* to stock and promote healthier food produce, encouraging people to use their local shops.
- *Food vouchers and coupons:* e.g. provided by local authorities by distributing 'money-off' coupons to local people or national government 'healthy start' vouchers for parents on a low income.
- *Farmers' markets:* where farmers and growers make up the majority of vendors.
- *Cooking club:* practical group cooking sessions to improve food skills working with a health professional. Members of the group will cook and taste different recipes.
- *Breakfast clubs:* providing healthy breakfast choices at school.
- *Box schemes:* customers receive a weekly box of fresh fruit and vegetables from a farmer that is distributed to a central place in the community. A group of people have to buy food regularly. Prices are more affordable as produce is bought directly from the farmer.
- *Lunch clubs and meals on wheels:* provide hot meals for older and disabled people; may be run by the local authority or a voluntary organization such as Age UK or the Women's Royal Voluntary Services (WRVS) (largest provider).
- *Food redistribution:* surplus food is moved from shops and supermarkets to day centres and homeless facilities to provide free meals.

[1] Nelson, M., Erens, B., *et al.*, (2007). *Low income diet and nutrition survey*. Food Standards Agency TSO, London. ℘ www.food.gov.uk.

[2] Dowler, E (2008). Policy initiatives to address low-income households' nutritional needs in the UK. *Proc. Nutr Soc*, **67**, 289–300.

- *Grow your own:* e.g. growing food in allotments, on wasteland, schools, parks, and in back gardens.
- *School Nutrition Action Groups:* multidisciplinary group that develops a whole school approach to better nutrition. See 🕮 Chapter 18, 'Model of a local school food policy,' p. 374.

Using a community development approach

The above local policy options need to be developed using a community development approach, i.e.

- Community-based, involving genuine partnerships between local residents, local workers, and professionals.
- Reaffirms community identity and meets local needs.
- Promotes active citizenship.
- Combats age, gender, and discrimination based on ethnicity.
- Encourages community participation.
- Addresses other social and cultural issues, as well as eating behaviour.
- Is flexible and responsive.

Useful websites

- www.ukfg.org.uk (The UK Food Group is network of NGOs working on global food and agriculture issues)
- www.sustainweb.org/foodaccess (The Food Access Network UK, formerly the Food Poverty project)
- www.fareshare.org.uk (Community Food Projects)
- www.refugeecouncil.org.uk
- www.cpag.org.uk (Child Poverty Action Group)
- www.endchildpoverty.org.uk
- www.actionforchildren.org.uk

Resources

Press, V. (2004). *Nutrition and food poverty. A toolkit for those involved in developing or implementing a local nutrition and food poverty strategy*. National Heart Forum/Faculty of Public Health. www.fph.org.uk/uploads/section_c.pdf.

Chapter 17

Nutrition intervention with individuals

Influences on food choice

Many factors interplay to influence the foods individuals choose, besides a basic physiological need to eat. Factors act on three levels: individual, societal, and national/international level (see Fig. 17.1). Interventions need to bear this in mind, so that they are matched at the right level.

Public health professionals working at a national/international level will be working primarily on the wider policy level, so they will need to be fully aware of these wider influences on food choice such as legislation, subsidies and taxes, world trade agreements, government policy, production methods and agricultural policy, advertising, plus global public health policy (WHO/FAO/UNICEF), global economic bodies (e.g. the World Bank, International Monetary Fund (IMF), World Trade Organization), intergovernmental agreements (International Conference on Nutrition, commercial interests (International Chamber of Commerce), European bodies (EU, Regional offices of WHO/FAO), and networks to promote public health (Healthy Cities Network, International Baby Food Network).

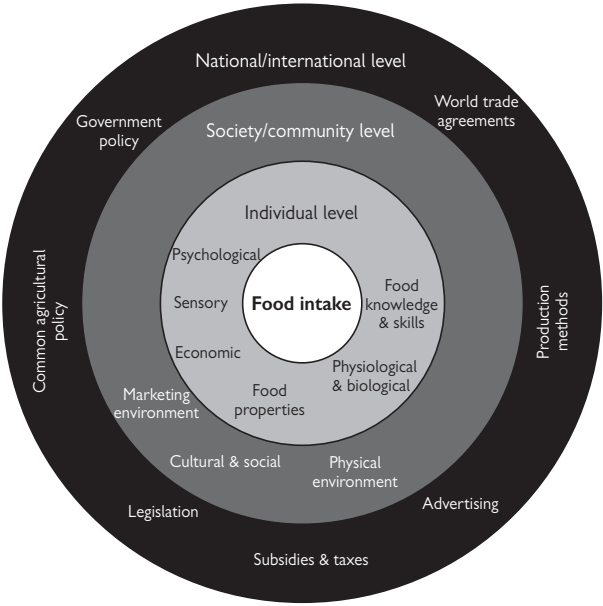


Fig. 17.1 Influences on food choice: individual, societal, and national/international level.

Health professionals working at the local level are able to influence some of the individual and societal/community influences on food choice (see Fig. 17.2). This could be during one-to-one consultations with patients and when developing appropriate public health nutrition programmes. It is not just 'what' is eaten that is important, but understanding 'why' and in which context it is eaten is crucial in helping people eat more healthily. Health professionals working locally will also need to have a wider vision of the international context of public health nutrition to be able to promote health, as there are conventions and international agreements that have an impact on a regional and local level. An example is the rapid increase in obesity, which is related to factors that are much wider than those that are modifiable locally. Professionals working in public health locally can be involved in advocacy work to try and stimulate change, e.g. lobbying government for stricter food and drink advertising regulations and nutrient standards for food procurement.

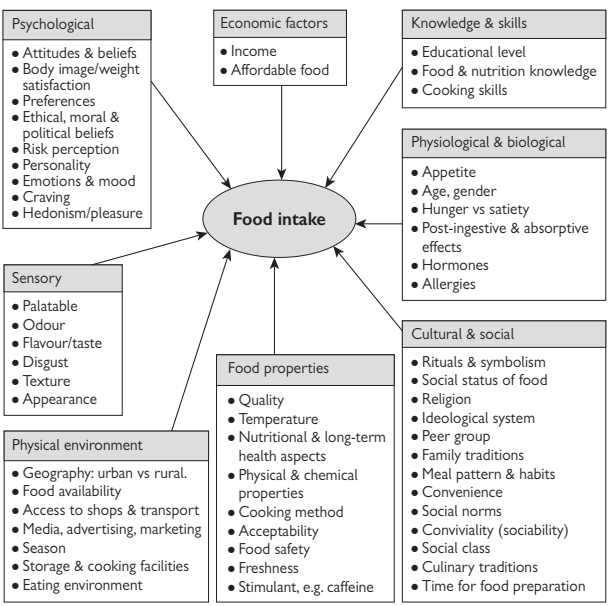


Fig. 17.2 Individual and societal/community influences on food choice.

Developing effective nutrition education messages

Nutrition education is a component of promoting healthier eating and is the process of applying scientific knowledge about diet–health to individuals' dietary behaviour to improve health literacy (nutrition and health related knowledge, attitudes, motivation, behavioural intentions, personal skills, and self-efficacy).

Nutrition education messages are most effective if they are:

- clear, simple, avoiding technical jargon;
- use foods rather than nutrients to communicate;
- consistent with other sources;
- personally relevant to the audience;
- sensitive to how consumers perceive the risk of unhealthy eating;
- positive (eat more fruit), rather than negative (eat less fat);
- emphasize the benefits of change;
- acknowledge barriers to change;
- avoid messages that stigmatize individuals;
- use persuasion, prompts, and reminders.

For further information see Contento (2006)[1]

[1] Contento, I.R. (2006). *Nutrition education—linking research, theory and practice*. Jones and Bartlett, Sudbury.

Planning nutrition education sessions

Where to start

Think of what you expect your audience to be able to do/to know before attending your session.

Then

- Think of them after they have attended it. What should they now be able to *do* as a result of that? (learning outcomes).
- The key word is *do*; so include active verbs when drafting learning outcomes (see Table 17.1).
- Learning outcomes should be measurable.
- Usually about 5–10 outcomes are sufficient.

Why do we need learning outcomes?

- To help the audience learn more effectively as they know what the goals are.
- To make it clear what the audience can hope to gain from attending a particular teaching session.
- To help practitioners to design materials more effectively by acting as a template for them (see 📖 this Chapter, 'Designing nutrition education materials', p. 338).
- To help choose an appropriate teaching strategy, e.g. lecture, seminar, group work.
- To assist team work as it is easier to share with colleagues what a particular activity is expected to achieve.

How are learning outcomes structured?

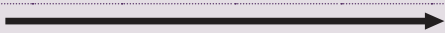
As well as being measurable, learning outcomes need to be matched to the type of learning expected. This is hierarchical with teaching 'knowledge' at a lower level of learning, whereas teaching 'evaluation' skills is the highest level (see Table 17.1).

- *Knowledge:* recalling appropriate previously learned information.
- *Comprehension:* understanding the meaning.
- *Application:* using previously learned information in new situations to solve problems that have single or best answers.
- *Analysis:* breaking down information into its component parts, examining and trying to understand the organizational structure of such information to develop divergent conclusions by identifying causes, making inferences.
- *Synthesis:* creatively applying prior knowledge and skills to produce a new or original whole.
- *Evaluation:* judging the value of material based on personal values/opinions, resulting in an end product, with a given purpose, without right or wrong answers.

See Table 17.2 for a sample lesson plan.

Table 17.1 Possible active verbs for defining learning outcomes

Knowledge	Comprehension	Application	Analysis	Synthesis	Evaluation
Arrange	Classify	Apply	Analyse	Arrange	Appraise
Order	Locate	Operate	Differentiate	Formulate	Judge
Define	Describe	Choose	Appraise	Assemble	Argue
Recognize	Recognize	Practice	Discriminate	Manage	Predict
Duplicate	Discuss	Demonstrate	Calculate	Collect	Assess
Label	Report	Schedule	Distinguish	Organize	Rate
Recall	Explain	Sketch	Categorize	Compose	Attach
List	Express	Employ	Examine	Plan	Score
Repeat	Review	Solve	Compare	Construct	Choose
Memorize	Identify	Illustrate	Experiment	Prepare	Select
Name	Select	Use	Contrast	Create	Compare
State	Indicate	Interpret	Question	Propose	Support
Relate	Translate	Write	Criticize	Design	Estimate
Reproduce			Test	Write	Evaluate



Lowest learning level — Highest learning level

Table 17.2 Sample lesson plan*

Session 1: Title, e.g. 'Current dietary recommendations'

- ***Audience***: adults in workplace setting
- ***Numbers expected*** = 20
- ***Duration of session***: 1 h
- ***Aim***: (general statement of intent): to explain dietary recommendations in food terms
- ***Learning outcomes***
  - State the 8 nationally agreed guidelines for a healthy diet
  - Describe the 5 food groups in the healthy eating food model of 'The Eatwell Plate'
  - List the healthier 'types' of these foods
  - Explain the reasoning for the current dietary guidelines in relation to health
  - Identify practical examples to help meet the dietary recommendations
  - Identify 6 factors that influence their food choice

Time	Activity	Resources
3 min	Introduction—plan for the session	Verbal and on the board
7 min	Nutrition and diet quiz: distribute quiz and ask participants to complete	Quiz handout
15 min	Go through the answers to the quiz to stimulate discussion:	
	Q1: Scientific reports and dietary recommendations	Slide1 Slide 2
	Q2: Dietary Reference values and terminology	Slide 3
	Q3: Public beliefs and attitudes to healthy eating	Slide 4
	Q4: Public views on health information	Handout
	Distribute quiz answers	
10 min	Healthy eating food model	Poster
	Food model leaflet and verbal explanation	
10 min	Practical examples of meeting recommendations	Case studies
10 min	Factors influencing food choice	Brainstorm
	Line exercise on white board, and feedback	Practical exercise
5 min	Sum up—back to original overhead showing plan	

*More information on teaching skills. Rogers, A. & Horrocks, N. (2010). *Teaching adults*, 4th edn. Oxford University Press, Oxford.

Designing nutrition education materials

When writing a diet sheet, a report, or patients' notes, a practitioner is intent on transmitting information to the reader. How well the practitioner succeeds will depend on several factors, including the readability of the text.

Readability

Readability is concerned with the problem of matching between reader and text. There is often a large discrepancy between readability and the average patient's reading ability. An accomplished reader is likely to be bored by simple repetitive texts. A poor reader will soon become discouraged by texts that s/he finds too difficult to read fluently.

A typical readability index uses an average sentence length and average number of words of 3–4 syllables per sample used (>200 tests exist). Tests do not account for the order of words in a sentence.

A suitable diet sheet/patient information needs to match the reading age of the general public (average range of 9–11 years).

Example of a readability test (Gunning 'FOG')

Select samples of 100 words, normally 3 such samples from 1 piece of writing.

- Calculate *L*, the average sentence length (number of words ÷ number of sentences). Estimate the number of sentences to the nearest tenth, where necessary.
- In each sample, count the number of words with 3 or more syllables. Find *N*, the average number of these words per sample. Then the grade level needed to understand the material = $(L + N) \times 0.4$.

So the reading age = $[(L + N) \times 0.4] + 5$ years.

Besides readability, the overall structure and presentation, organization of text, and vocabulary used are important when evaluating the quality of nutrition education materials. See Box 17.1 for a checklist for evaluating patient information.

Box 17.1 Suggested checklist for evaluating the quality of educational materials for patients and the general public

Content

- Is there a clear description of the purpose and structure of the text?
- Are headings present? If so, are they appropriate?
- Are the facts correct and up to date?
- Is the quality and strength of the evidence discussed?
- Does the material take account of current government policy?
- Are nutrition messages clear, unambiguous, and consistent throughout?
- Are any statements about nutrition placed within the context of a healthy lifestyle and based on the 'Eatwell Plate'?
- Is the source of information given (e.g. nutrition and dietetic service, British Medical Association)?
- If the use of a branded product can be justified in terms of helping users to identify types of products, is the use sparing and in a relevant context?
- Are references to product names used as examples only, so that single products are not favoured over others?
- Where there is reference to particular foods, are generic groupings used?
- Have contact numbers, addresses, or websites been given for further information, e.g. self-help groups?
- For commercial literature, are logos and brand names used sparingly and in context?
- Is the date of production included?

Is the material appropriate for the intended audience?

- How technical is the vocabulary in the text? Are all acronyms and jargon explained?
- How readable is the text?
- Is it clear who the writer and intended audience are?
- Have users been consulted? Have materials been pretested for comprehension?
- Is the visual layout satisfactory, e.g. layout, font size, large enough for intended audience, visual appeal?
- Is the information adapted for the socio-cultural characteristics of target audience in terms of language and food habits?

Communication and counselling skills

Practitioners use their communication skills to inform, educate, and facilitate motivation in others by enabling patients to make informed choices about food and lifestyle. Practitioners need to draw on a repertoire of '*helping*' skills that enable them to encourage behaviour change. There are a number of theoretical models underpinning this way of working, but essentially it is based on a 'person-centred model' where practitioner and patient agree the plan of action.

Essential practitioner characteristics

A number of specific skills and qualities need to be practiced to improve the practitioner's ability to listen, respond, and reflect empathetically.

- *Unconditional acceptance* accepting and respecting patients, not judging.
- *Congruence* genuine, being sincere and not defensive.
- *Empathy* understanding a patient's personal meanings.
- *Open questioning style* (see Table 17.3)
- *Good listening skills* (see Table 17.4): appropriate use of active versus passive listening.
- Awareness of the effect of *non-verbal communication*: posture, gestures, appearance, voice, eye contact, and facial expressions.
- A *client-centred approach* to give more control to the patient, rather than the practitioner; build on the patient's expertise about him or herself.
- Recognize that the patient has the *right to decide* if s/he wants to change behaviour or not.

Value of counselling skills for motivating dietary change

- Helps establish the current scenario.
- Explores preferred situation and sets appropriate goals with the patient.
- Provides a supportive relationship that may improve adherence to dietary change.
- Encourages the patient to be more 'empowered' to make appropriate choices.
- Helps patients to explore their feelings about changing their eating behaviour.
- Assists patients in the selection of problem-solving strategies to encourage dietary change.

Table 17.3 Asking questions: open vs. closed questioning

Open questions	Closed questions
Include 'what', 'how', 'where', and 'when'	Invite a monosyllabic response, such as 'yes or 'no'
e.g. *Tell me about where you do your shopping?*	e.g. *Have you been to the supermarket this week?*
Avoid 'why' questions as patients can become defensive	
Encourage patients to talk in more depth	Hard to establish empathy. Can be useful for clarifying information.
Help the patient keep the control, rather than the helper	The dietitian/helper retains control over the interview

Table 17.4 The process of listening

Active	Passive
Dynamic process includes using:	Sit back whilst the patient talks.
'Minimal encouragers (e.g. 'go on', 'uh-huh', and allowing silence)	
Paraphrasing (repeating back the general content of what was said).	
Reflection (feeding back to show patients that they have been understood)	
Summarizing (condense the substance of what was said).	
Giving full attention (attending): face the patient squarely; adopt open posture; lean towards patient; maintain eye contact	Full attention is not given; may start to think about something else
Boredom less likely for practitioner	Practitioner can feel bored or irritated
The patient feels valued	The patient feels undervalued

Health behaviour models

Various models of health behaviour have been developed that make an important contribution to understanding dietary behaviour. They acknowledge that dietary 'knowledge' alone is not necessarily enough to lead to behaviour change. Health behaviour models conceptualize the social cognitive variables important in determining behaviour. Such models focus on the idea that the attitudes and beliefs a person holds can influence their dietary behaviour.

They include:

- Social cognition theory[1] that assumes that behaviour is governed by expectancies, incentives and social cognitions.
- Theory of planned behaviour[2] which emphasizes behavioural intentions as the outcome of several beliefs (attitudes to the behaviour, subjective norm, and perceived behavioural control).

They take account (in different ways) of:

- whether individuals are fatalistic about their health (locus of control);
- whether individuals feel able or confident to change certain aspects of health behaviour (self-efficacy);
- the value individuals place on their health (health as a valve);
- the stage in which individuals can be placed in relation to their health behaviour (stage of change).

Further information can be found in Ogden (2007).[3]

See 🕮 Chapter 21 'Management: behaviour change strategies', p. 420.

Stage of change

One model that is widely used to understand health-related behaviour change is the 'transtheoretical model of change'.[4] This model regards individuals as traversing through 5 phases (Fig. 17.3 and Table 17.5). Movement through the stages is not necessarily linear; individuals may relapse to earlier stages several times, but this does not necessarily mean that they are starting at the beginning. The practitioner's role is to facilitate a natural process of change, matching the support given to the stage where the patient is. It is often difficult to pinpoint which stage a person is in and indeed they can move from stage to stage during the course of an interview, whereas others get 'stuck' in a particular stage. Therefore, the model should only be used as a guide.

[1] Bandura A (1977) Self efficacy: towards a unifying theory of behaviour change. Psychological Review. **84**: 191–215.

[2] 🖱 On the Theory of Planned Behaviour: 🖱 www.people.umass.edu/aizen/tpb.html.

[3] Ogden, J (2007)3 Health Psychology—a textbook. 4th edn. OUP.

[4] Prochaska, J.O. and DiClemente, C.C. (1986). The transtheoretical approach. In the Handbook of eclectic psychotherapy (ed. J.C. Norcross), pp.163–200.Bruner/ Mazel, New York.

Table 17.5 Stage of change and associated practitioner role

Stage	Practitioner role
Pre-contemplation Not interested in change or 'in denial or immune to their health problems'	Elicit open discussion to assess how the current situation is perceived and to highlight health risks about their unhealthy behaviour to encourage the possibility of change
Contemplation Ambivalent or thinking about change	Help the patient weigh up the pros and cons of changing compared with those for not changing
Preparation Preparing for change	Support in developing their action plan for change. Suggest or give educational material
Action Actively modifying habits or environment	Helping the patient to set clear, realistic goals including rewards. Praise action taken by patients
Maintenance Sustaining new, healthier habits and preventing relapse *Relapse*	Reassuring patients that occasional 'lapses' are normal, so that they are not so discouraged that they give up. However, most patients will go through the stages of change several times before changes become established

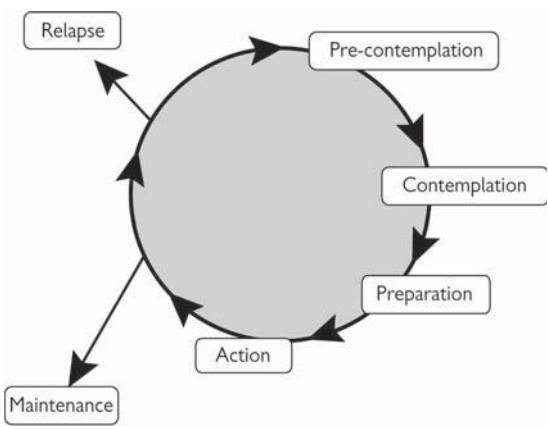


Fig. 17.3 The 'stage of change' model of Prochaska and DiClemente.

How do you know which stage a patient is in?

Assignment of individuals to one of the stages may be done intuitively during discussion with patients or more formally by asking a limited number of mutually exclusive questions and applying a basic algorithm (Table 17.6). However, it is important that assignment of individuals to a particular stage does not get in the way of the helping process. The focus should be on the interpersonal relationship. For example,

- *Q1:* In the past month, have you been actively trying to lose weight? Yes/No.
- *Q2:* In the past month, have you been actively trying to keep from losing weight? Yes/No.
- *Q3:* Are you seriously trying to lose weight to reach your goal weight in the next 6 months? Yes/No.
- *Q4:* Have you maintained your desired weight for more than 6 months? Yes/No.

Table 17.6 Scoring for stage of change

Stage	Q1	Q2	Q3	Q4
Pre-contemplation	N	N	N	
Contemplation	N	N	Y	
Action	Y			N
Maintenance	Y			Y

Motivational interviewing

Motivational interviewing (MI) is a client-centred, directive method for enhancing intrinsic motivation to change by exploring and resolving ambivalence. It is not just a set of techniques, but relies heavily on good reflective listening skills. A large systematic review assessing the evidence regarding behaviour change theories in relation to diet, found strong evidence that a combination of behavioural theory and cognitive behavioural theory (foundation for cognitive behavioural therapy (CBT)) facilitates dietary change[1]. MI combined with CBT was shown to be highly effective. CBT assumes that behaviour is learned and can therefore be unlearned using cognitive and behavioural strategies that focus on both external (environmental stimulus, reinforcement) and internal (thoughts) factors.

Decisional balance Decisional balance is used as a tool to enhance motivation to change. Thinking through the pros and cons of both changing and not making a change is one way to help patients make sure they have fully considered a possible change. This can help them to 'stick' with their plan in stressful/tempting moments. Patients could be asked to reflect on for example, the pros and cons of making changes to lose weight, (see Table 17.7).

Table 17.7 One patient's pros and cons of making changes to lose weight

Benefits/pros of making changes	*Benefits/pros of not changing*
• Feel better about myself, more confident. • Fit into all those clothes hanging in the wardrobe! • Save money as will be eating less. • Help control my blood pressure.	• Not having to change family's routine. • Partner likes me chubby. • Still being able to eat lots of tasty food.
Cons of making changes	*Cons of not changing*
• Will miss tasty food. • Will be hungry at first. • Partner might not like it if I am too slim.	• Health: could develop diabetes in the future. • Blood pressure might get worse. • Will carry on feeling a bit of a failure due to being self-conscious about my weight.

[1] Spahn, J.M., Reeves, R.S., Keim, K.S., *et al.* (2010). State of the evidence regarding behaviour change theories and strategies in nutrition counselling to facilitate health and food behaviour change. *JADA*, **110** (6), 879–891.

Planning change

Box 17.2 A change plan for patient wanting to lose weight

Once a patient has made the decision to change, a change plan can be constructed with the help of the practitioner.

The changes I want to make are:
- List specific areas or ways in which you want to change
- Include positive goals (beginning, increasing, improving behaviour), *e.g. have a healthy snack mid-afternoon so that I eat less food when I return home from work; eat smaller quantities at mealtimes*

The most important reasons why I want to make these changes are:
- What are some likely consequences of action and inaction?
- Which motivations for change seem most important to you, *e.g. to feel better about myself, more confident*

The steps I plan to take in changing are:
How do you plan to achieve the goals? Are there some specific first steps you might take? When, where and how will these steps be taken, *e.g. have a shower/drink of tea when I get in from work to avoid 'picking' before mealtime; weigh out food quantities at first so I can see what I am aiming for to be able to lose weight*

The ways other people can help me are:
List ways that others can help support you in changing. How will you go about getting others' support, *e.g. Ask my partner not to eat snacks in front of me or cook high calorie meals*

I will know that my plan is working if:
- What do you hope will happen as a result of the change?
- What benefits can you expect from the change, *e.g. Lose weight, around 1 kg a week*

Some things that could interfere with my plan are:
- Anticipate situations or changes that could undermine the plan.
- What could go wrong? How might you stick with the plan despite the changes or setbacks, *e.g. lack of self-control, feeling hungry and tired; could have a smaller quantity of the food I am trying to resist or plan what I want to eat beforehand so that I know I can't have the extra nibbles*

Further information on behaviour change

Gable, J. (2007). *Counselling Skills for dietitians.* 2nd edn. Blackwell, Science, Oxford.
Norcross, J.C., Prochaska, J.O., and DiClemente, C.C. (2010). *Changing for good: a revolutionary six-stage program for overcoming bad habits and moving your life positively forward.* Kindle edn, *New York.*
Rollnick, S., Miller, W.R., & Butler, C.C. (2008). *Motivational Interviewing in health care: helping patients change behavior (applications of motivational interviewing)*, 3rd edn. Guilford Press, New York.
Pearson, D. & Grace, C. (2011). *Weight Management: A Practitioner's Guide.* John Wiley and Sons Ltd, Chichester.

Chapter 18

Nutrition intervention with populations

National food and nutrition policy

The major causes of mortality and morbidity in the UK are cardiovascular disease (CVD), type 2 diabetes, and cancer; all have a strong nutritional aetiology. Both public policy and direct contact with patients present opportunities to reduce risk factors associated with the major non-communicable diseases (poor diet, physical inactivity, smoking, and obesity). Strategies for preventing these diseases are developed at a national and local level in the UK.

National bodies influencing UK food and nutrition policy

Food Standards Agency

Although the Food Standards Agency (FSA) is a government agency, it works 'independently' of the government as it does not report to a specific minister. However, it is accountable to parliament via health ministers, and to the devolved administrations of Scotland, Wales, and Northern Ireland for its activities within these areas.

Further information is available at: www.food.gov.uk.

Main roles since 2010

- Reduce food-borne illness by improving food safety throughout the food chain.
- Improve the enforcement of food law.

Since 2010 the FSA's role changed and the DH (see 📖 p. 353) became responsible for nutrition policy in England, and the Department for Environment, Food and Rural Affairs (DEFRA) for food labelling not related to food safety and food composition policies in England.

The Scientific Advisory Committee on Nutrition

In the past (from 1963 to 2000), the government relied on expert advice on nutrition from the Committee on Medical Aspects of Food and Nutrition (COMA) Policy, which produced a series of reports that were used to inform nutritional policy. Since the establishment of the FSA in 2000, the Government replaced COMA with the Scientific Advisory Committee on Nutrition (SACN; www.sacn.gov.uk), which provides independent expertise to the government.

SACN's remit includes advising on:

- nutrient content of foods; defining a balanced diet/nutritional status;
- nutritional problems that impact on wider public health policy, where nutrition is one of several risk factors, e.g. obesity, cancer, CVD, and osteoporosis;
- nutrition of vulnerable groups, e.g. infants, older people, and disadvantaged groups;
- nutrition monitoring and surveillance;
- research needs arising from the above areas.

The Department of Health

Since 2010, the DH is entirely responsible for nutrition policy in England. The DH is responsible for health protection, health improvement, and health inequalities issues in England. One section of the DH is involved in public health. Other sections of the DH coordinate National Service Frameworks (NSFs) and the National Institute for Health and Clinical Excellence (NICE) provides guidance, e.g. NSFs for coronary heart disease (CHD), diabetes, children and young people, and on obesity prevention and management. NICE provides independent guidance on the promotion of good health and the prevention and treatment of ill health (www.nice.org.uk).

There are also many initiatives in devolved administrations:

- Scotland's Health Improvement Agency: www.healthscotland.com/;
- Health in Wales: www.wales.nhs.uk;
- The Department of Health, Social Services and Public Safety in Northern Ireland: www.dhsspsni.gov.uk/.

National public health nutrition strategies

The NHS White Paper—*Equality and Excellence* (2010)—marks a change in the approach to public health for England that may influence the initiatives listed below. (See www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsPolicyAndGuidance/DH_117353.

Initiatives targeting children

In the UK, overweight affects some 30% of all school-age children and is particularly prevalent among children from lower socio-economic status families. To curb the rising trend in childhood obesity, national targets were set in 2004 to halt the year-on-year rise in obesity in children under 11 years, and there does seem to have been a leveling off of obesity prevalence in younger children as obesity fell slightly in this age group to 10.2% (boys) and to 8.9% (girls). However, in children aged 10–11 years obesity prevalence increased marginally from 19 to 20% (boys) and from 15.8 to 16.5% (girls) between 2006 and 2009. For further information see:

www.noo.org.uk/NOO_about_obesity/child_obesity/epidemiology

See Chapter 14, 'National child measurement programme', p. 289.

The school fruit and vegetable scheme

Under the scheme, all 4–6-year-olds in Local Education Authority maintained infant, primary, and special schools are entitled to a free piece of fruit or vegetable each school day, if schools have 'chosen' to participate in the scheme. It was introduced after the NHS Plan 2000 included a commitment to implement a national school fruit scheme by 2004. The School Fruit and Vegetable Scheme is part of the 5 A DAY programme to increase fruit and vegetable consumption. Further information is available at: www.5aday.nhs.uk.

Nutritional standards for school lunches and other school food

In 2006, 617 million school meals were served in England alone, so they represent an ideal opportunity to influence the nutritional quality of children's diets. Each country in the UK (England, Northern Ireland, Scotland and Wales) has developed food and nutrient-based standards for school meals (for more detail see Evans and Harper (2009)[1]). The Government reintroduced compulsory national nutritional standards for school lunches in England in April 2001 and these standards were revised again in 2007 (see www.schoolfoodtrust.org.uk/) to cover school lunches and other school food provision. Northern Ireland and Scotland introduced standards in 2008.

Further information is available from www.education.gov.uk and www.parliament.uk/documents/post/postpn339.pdf.

See Chapter 10, 'Schools', p. 206.

[1] Evans, C.E. and Harper, C.E. (2009). School meals standards in the UK. *J. Hum. Nutr. Dietet.* **22**, 89–99.

The School Food Trust (UK)

The School Food Trust is an independent body with the remit of transforming school food and food skills. It was set up as Non Departmental Public Body in 2005 and is funded by the Department for Education (DfE) to promote the education and health of children and young people by improving the quality of food supplied and consumed in schools. In 2007 the Trust registered as a Charity. The Trust is charged with taking forward School Meals standards in the UK to improve school food and food skills to improve health and education for school age children and young people.

🔗 Further information www.schoolfoodtrust.org.uk/.

The Healthy Schools Programme

The Healthy Schools Programme is a joint initiative between DfE and DH, which promotes a whole school/whole child approach to health. The programme has existed since 1999. It is recognized as a key delivery mechanism in the Children's Plan (DCSF, 2007) and in Healthy Weight, Healthy Lives (DH 2008), a 21st Century White Paper reference to tackle rising levels of childhood obesity, as well as proposing means of improving poor diet and lack of activity generally. The programme has develops a range of nutrition-related activities and projects in schools to complement and add value to the wide range of other initiatives in schools. The aim is to introduce a programme that follows children throughout the school day, using a whole school approach.

🔗 Further information is available at: http://home.healthyschools.gov.uk/ and www.education.gov.uk.

See example in 🕮 this Chapter, 'Model of a local school food policy', p. 374.

Controlling food and drink marketing to children

Controlling food and drink marketing to children is a measure under consideration to combat childhood obesity. The UK currently employs a combination of statutory and government approved private sector self-regulation techniques to control commercial promotion of food to children. Statutory rules apply to child-targeted TV advertisements for high fat, sugar or salt (HFSS) foods as defined by nutrient profiling. 'Child-targeted' marketing is defined as marketing during preschool children's programmes, during programmes made for children <16 years in children's airtime, and youth-orientated programming that attracts a significantly higher-than-average proportion of viewers <16 years (i.e. the proportion of viewers <16 years is 20% higher than the general viewing population). The rules apply to commercial and public service broadcast channels, and all cable and satellite channels.

The rules also included additional measures that banned the use of licensed characters, celebrities, health claims, and free gifts in HFSS food advertisements directed at young children. The debate now concerns non-broadcast media, such as print and digital media, including the internet (see Box 18.1).

Box 18.1 International Code on Marketing of Foods to Children

In 2008 the International Obesity Task Force and Consumers International published proposals for an International Code on Marketing of Foods to Children which sought to emphasize the need to protect children in countries where regulation is weak or enforcement capacity poor by requiring the commercial sector to agree a minimum set of principles, including a ban on:

- TV and radio adverts between 6am and 9pm that promote unhealthy foods;
- use of cartoon characters, celebrities, or competitions promoting unhealthy foods;
- inclusion of free gifts, toys, or items for children to collect in unhealthy foods;
- promotion of unhealthy food products in schools;
- marketing of unhealthy foods using new media, such as the internet and text messages.

The proposed Code has been submitted by a group of international non-governmental bodies as part of the WHO consultation process.

Further information is available at: IOTF and CI (2008). Recommendations for an International Code on Marketing of Foods and Non-Alcoholic Beverages to Children. www.iotf.org/documents/ConsumersInternationalMarketingCode.pdf

Initiatives targeting the whole population

Since 1990 a series of white papers setting national targets for the prevention of obesity and diet-related disease have been produced by the DH for England. Starting with *Health of the nation* (1991) → *Saving lives: our healthier nation* (1999) → *Choosing health: making healthier choices easier* (2004) → *Healthy Weight, Healthy lives* (2008).

Current national strategy documents are the following:

- *Choosing Health: making healthier choices easier* (DH, 2004). Outlines how health professionals can provide advice on disease prevention.
- *Delivering choosing health: making healthy choices easier* (DH, 2005). Sets out objectives on major health issues, including those relating to nutrition policy.
- *Choosing a better diet: a Food and health action plan* (DH, 2005). The DH has worked across government, with the food industry, and other stakeholders to establish a national plan to help people in England improve their diets. Available at: www.bda.uk.com/Downloads/ChoosingBetterDiet.pdf
- *Choosing activity: a physical activity action plan* (DH, 2005).
- '*Tackling health inequalities: a programme for action*' laid the foundations to reduce the health gap on infant mortality and life expectancy.
- *Healthy Weight, Healthy lives* (2008) to tackle obesity. Available at: www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsPolicyAndGuidance/DH_082378

- Food Matters (2008) integrates the impact of food on the environment. Available at: www.cabinetoffice.gov.uk/media/cabinetoffice/strategy/assets/food/food_matters1.pdf
- *Change4Life campaign*, which uses social marketing to change behaviour in relation to nutrition and physical activity initially in families with children aged five to 11 years; followed by adults aged 45–65 years. It is the marketing component of the Government's 'Healthy Weight, Healthy Lives' strategy and involves partnership with the food industry, which has been controversial. Available at: www.dh.gov.uk/en/Publichealth/Change4Life/index.htm

Front of pack food labelling scheme. The EU parliament voted for guideline daily amounts (GDAs) in 2010, which were proposed by the food industry, rather than the more consumer-friendly traffic light scheme, which had been advocated by the FSA, and many consumer and health promotion groups. From 2010 responsibility for food labelling shifted from the FSA to DEFRA in the UK.

Further information on food labelling is available at: ww2.defra.gov.uk, www.dh.gov.ukw, and www.bhf.org.uk/keeping_your_heart_healthy/top_tips_for_keeping_healthy/understanding_food_labels.aspx

Weight Wise campaign

Weight Wise is a nutrition campaign co-ordinated by the British Dietetic Association to increase awareness of the benefits of a balanced and varied diet in achieving and maintaining a healthy weight. It started in June 2002 and is supported by an interactive website www.bdaweightwise.com, which provides free information and support for anyone wanting to reach a healthier weight.

Further reading

Caroline Walker Trust (2009). *Nutrition policy across the UK*, briefing paper. Available at www.cwt.org.uk

Surveys to monitor nutritional status in the UK

Household food consumption surveys

An example is the National Food Survey. In the UK, the National Food Survey (NFS) was established in the 1940s to monitor the diet of the urban 'working class' population during the war years. It was extended to cover all households in the general population in the 1950s and to collect data on food expenditure and consumption. From 2001, the Family Expenditure Survey (1961–2001) and the NFS (1974–2000) were replaced by a new combined survey, the Expenditure and Food Survey (EFS). From 2008, the EFS became the Living Costs and Food Survey (www.esds.ac.uk/findingData/efsTitles.asp). Each year about 8000 households take part in the survey. The household member who does most of the food shopping is asked some questions about the household and its food purchasing. They are then asked to keep a diary for 7 days, recording food coming into the household, including quantities and expenditure and some detail of the household meals (including snacks and picnics prepared from household supplies).

Individual dietary surveys

The National Diet and Nutrition Survey (NDNS) rolling programme is a continuous cross-sectional survey of the food consumption, nutrient intakes, and nutritional status of people aged 18 months and older living in private households in the UK. It covers all 4 countries of the UK and is designed to be representative of the UK population.

The NDNS is carried out by a consortium of 3 organizations: the National Centre for Social Research (NatCen), Medical research Council (MRC) Human Nutrition Research (HNR) and University College London (UCL) Medical School.

The report of the first year of the NDNS rolling programme (February 2008 to March 2009) can be downloaded at www.food.gov.uk/science/dietarysurveys/ndnsdocuments/. This report focuses on food consumption and nutrient intakes for adults aged 19–64 years and for children aged 1.5–3 years, 4–10 years, and 11–18 years. Intakes are compared with government recommendations and comparisons with findings from previous surveys are also made.

The results of the survey are used to develop nutrition policy and to contribute to the evidence base for Government advice on healthy eating. Also see 🕮 Chapter 3 'Current dietary patterns', p. 30.

Health Survey for England

The Health Survey for England comprises a series of annual surveys beginning in 1991. The series is part of an overall programme of surveys commissioned by the DH and designed to provide regular information on various health topics in England for adults >16 years and children since

1995, including infant feeding practices. A number of core questions are included every year, but each year's survey also has a particular focus on a disease or condition or population group. Topics are brought back at appropriate intervals in order to monitor change. The survey combines questionnaire-based answers with physical measurements and the analysis of blood samples. Blood pressure, height and weight, smoking, drinking, and general health are covered every year. An interview is conducted with each eligible person in the household.

Dietary data are qualitative and based on frequency and type of foods consumed with a food record or questionnaire. Weight and height is usually measured, and additionally waist and hip circumference in more recent surveys.

Since 2001, the survey covers all ages. Information is obtained directly from persons aged ≥13 years. But, information about children aged <13 years is obtained from a parent, with the child present.

Further information is available at: http://www.esds.ac.uk/government/hse/.

Policy options for preventing obesity

It is widely recognized that preventing obesity requires more than a health or medical perspective; it needs to be viewed from a wider societal and economic context that involves partnership working on a population-wide basis, as obesity touches all of society (Box 18.2).

There is a wealth of potential environmental level strategies for the prevention of obesity, as highlighted by the World Health Organization.[1]

The Foresight report 'Tackling Obesities: Future Choices (2007)[2] noted that although the environment has a role, alone these are unlikely to solve the problems of increasing obesity and declining physical activity levels. They recommend that a range of complementary strategies are needed that address the individual, social and environmental determinants of behaviour. See 🖱 www.bis.gov.uk/foresight/our-work/projects/current-projects/tackling-obesities.

Box 18.2 Key stakeholders for obesity prevention

Developing strategies that will involve structural change to improve the obesogenic environment will require the participation of a range of key stakeholders, for example:

- Governmental (departments of: health; nutrition; food; food and agriculture; transport; education; family and social care; advertising control).
- Food production system (producers; farmers; unions; food industry representatives; large retailers: supermarkets; small retailers; catering).
- Health systems (pharmaceutical industry, general practitioners; specialist medics, dietitians; nutritionists, nurses, health promotion specialists).
- Media (advertisers, newspapers, TV and radio, female press).
- Education system (pre-school care, schools and colleges, universities).
- Workplace (unions, large vs. small institutions).
- NGOs (consumer associations, marginal group associations, low income associations, family groups).
- Local communities.

Possible policy options for obesity prevention

Laws and regulations

- Mandatory nutritional information labelling for energy-dense food.
- Controls on the advertising and promotion of food and drink products, particularly to children. See 🕮 'Controlling food and drink marketing to children', p. 355.
- Agricultural policy reform to encourage production of cheaper fruit and vegetables.
- Incentives to improve nutrient composition of processed foods.

[1] World Health Organization (2004). *Global strategy on diet, physical activity and health.* Geneva: WHO. Available at: www.who.int/dietphysicalactivity/en/.
[2] Foresight report (2007). *Tackling obesities: future choices.*

Transport policies and town planning

- Provide improved facilities for walking and cycling.
- Improve conditions for pedestrian travel to school.
- Plan for the use of streets as social spaces rather than just for cars.
- Improve public transport.

Economic incentives

- Tax changes to alter patterns of food consumption and to reduce consumption of energy-dense foods.
- Public subsidies on healthy foods to improve patterns of food consumption.
- Reduce car tax for those who use public transport during the week.

Food and catering standards

Develop nutrition standards and guidelines for institutional catering services, e.g. school meals, workplaces, prisons.

Food production

Encourage use of land in urban areas for growing fruit and vegetables for use by households, e.g. allotments.

Promotion of healthier behaviour

- Improve training for health professionals in obesity prevention, and those involved in diagnosing and counselling those at risk of obesity.
- Use the media to promote positive behaviour.
- Educate the public about the main causes of obesity so that stigmatization of the obese is reduced.
- Raise awareness in the general public about the need for collective action to improve the environment to one that encourages rather than discourages healthy behaviour.

Schools

- Encourage training in practical food skills for children.
- Ensure provision of healthy, tasty school meals.

See 🕮 this Chapter, 'Model for a local obesity strategy', p. 375.

Further information: obesity prevention

Delpeuch, F., Maire, B., Monnier, E., and Holdsworth, M. (2009). *Globesity—a planet out of control. European Union: Diet, Physical Activity and Health—EU Platform for Action.* Available at: http://ec.europa.eu/health/nutrition_physical_activity/platform/index_en.htm.

Earthscan Books, London. http://www.earthscan.co.uk/?tabid=56997

European Childhood Obesity Group (ECOG) . Available at: www.ecog-obesity.eu/

International Obesity Task Force. Available at: www.iotf.org/WHO. Available at: www.who.int/dietphysicalactivity/en/

Jain, A. (2004). Fighting obesity: evidence of effectiveness will be needed to sustain policies. *Br Med. J.* **328**, 1327–8.

Kremers, *et al.* (2009). Systematic prevention of overweight and obesity in adults: a qualitative and quantitative literature analysis. *Obesity Rev.* **11**, 371–9.

Kumanyika, S., Jeffery, R.W., Morabia, A., *et al.* (2002). Obesity prevention: the case for action. *Int J Obesity* **26**, 425–36.

National Heart Forum (2007). *Lightening the load: tackling overweight and obesity. A toolkit for developing local strategies to tackle overweight and obesity in children and adults*. Faculty of Public Health, London. Available at www.fphm.org.uk/resources/AtoZ/toolkit_obesity/obesity_toolkit_intro.pdf

National Obesity Observatory, which provides a single point of contact for data, evaluation and evidence related to weight status and its determinants. Available at: www.noo.org.uk.

Sustrans is sustainable transport charity based in UK and works to encourage people to walk, cycle, and use public transport to benefit health and the environment. Available at: www.sustrans.org.uk/assets/files/AT/Publications/PDFs/FH14_activetravel_and_obesity.pdf

Local food and nutrition policy

See Box 18.3 for the process of developing food policies.

Box 18.3 Process of developing national and local food policy

Scientific link between diet and health and disease
⇓
Assessing diet and nutritional status of population
⇓
Development and implementation of national government policies
Government's political philosophy → ⇓ ← Lobbying from different stakeholders
Development and implementation of local policies based on national priorities and those of concern locally

Translating government policy into local action

Local (regional/city-wide) food and health policies were initiated in the UK in the early 1980s in response to growing consensus about the role of diet in the prevention of disease and by the late 1980s most health authorities had their own policy. These usually contained a food safety component and promoted the nutrition education messages emerging at that time (less fat, more fibre, and less sugar), primarily in NHS catering settings, although some had community activities.

Publication of national public health strategy documents (see 📖 this Chapter, 'National food and nutrition policy', p. 350) stimulated the production of local health plans from a range of authorities and organizations, which widened the focus from NHS settings to multi-sectorial partnerships within the community. This meant that wider perspectives on nutrition evolved incorporating issues such as food production, biodiversity, and inequality and food access (see Fig. 18.1 and Box 18.4). Although the nutrition messages relating to macronutrients are still relevant, there has been a shift towards encompassing nutritional adequacy.

Box 18.4 The role of local food and nutrition policy

- To provide a political mandate and support for implementation
- To make available a scientifically accurate document of what constitutes a 'healthy diet' to a wide audience
- To encourage consistent dietary messages by health professionals and other agencies who have a role in nutrition education
- To ensure, by collaboration with other local agencies and employers, that it is easier for citizens to eat a healthy, sustainable diet
- To use a population-wide approach to try and shift the distribution of risk factors for diet related non-communicable disease
- To develop strategies that makes healthier behaviour the 'social norm' in key settings therefore lowering risk in the whole population
- To provide nutritional advice for certain groups within the population who have specific nutritional needs, e.g. children, older people
- To facilitate a wider choice of foods within and outside the NHS.

Fig. 18.1 Example of the wide scope of a holistic, sustainable local food policy. (Reproduced with kind permission of FIG, Greater Nottingham.)

The Following list expands on the points listed in Fig. 18.1.

- *Food, health, and nutrition:* to improve the diet of local citizens by providing consistent and appropriate nutrition messages and addressing the potential barriers to achieving a healthier diet.
- *Food and mental health:* to develop awareness of the emotional and psychological dimensions of food growing and consumption.
- *Organic production:* to stimulate expansion of production and consumption of locally-produced organic food.
- *Genetically-modified food:* to raise awareness about the different perspectives that exist regarding genetically-modified organisms.
- *Food safety*: to ↓ cases of food and water-borne diseases.
- *Waste and composting:* to ↓ the amount of waste associated with production, consumption, and food sent to landfill or incineration.
- *Animal welfare:* to promote high standards of animal welfare.
- *Inequality and food access:* to address issues of social exclusion and ensure people on a low income have access to healthy, affordable food.
- *Local food:* to increase the local consumption of locally produced food.
- *Global perspective:* to raise awareness of the effects of the world trade system on small farmers in the developing world.
- *Food production and biodiversity:* to encourage methods of food production that protects and enhances biodiversity.

See 🕮 Chapter 19, 'Sustainable diets', p. 379.

Useful websites

- www.sustainweb.org (Sustain: The alliance for better food and farming)
- www.groundworkgreaternottingham.org.uk/fig (The Food Initiatives Group).
- www.invo.org.uk (national advisory group funded by the DH aims to promote and support active public involvement in the NHS and health-related local policy development).

Conducting nutrition interventions

See Box 18.5 for the steps involved.

Box 18.5 Steps in conducting nutrition interventions

STEP 1 Defining the problem

- Needs assessment of nutrition priorities: scale of the problem using epidemiological and sociodemographic information
- Analyse possible modifiable determinants, e.g. environmental, behavioural, economic, and social
- Identify the main partners from the planning stage
- Assess community needs and perceived priorities
- Define measurable short and long term nutrition and health goals.

STEP 2 Generating solutions

- Aim for inter-sectorial working with a range of stakeholders
- Use tacit knowledge (know-how) from practitioners
- Review and refer to explicit knowledge sources, i.e. research and evidence based guidelines to build on theoretical principles
- Use participatory appraisal methods with the local community to develop sustainable solutions.

STEP 3 Building capacity

- Develop institutional framework to act and identify support structures
- Mobilize resources needed: funding, key actors, materials
- Training and infrastructure development
- Raise public and political awareness
- Define the management and operational roles of different partners.

STEP 4 Implementing the intervention

- Elaborate an action plan (strategies and activities).
- Verify that there are a range of complementary interventions on:
  - *education*, e.g. media, educating patients, school education
  - *social mobilization*, e.g. community development, mass communication to influence social, environmental and economic factors that influence health
  - *advocacy*, e.g. lobbying for political/organizational/structural change
- Monitor using process measures to assure quality in practice, allow feedback on implementation, participant and practitioner response.

STEP 5 Evaluating if the programme meets its objectives

- *Health promotion outcomes:* include health literacy (knowledge, attitudes, and beliefs, self-efficacy); social action (community participation, social norms, public opinion); and healthy public and organizational policy (policy statements, legislation, resource allocation and organizational practice)
- *Intermediate health outcomes:* include healthy behaviour (food consumption), healthy environments (urban planning to encourage physical activity), and effective health services (provision, access)
- *Health & social outcomes:* long-term goals of ↓ morbidity, e.g. ↓ obesity, cardiovascular disease, and ↑ quality of life and equity.

Case study of a local food and health policy

Case study: Leicestershire food and health plan

The development, implementation, monitoring, and evaluation of a food and nutrition policy within a local health plan in Leicestershire.

Development

To develop an action plan to target people at increased risk of poor nutrition, i.e. lower income groups, minority ethnic groups, and generally increase public awareness of healthier eating. A 'Healthy Eating Group' was established, led by the county's Nutrition and Dietetic Service with membership drawn from a wide range of agencies, i.e. local authorities, universities, school meals service, community health groups, community dental service, public health medicine, health promotion service (physical activity), primary care development, voluntary organizations, occupational health, and environmental health. The group's remit was to develop a public health nutrition strategy by devising a 3-year action plan that would work towards achieving the national public health nutrition targets.

Implementation

The action plan illustrated in Table 18.1 describes how the policy was executed, monitored, and evaluated in a range of settings. This was based on describing current nutrition interventions and prioritizing additional interventions for funding. Further resources were required for many of these activities to be carried out, and the priorities for funding were identified and sought within and outside of the local region including the National Lottery. All of the new projects funded had in-built evaluation and none of them could have evolved as they did without working in partnership with several of the agencies represented on the Healthy Eating Group.

Monitoring and evaluation

Methods for monitoring and evaluation were integrated into the implementation strategy (Table 18.1). A combination of quantitative and qualitative research techniques was used to assess programme effectiveness (process, impact, and outcome measures). The evaluation of food policy implementation is based on assessing the effectiveness of interventions in meeting their objectives, e.g. changing attitudes and behaviour, rather than focusing solely on health outcomes relating to disease incidence.

A profile of possible indicators of success and a data collection system that fits into the existing framework of activity were developed. One of the related challenges is how to demonstrate the validity of programmes. It may be that a programme on its own is insufficient but that the 'community of action' is effective in increasing the likelihood of change.

Table 18.1 Example for implementing a local food and nutrition policy for preventing nutrition-related chronic disease by setting and target group

Strategy/action	Partners	Indicators of success
Schools: pupils, teachers, parents, governors		
• Fruit in tuck-shops • Healthy food awards • School nutrition action groups • School meals liaison working group • School nurse support and training • Production of nutrition resources • Nutrition in taught curriculum • Developing breakfast clubs • National School Fruit scheme • Cooking skills clubs	• Teachers • Parents • Governors • Pupils • School nurses • Dietitians • Health promotion officers • School meals service • County Council	• ↑ in nutritional quality of school meals using audit • ↑ number of secondary schools with healthy food scheme • ↑ number of schools with nutrition policy • ↑ uptake of healthy choices at breakfast clubs.
General public		
• Public exhibitions • Healthy food awards • Using local media, e.g. radio, TV • Availability of healthy food at retail outlets • Train local people to work in their communities to promote healthy eating • Develop 'cook and eat' sessions • Nutrition education in adult leisure classes	• Health promotion officers • Health professionals • Dietitians • Public interest • Local media • Environmental health officers • Community food workers	• Review attendance profile at public exhibitions • ↑ number of healthy food awards in establishments for the public • ↑ media contacts • ↑ availability of healthy food at retail outlets • ↑ number of food workers trained

(Continued)

Table 18.1 *(Cont'd)*

Strategy/action	Partners	Indicators of success
• Support local allotment schemes for fruit and vegetable production • Community weight management classes		
Workplace: all employees		
• Promotion of healthy food awards • Weight management groups	• Managers, employees • Dietitians, catering managers • Environmental health officers • Health promotion officers • NHS Trusts	• ↑ number of healthy food awards • Evaluating if the scheme changes attitudes and eating habits of target groups
Hospitals: staff and patients, patient groups		
• Better hospital food programme • Healthy food award for patient and staff meals • Support nutritional guidelines in hospital meals	• Dietitians • Catering staff • Hospital management • WRVS	• Audit of nutritional value of delivered meals • Audit of food policy promotion and awareness in hospitals
Primary health care: patients, primary health care teams (PHCTs)		
• Diet sheets and infant feeding policy • Training PHCTs • Production of nutrition resources for patients • Promotion of food policy to patients	• GPs, practice nurses, health visitors, school nurses, district nurses, dietitians, dental practitioners	• ↑ number of practices working to minimum nutrition standards • ↑ number of trained PHCTs • ↑ nutritional knowledge and practice of PHCTs • ↑ uptake of nutrition resources

Homeless and those living in bed and breakfasts and facilities for the homeless		
• Practical nutrition resources • Nutrition information pack for homes providing meals • Developing nutritional standards for meals provided	• Dietitians • City Councils • Health promotion officers • Staff at hostels and bed and breakfasts	• ↑ nutritional quality of meals provided • ↑ nutritional knowledge of catering staff and home leaders
Social services and residential homes, e.g. elderly, learning disabilities		
• Food policy for older people • Community mental health team nutrition pack • Lunches at day care centres • Training carers, e.g. Age UK • Nutrition package to social services/residential homes for healthy food award	• Carers • Dietitians • Social services • Residents • Private home care • Mental health dietitians • Community mental health teams	• ↑ nutritional quality of meals • ↑ nutritional knowledge of carers • ↑ uptake of appropriate nutrition resources • ↑ number of key workers trained • ↑ number of homes applying for healthy food award
Under 5s at day nursery, playgroups, and childminders		
• Nutrition policy for <5s • Training health professionals and nursery staff • Advising on meals at nurseries • Nutrition guidelines for nurseries	• Health visitors • GPs • Midwives • Dietitians • Day nursery staff	• ↑ number of key workers receiving training • ↑ knowledge of workers related to nutrition of <5s • ↑ quality of meals at day nurseries

Tips for implementing and evaluating local food and nutrition policy

Box 18.6 Tips for implementing and evaluating local food and nutrition policy

- *Good management:* a named individual is responsible for co-ordination
- Clear objectives have to be defined, even though on-going monitoring may mean these evolve as the programme develops
- The policy should reflect local priorities and political structures
- Use a population-wide approach to try and shift the distribution of risk factors for diet-related non-communicable disease
- Develop strategies that help make healthier behaviour into the social norm, ∴ lowering risk in the whole population
- Develop sustainable solutions by combining know-how from practitioners, evidence-based practice, and local community participation
- Use an inter-sectorial approach, as the combined contribution becomes more than the sum of the contributions of individual agencies
- Priorities for funding for new projects should be identified by a multidisciplinary group if they are to represent community needs
- Awareness of the pressures and changes occurring in other organizations that may affect policy implementation is essential
- Identify barriers locally to healthier living and support local action to overcome them
- Link local action to national public health programmes
- Monitoring techniques need to be realistic and flexible enough to evolve with programmes
- Include means of measuring how effective the intervention is to optimize use of resources and assess the quality of programmes
- Achieve an appropriate balance between expenditure on implementation and evaluation (can be expensive to evaluate)
- May be better use of time and resources to carry out in-depth evaluation of key projects/activities rather than seek to evaluate them all
- Develop strong links with appropriate university departments to develop the skills for evaluation of ongoing and new work.

Model of infant feeding policy

The composition of the working party could be as follows.

- Nutrition and dietetic service (especially hospital and community pediatric dietitians working with sick children, community dietitians working with well children).
- Community and hospital pediatricians.
- Health visitors.
- Community dental health service.
- Midwives.
- Children's hospitals nurses.
- Surestart/Children's Centre representative.
- Representative of local council responsible for nursery schools.
- Community/voluntary groups, e.g. National Childbirth Trust, parent groups.

Suggested topics for a joint local community–hospital based infant feeding policy could be the following.

- Feeding policy aims.
- Applying healthy eating guidance to this age group after 2 years.
- *Breastfeeding:* diet, drug use, tips.
- *Infant formula milks:* types, preparation.
- *Other drinks:* goat, sheep, cows, soya milk, juice, bottled/tap water (see 📖 Chapter 13, 'Weaning', p. 258 for advice on suitability).
- *Weaning:* practical guidelines for first year of life.
- *Vitamin and mineral supplements:* during pregnancy, lactation, and in infancy.
- *Special dietary considerations:*
  - religious and ethnic groups;
  - vegetarians and vegans;
  - food allergy and food intolerance;
  - diarrhoea and constipation;
  - iron deficiency anaemia;
  - faltering growth.
- Faddy eaters and behavioural management.
- Food safety.
- *Action plans for implementation:*
  - training programme for key staff;
  - applying guidelines to food provided in nursery schools/units.
- *Regular monitoring and evaluation:* e.g. annual updates to infant feeding practice and 5-yearly review.
- *Appendices:* growth charts, dietary reference values, and key contacts.

See 📖 Chapter 13, 'Infants and preschool children', p. 237.

Model of a local school food policy

Suggested topics for a model school food policy incorporating a 'whole school approach' could be the following.[1]

- Food policy aims.
- *Equal opportunities:* Strive to provide equal access of opportunity for all.
- *Curriculum opportunities:*
  - leading by example and staff training;
  - visitors in the classroom;
  - resources for teaching;
  - evaluation of pupils' learning.
- *Food and drink provision throughout the school day:*
  - Breakfast;
  - food and nutrient based standards for school lunches and school food;
  - school fruit scheme (if applicable);
  - tuck-shop (if applicable);
  - vending machines (if applicable);
  - out of hours learning;
  - use of food as a reward;
  - drinking water provision.
- *Food and drink brought into school:*
  - Snacking;
  - mobile caterers serving food on school premises;
  - packed lunches brought to school by pupils.
- *Growing food:*
  - opportunity to see food growing;
  - discuss where food is produced;
  - importance of fruit and vegetables as part of a healthy diet.
- *Special diets:*
  - religious and ethnic groups;
  - vegetarians and vegans;
  - food allergy and food intolerance.
- Food safety.
- Action plans for implementation.
- *Regular monitoring and evaluation:* e.g. annual survey of the views of teachers, pupils, and parents about the school eating environment.

NB. Implementation of the government report *Turning the tables: transforming school food—recommendations for the development and implementation of revised school lunch standards* (2005) means all schools need to develop a food policy.

See 🕮 Chapter 14, 'School-aged children and adolescents', p. 279.

[1] Adapted from the School Food Action Group, Greater Nottingham Food Initiatives Group.

Model for a local obesity strategy

See Box 18.7 for a sample strategy.

Box 18.7 Sample strategy for a 'health community' for the prevention and management of obesity

An example of the composition of working party:

- Nutrition and dietetic service: community and hospital
- Consultant diabetologist and/or diabetes specialist nurse
- General practitioner
- Health visitors
- Health promotion officers specializing in health behaviour
- Public health specialist
- Practice nurse
- Consultant surgeon
- Pharmaceutical advisor
- Cardiac rehabilitation nurse
- Consultant psychiatrist
- Local authority representatives.

Suggested areas to include in the strategy

- *Background:* obesity as a health problem nationally and locally; related strategies locally; defining at risk groups locally
- *Evidence base:* models of obesity prevention and management
- National and local strategic direction
- *Aims/objectives of obesity strategy:*
  - primary health care;
  - wider community;
  - secondary care;
  - partnership approaches.
- Framework for obesity prevention and care pathway for management of overweight and obesity.
- *Action plans for implementation:*
  - training programme for key staff;
  - timescale;
  - key areas for action.
- Regular monitoring and evaluation of key aspects.

NICE guidance on obesity prevention and management is planned from 2007.

Definitions in health promotion

Nutrition intervention programmes draw on health promotion techniques.

Health promotion

Health promotion is almost always concerned with change and has been defined by the WHO as a process enabling people to exert control over and improve their health, whilst recognizing that the shaping of a healthy environment contributes to improving health status. Health is a positive concept, emphasizing social and personal resources, as well as physical capacities. Therefore, health promotion is not just the responsibility of the health sector, but goes beyond healthy behaviour to well-being.

Participation is essential to sustain health promotion action. The First International Conference on Health Promotion was held in Ottawa, Canada, in 1986, producing what is now widely known as the Ottawa Charter for Health Promotion.

The Ottawa Charter (1986)

Identified 3 basic strategies for health promotion:

- *Advocacy* for health to create the essential conditions for health.
- *Enabling* all people to achieve their full health potential.
- *Mediating* between different interests in society in pursuit of health.

These strategies are supported by 5 priority action areas as outlined in the Ottawa Charter:

- Build healthy public policy.
- Create supportive environments for health.
- Strengthen community action for health.
- Develop personal skills.
- Re-orientate health services.

The Jakarta Declaration (1997)

This was taken a step further at the 4th International Conference on Health Promotion (Jakarta, Indonesia, 1997). The Jakarta Declaration that emerged in *Leading health promotion into the 21st century* confirmed that these strategies and action areas are relevant for all countries. Comprehensive approaches to health development are the most effective. Those that use combinations of the above 5 strategies are more effective than single-track approaches. It identified 5 priorities:

- Promote social responsibility for health.
- Increase investments for health development.
- Expand partnerships for health promotion.
- Increase community capacity and empower the individual.
- Secure an infrastructure for health promotion.

Health education

Health education is not only concerned with the communication of information, but also with fostering the motivation, skills, and self-efficacy necessary to take action to improve health. Health education includes the

communication of information concerning the underlying social, economic, and environmental conditions impacting on health, as well as individual risk factors and risk behaviour, and use of the health-care system. Thus, health education may involve the communication of information, and development of skills that demonstrate the political feasibility and organizational possibilities of various forms of action to address social, economic, and environmental determinants of health.

Health protection

Is the avoidance or reduction of potential harm from exposure through organized efforts, including legislation. In recent years health protection has become a major feature of public health governance. Health protection includes food and water safety.

Qualitative and quantitative methods for evaluation in health education

Basic experimental design and particularly a randomized control design, is well established as the ideal method for evaluation. In the field, it is not always possible to meet the basic criteria for such an experimental design, and doing so can reduce programmes to unreal 'sterile' interventions that are not appropriate to real life situations.

Using multiple methods in health promotion evaluation improves the power of the evaluation and the validity of the conclusions.

A distinction is often made between qualitative and quantitative approaches to evaluation.

- *Quantitative research* examines patterns of behaviour or attitudes by assessing how certain factors influence the expression of these patterns. Particularly useful to estimate net effects of programmes, e.g. whether dietary changes can be attributed to the intervention.
- *Qualitative methods* attempt to determine the meaning and experience of the programme for those involved and to interpret the effects that *may have been observed. Particularly useful for measuring the process.*

Further information: health promotion

Bambra, C. (2009). Fear of the dark? A beginner's guide to undertaking systematic reviews of public health policy interventions. *J. Epidemiol Community Health*.

Donaldson, L.J. and Scally, G. (2009). *Donaldsons' essential public health*, 3rd edn. Radcliffe Publishing, Oxford.

Hickson, M. (2008). *Research Handbook for Health care professionals*. Blackwell Publishing, Oxford.

Naidoo, J. and Wills, J. (2009). *Foundations for health promotion (public health and health promotion)*, 3rd edn. Elsevier, London.

Pencheon, D., Guest, C., Melzer, D., *et al.* (2006). *Oxford handbook of public health practice.* Oxford University Press, Oxford.

Thorgood, M., and Coombes, Y. (2010). *Evaluating health promotion: practice and methods*, 3rd edn. Oxford University Press, Oxford.

The Institute of Health Promotion and Education. www.ihpe.org.uk/home/index.htm

The Faculty of Public Health. www.fphm.org.uk

Public Health Agency, Northern Ireland. www.publichealth.hscni.net/

The Royal Society for Public Health www.rsph.org.uk/

The UK Public Health Association. www.ukpha.org.uk/

WHO. Health Promotion. www.who.int/topics/health_promotion/en

The Chartered Institute of Environmental Health. www.cieh.org

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Chapter 19

Sustainable diets

Sustainability and nutrition

The public health nutrition field has identified a need to encompass the inter-relationship of man with his environment (The Giessen Declaration, 2005). Ecological public health nutrition places nutrition within its wider structural settings including the political, physical, socio-cultural and economic environment that influence individual behaviour and health. As a consequence, it includes the impact of what is eaten on the natural environment as well as the impact of environmental and climate change on all components of food security, i.e. on what food is available, accessible, utilizable and stable (Schmidhuber & Tubiello, 2007).[1]

The concept of incorporating sustainability into the human diet, from the perspectives of both environmental capabilities in food production terms, and the provision of nutritional guidance to inform citizens, was first addressed over 20 years ago (Gussow *et al,* 1986).[2] Despite this, it is still unclear exactly what constitutes a sustainable diet (Lang *et al,* 2009).[3]

Sustainable nutrition policy

Much progress has been made in the UK over the last few years in incorporating environmental sustainability into government nutrition policy, largely thanks to the Sustainable Development Commission's work (SDC, 2009).[4] This has included a shift towards providing sustainable hospital food (DH, 2009),[5] the introduction of the Healthier Food Mark Scheme for public sector caterers and a campaign to reduce household waste (WRAP). Sustainable nutrition policy should combine the 'three pillars' of social, economic and ecological sustainability, as set out at the 2005 World Summit.

There is currently (2011) no government advice in the UK on what a sustainable, healthy diet should be. However, the UK based NGO 'Sustain' provides guidance, which is a useful starting point to advise the general public (Box 19.1).

[1] Schmidhuber, J. & Tubiello, F.N. (2007). Global food security under climate change. *Proc. Natl Acad. Sci.* **104**, 19703–8.
[2] Gussow, J.D., and Clancy, K.L. (1986). Dietary guidelines for sustainability. *J. Nutr. Educ.* **18**(1), 1–5.
[3] Lang, T., Barling, D., and Caraher, M. (2009). *Food policy: integrating health, environment and society*. Oxford University Press, Oxford.
[4] Department of Health (2009). *Sustainable food—a guide for hospitals*. Department of Health, London.
[5] Sustainable Development Commission (2009). *Setting the Table. Advice to Government on priority elements of sustainable diets*. SDC, London.

Box 19.1 Sustainable nutrition advice for the general public

- Buy local, seasonally available ingredients as standard
- Buy food from farming systems that minimize harm to the environment, such as certified organic produce
- Reduce the amount of foods of animal origin (meat, dairy products and eggs) eaten
- Stop buying fish species identified as most 'at risk' by the Marine Conservation Society
- Choose Fair-trade-certified products for foods and drinks imported from poorer countries
- Avoid bottled water and instead drink plain or filtered tap water.
- Make sure meals are made up of generous portions of vegetables, fruit and starchy staples like whole grains, cutting down on salt, fat and oils.

Sustainable development

Defined as 'Development that meets the needs of the present without compromising the ability of future generations to meet their own needs' (World Commission on Environment and Development, 1987).

Agenda21

Agenda 21 is an action plan for sustainable development for the world in the 21st century. It was drawn up at the U.N. 'Earth Summit' in Rio in 1992, a gathering of 179 heads of state.

Agenda 21 commits governments to the following objectives:

- Meeting the basic essentials for health, i.e. safe food and water, sanitation and housing.
- Controlling communicable diseases.
- Protecting vulnerable groups, such as children.
- Reducing the health risks caused by pollution, excessive energy consumption and waste.

Local Agenda21

- Action plan for local development.
- Aims to bring together local government, business, voluntary, and community sectors to assess and meet local needs in a way that is sustainable.
- Includes transport issues, and reducing social exclusion.

Food security

The idea of food security was developed in 1974 at the World Food Summit in response to concerns about rapidly rising food prices that threatened the world food system. Since then many definitions of food security have emerged (>100 different possibilities!). Food security definitions have become broader, shifting from focusing on availability, to access; ↑ importance given to quality, not just quantity; incorporation of the notion of well-being in its broadest sense and not just related to food; changing scale from global/national to households/individuals.

Widely defined as meaning that 'people at all times should have physical and economic access to sufficient, affordable, safe and nutritious food necessary and appropriate for a healthy life, and the security of knowing that this access is sustainable in the future' (Food & Agriculture Organization (FAO)). This definition of food security incorporates 4 dimensions of whether food is:

- available (production, distribution, trade);
- accessible (affordable, quality and quantity);
- utilizable (nutritional value, social value, food safety);
- stable.

Some commentators have stated that sustainability of food production and consumption should be explicitly mentioned in the FAO's food security definition. It is suggested that there is no need to increase cultivated land, as this would damage ecosystems and biodiversity, but instead, to support 'sustainable intensification', i.e. generating greater yields using less water, fertilizer and pesticides.[1]

See 🕮 Chapter 16, 'Eating on a low income', p. 318.

[1] Nature (2010) The growing problem. Vol 466.

Climate change and obesity

Relationship of obesity and climate change

How does food consumption have an impact on climate change? Around 15% of the global population is now obese or overweight (2010) and carbon emissions have ↑ from 250 ppm (50 years ago) to 380 ppm in 2007 (Egger, 2008).[1] Some have suggested that it is no coincidence that countries with higher obesity rates tend to have higher carbon emissions, such as the US.

The cost of obesity and overweight is now starting to be felt and the cost to the UK economy alone is an estimated £10 billion annually, which is projected to ↑ 5-fold in the next 40 years due to ↑ obesity rates. Many of the costs from ↑ obesity worldwide will be carbon intensive, e.g. ↑ reliance on medical services and drugs for 'managing' obesity, as well as managing its health consequences, e.g. cardiovascular disease, type 2 diabetes and cancers.

Causes of obesity and climate change

↑ consumption of food, especially energy-dense processed foods, accompanied by ↓ physical activity contribute to both obesity and climate change. The relationship between obesity and greenhouse gas emissions is shown in Fig. 19.1 and Box 19.2.

Box 19.2 Greenhouse gases

- Methane (CH_4), carbon dioxide (CO_2), and nitrous oxide (N_2O), are the main contributors to a rise of the global temperature of 0.4°C since the 1970s.
- Twenty-two per cent of global greenhouse gases come from agriculture (McMichael *et al.*, 2007) and livestock production accounts for about 80% of this.
- Methane and N_2O are closely related to livestock production and a greater by product of this sector than CO_2. These are mainly produced by the digestive system of ruminants (enteric fermentation).
- Food production makes a significant contribution to carbon emissions, equally split between food production, distribution and retailing; energy used in buildings; transport/travel; consumption of other goods and services than food (Griffiths et al. 2008).
- Although low-income countries produce only 20% of CO_2 emissions, they produce more than half of N_2O and nearly 2/3rds of CH_4.

McMichael, A.J., Powles, J.W., Butler, C.D., and Uauy, R. (2007). Food, livestock production, energy, climate change, and health. *Lancet.* 2007, **370**(9594), 1253–63.

[1] Egger, G. (2008). Dousing our inflammatory environment(s): is personal carbon trading an option for reducing obesity – and climate change? Obesity Rev. **9**(5), 456–63.

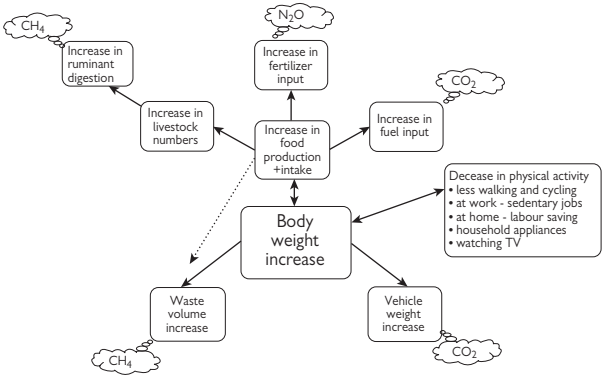

Fig. 19.1 How obesity and greenhouse gas emissions are linked.
Source: Delpeuch *et al.* (2009) *Globesity—a planet out of control*. Earthscan Books, London.

Diet and climate change

The food chain contributes ~1/5th of UK greenhouse gases emissions and is a major source of waste.

- ↑ demand for convenience food → ↑ CO_2 emissions due to production and processing, carbon intensive packaging, as many prepared foods use plastic packaging which is oil dependent (Stern, 2006).[2]
- ↑ in demand for convenience foods has contributed to a diet that is more energy-dense.
- Populations need to ↓ consumption of energy-dense foods to ↓ CO_2 emissions.
- Diets of obese people has been linked to climate change because they need to eat more calories and larger portions to meet their BMR to maintain body weight ∴ producing more organic waste, including CH_4 production when the waste decomposes!
- World meat consumption varies greatly between lower (47g/day) and higher-income countries (224 g/day) (McMichael *et al*., 2007).[3] Meat consumption is ↑, especially in countries in rapid economic and nutritional transition, e.g. S. and E. Asia. This has obvious implications for greenhouse gas emissions, as well as for obesity.
- Lowering meat consumption in high-income countries to 90 g/day (of which no more than 50 g/day should come from red meat from ruminant animals), which would allow lower income countries to converge towards this level. This would require an unprecedented shift in the eating habits for many.

[2] Stern, N. (2006). *Stern Review on the economics of climate change*. Bodley Head, Reading.
[3] McMichael, A.J., Powles, J.W., Butler, C.D., and Uauy, R. (2007). Food, livestock production, energy, climate change, and health. *Lancet* **370**(9594), 1253–63.

- The British Meat and Livestock Commission suggest that eating meat can be made more sustainable by choosing British meat, which would have less transportation carbon costs, and by changing what cows are fed to reduce methane production.

It is unclear whether eating organic meat is a less carbon intensive option.

Physical activity and climate change

The key drivers in how a lack of physical activity contributes to climate change are:

- ↑ car use, which ↓ physical activity levels, particularly that involving travel to and from work and → ↑ carbon emissions.
- At work, people are more sedentary due to ↑ in service and commercial sector sedentary jobs and ↓ in agricultural work, which could have ↓ daily energy expenditure up to 1000 kcals (Egger, 2008).[4]
- Host of labour saving household appliances, such as the washing machine, dishwasher, vacuum cleaner, that ↓ energy expenditure and ↑ carbon emissions.
- It is estimated that ~ 40% of car journeys in the UK are <2 miles, which could be walked in less than 30 min.
- Car drivers walk less than adults who don't own cars, leading to almost 1 h less walking every week.
- Heavier individuals use more fuel when using transport.
- Watching TV or computers → to CO_2 emissions and weight gain.
- Redesigning the built environment to make it easier to walk and cycle, would help ↓ obesity and climate change.

Policy options for obesity and climate change

Some of the policies mentioned in 🕮 Chapter 18, 'Nutrition intervention with populations,' 'Policy options for preventing obesity' could also reduce greenhouse gas emissions. The UK government's 'Foresight report' also focused on how a sustainable response to obesity could be delivered.

Other suggestions are:

- >50% of global population live in cities; therefore, changes in urban design are fundamental to making physical activity easier and the norm, benefiting both carbon emissions and body mass index (BMI).
- A low carbon transport system involving walking/cycling will help to ↓ obesity and national/local governments can provide safe cycle lanes, footpaths and wide public transport routes.
- Ensuring sustainable catering and food procurement policies so that local foods are sourced wherever possible, particularly for basic foods produced with minimal processing. Low energy-dense foods are more sustainable for the environment, as they are less carbon intensive.
- Supermarkets can influence greenhouse gas emissions by resolving existing tensions between how diets can be both healthy and sustainable (including sustainably sourced fish, meat and dairy). A policy that encourages supermarkets and food manufacturers to demand reformulated products, shift marketing to healthier products and introduce front of pack food

[4] Egger, G. (2008). Dousing our inflammatory environment(s): is personal carbon trading an option for reducing obesity—and climate change? *Obesity Rev.* **9**(5), 456–63.

labelling based on the traffic light system, will help ↓ obesity, and direct consumers away from more carbon intensive foods.

- Educating the public to change their attitudes to both obesity and behaving in a more sustainable manner can be part of the solution, but alone this is not enough. Changes in attitude may be more successful if carbon-intensive behaviours become taboo.

Useful websites and further reading

Bere, E., & Brug, J. (2008). Towards health-promoting and environmentally friendly regional diets- a Nordic example. *Publ Health Nutr* **12**(1), 91–6.

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Chapter 20

Global nutrition

Global nutrition problems

Problems that persist despite much effort

- *Chronic low energy intake (hunger):* >1 billion people and 1/3 children. Highest rates of hunger are in Sub-Saharan Africa, closely linked with poverty, but most of the world's undernourished are in Asia.
- *Low birth weight:* 13 million.
- *Chronic underweight in infants:* 150–200 million.
- *Micronutrient deficiency (hidden hunger):* 2 billion:
  - vitamin A deficiency—responsible for 6.5% of all deaths of <5s;
  - iron deficiency anaemia—responsible for 20% of maternal deaths.
- *Global acute malnutrition (GAM) or wasting:* 55 million cases globally of which 19 million have severe acute malnutrition (SAM).

Most cases are found in South Asia and Sub-Saharan Africa.

Problems that are increasing in lower income countries

- *Obesity* (worldwide epidemic according to WHO): 396 million adults.[1]
- *Overweight:* >1 billion; ↑ in children.
- Predictions are that overweight/obese prevalence will affect > 50% of world's population by 2050.
- Dietary-related chronic disease (cardiovascular disease (CVD), diabetes, cancers).

Age groups affected

Newborn and infants

- Low birth weight <2.5 kg (intrauterine growth retardation).
- Problems of physical and psychomotor development.

Children and adolescents

- Faltering growth.
- Overweight and obesity.

Adults

- Underweight, chronic hunger (chronic low energy intake).
- Overweight, obesity and nutrition related chronic disease.

All ages

- Micronutrient deficiencies.

[1] Kelly, T., Yang, W., Chen C. S., Reynolds, K., and He, J. (2008), Global burden of obesity in 2005 and projections to 2030. *Int. J. Obesity* **32**(9), 1431–7.

Types of childhood 'malnutrition'

- Weight-for-height < threshold = *wasting* (also known as 'acute malnutrition').
- Height-for-age < threshold= *faltering growth or stunting* when adult (also known as 'chronic malnutrition').
- Weight-for-age < threshold= *underweight* (also known as 'global acute malnutrition').

See 🕮 Chapter 25, 'Malnutrition universal screening tool', p. 504 and 'Undernutrition', p. 508 and 🕮 Chapter 13, 'Infant growth and development', p. 238 and 'Faltering growth', p. 266.

The consequences of undernutrition in low-income countries

The consequences of undernutrition are enormous.

- ↑ *Mortality rates in children:* 1/2 of deaths of under fives, i.e. 6 million each year, are linked to poor nutrition.
- Long-term illness.
- Infection.
- Deficiency disease.
- Impaired development → impact on local and national economy.
- Physical development.
- Short stature.
- Mental development.

Consequences of short stature for individuals/populations

Being small in itself is not a problem, but there is a relationship between maximal physical work capacity or the capacity to maintain physical effort and lean body mass. There is a generational effect, i.e. a stunted mother increases the risk of having a newborn with faltering growth.

Faltering growth can be a symptom of a poor diet and underlying health problems. It is indicative of an increased risk to health (morbidity, mortality, poor physical, and psychomotor development). The observation of problems of stunting (adults) or faltering growth (child) are important on a population level and suggest the need for public health interventions but also the development of programmes to fight social and economic deprivation.

Further information see ACC/SCN, UNICEF, 1989; Lancet 2008 Maternal and Child Undernutrition series.

Infant feeding

See 🕮 Chapter 13, 'Infant growth and development', p. 238; 🕮 'Breast versus bottle feeding', p. 242; 🕮 'Promoting and establishing breast-feeding', p. 246, and 🕮 'Weaning', p. 258.

Causes of global undernutrition

Undernutrition is caused by a range of factors and is rarely due to a simple lack of food. People vulnerable to malnutrition are those who depend on others, especially children and the elderly. Risk factors for hunger include:

- low food intakes;
- low levels of female schooling, education, and status;
- poor access to sanitation and clean water;
- low national expenditure on health and education;
- poor food supply;
- unequal world trade, food distribution;
- drought;
- war;
- introduction of formula feed instead of breast;
- weaning onto poor quality foods;
- large families, but population growth worldwide is slowing down;
- mothers feeding children before themselves;
- infection → ↓ appetite, ↑ diarrhoea, malabsorption, BMR → ↓ food intake and ↑ nutrient losses and requirements.

A conceptual framework of the causes of undernutrition presents different levels of causative factors. Without a clear understanding on the multiple causes of undernutrition, it is very difficult to successfully reduce the prevalence of undernutrition.

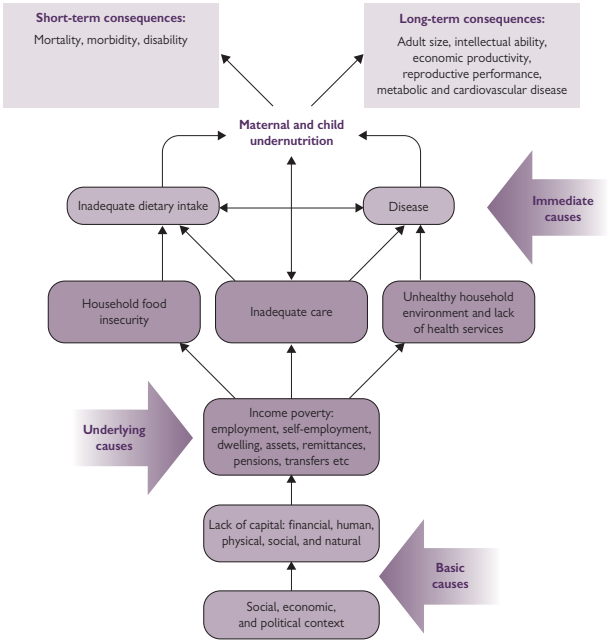

Fig. 20.1 Causal framework and consequences of maternal and child undernutrition. Reprinted from *The Lancet*, **371**, issue 9608, Black *et al*., Maternal and child undernutrition, 2008. With permission from Elsevier

Millennium development goals

The World Food Summit in 1996 set 8 millennium goals for 2015 to help reduce hunger (see Box 20.1).

Box 20.1 The millennium development goals

- **GOAL 1:** halve between 1990 and 2015, the proportion of people whose income is < $1 a day and halve between 1990 and 2015, the proportion of people who suffer from hunger.
- **GOAL 2:** ensure that by 2015, children everywhere, boys and girls alike, will be able to complete a full course of primary schooling.
- **GOAL 3:** eliminate gender disparity in primary and secondary education by 2005 and in all levels of education before 2015.
- **GOAL 4:** reduce the under-five mortality rate by two-thirds between 1990–2015.
- **GOAL 5:** reduce the maternal mortality ratio by three-quarters between 1990–2015.
- **GOAL 6:** halt and begin to reverse the spread of HIV/AIDS by 2015; halt and begin to reverse the incidence of malaria and other major diseases by 2015.
- **GOAL 7:** integrate the principles of sustainable development into country policies and programmes; halve the proportion of people without sustainable access to safe drinking water by 2015; achieve a significant improvement in the lives of at least 100 million slum dwellers by 2020.
- **GOAL 8:** develop a global partnership for development.

Further information: www.fao.org/docrep/009/a0750e/a0750e00.htm
Also www.un.org/millenniumgoals

❗ Reviews of progress made towards millennium development goals (MDGs) suggest that these will not be attained.

Iron deficiency anaemia globally

- Most common nutritional deficiency worldwide- affecting ¼ of global population, especially <5-year-olds and women.[1]
- Estimated anaemia prevalence is 47.5% of <5-year-olds; 41.8% of pregnant women and 30.2% of non-pregnant women.
- Highest prevalence in S. Asia > S.E Asia >Sub-Saharan Africa.
- Low iron intakes → ↓ physical activity → ↓ productivity.
- Affects low, middle, and high income countries, especially the poor and least educated, children, and pregnant/lactating women.

Public health consequences of iron deficiency

- *Educational deficits:* poor cognitive development, motor skills and school achievement:
  - *severe health implications:* ↑ child and maternal deaths;
  - *national productivity:* ↓ physical and mental ability ∴ ↓ productivity;
  - *poor diet:* food insecurity; reliance on plant-based staple foods;
  - *poor iron stores:* habitually poor diet and high demands;
  - *pregnancy:* blood loss after delivery and pregnancies close together.
- *Maternal deficiencies:* babies born with low reserves; breastmilk may be low in iron, if diet is low in iron.
- *Malabsorption:* intestinal infections (diarrhoea).

Preventing iron deficiency anaemia

WHO suggest that iron deficiency anaemia is best dealt with by tackling all risk factors simultaneously by:

- *Improving iron intakes:* e.g. fortification/supplementation/education.
- *Infection control,* e.g. combat hookworm and malaria.
- *General improvements* in nutrition and dietary diversity.

See 🕮 Chapter 6, 'Iron', p. 128 for further information.

[1] McLean *et al.* (2008) Worldwide prevalence of anaemia, WHO vitamin and mineral information system, 1993–2005. *Pub Health Nutr* **12**(4), 444–54.

Iodine deficiency disorders globally

WHO (2007) estimated that iodine deficiency remains a public-health problem globally, with 47 countries still affected, but the prevalence has fallen (see Box 20.2). Two billion people have insufficient iodine intake globally, with SE Asia and sub-Saharan Africa most affected, including 1/3rd of all school-aged children.

Iodine-deficiency disorders are the most important cause of preventable mental retardation worldwide, and their elimination could contribute to at least 5 of the Millennium Development Goals (see Box 20.1):

- eradicate extreme poverty and hunger;
- achieve universal primary education;
- reduce child mortality;
- improve maternal health;
- develop a worldwide partnership for development.

Iodization of salt is the most effective strategy to control iodine deficiency, which also contributes to economic and social development. Worldwide, the number of households using iodized salt has risen to more than 70%, therefore reducing iodine deficiency.

International action organized by a coalition of international organizations—including International Council for Control of Iodine Deficiency (ICCID), WHO, Micronutrient Initiative, and UNICEF, national iodine-deficiency disorders control committees, and the salt industry.

Box 20.2 Public health implications of iodine deficiency

Children

- Cretinism, goitre
- Growth impairments
- Brain growth- mental impairment.

Adults

- Goitre
- Mental impairment (apathy, loss of initiative, decision-making).

Communities

- Loss of economic development
- Lower productivity
- Social problems.

See 🕮 Chapter 6, 'Iodine', p. 140 for further information

Vitamin A deficiency globally

- Vitamin A deficiency (VAD) is the most common manifestation of poor nutrition globally besides undernutrition.
- Sub-clinical state affects 190 million children.
- Clinical deficiency affects 14 million children, but adults also at risk.
- VAD → xerophthalmia, which can → blindness: 250–500 thousand children go blind each year due to deficiency.
- VAD results in ↑ susceptibility to infection, such as diarrhoea and respiratory infections.

See 🕮 Chapter 6, 'Vitamin A', p. 94 for further information.

Risk factors for deficiency

- *Poor growth:* VAD associated with stunting.
- *Presence of other nutrient deficiency:*
  - Undernutrition;
  - zinc, selenium, iodine deficiencies.
- *Low socioeconomic status:*
  - poverty is the main risk factor for VAD;
  - clinical VAD will only be seen in the most impoverished nations.
- *Young age:*
  - occurs at all ages, but rare in children under 2;
  - 2–4-year-olds at greatest risk.
- *Gender:* 20% more boys develop VAD than girls.
- *Poor diet:*
  - poorer countries have low intakes of animal produce (meat, eggs);
  - reliance on carotenoids, but carotenoid bioavailability only 3–10% vs. 80% for retinol;
  - where rice is staple food (S and SE Asia) VAD will be more likely.

Prevention of vitamin A deficiency

Food-based approaches

- Aim to educate, provide opportunity to take more vitamin A in diet.
- Diet diversification.
- Fortification.

Supplementation

- Aim to provide high-dose of vitamin A, 2–3 times per year.
- E.g. schools to run vitamin A days, national campaigns.
- Target women at end of pregnancy—they will pass on vitamin A in breastmilk.

Immunization/supplementation

Builds occasional high dose supplement into protocols for immunization.

Vitamin A supplementation has been the cause of controversy.[1] The World Public Health Nutrition Association has questioned the motives and validity of the current practice of providing regular supplements of massive medicinal doses of vitamin A to children aged 6 months–5 years. They suggest that food-based approaches are more sustainable, favouring vitamin A rich plant oils, e.g. palm oil, promoting breastfeeding, and use of plant sources of carotenoids. Others argue that this needs to be balanced with vitamin A supplementation, which is one of the most cost-effective interventions that exists, as outlined in the *Lancet* 2008 series and the Copenhagen Consensus 2008, so arguably it is more a matter of assessing needs better.

[1] Latham, M. (2010). The great Vitamin A fiasco. *World Nutr.* **1**, 12–45. Available at: www.wphna.org.

Nutrition transition

Nutrition transition[1] is defined as a shift from a traditional diet using local foods to eating more:

- processed foods;
- food of animal origin: dairy, meat, eggs;
- food energy, especially from fat, sugar;
- 'fast foods' and soft drinks become easily available and affordable;
- diversity → more diversity and as a consequence a reduced intake of fruit and vegetables, cereals, and non-starch polysaccharides (NSP). It is accompanied by a ↓ in physical activity levels.

Nutrition transition is stimulated by a number of factors:

- ↓ in relative price of certain foods (oil/sugar);
- ↑ urbanization, that modifies lifestyle, food patterns, and energy expenditure. 45% of the population of low and middle income countries now live in urban areas;
- culture—obesity and overweight as signs of affluence;
- globalization of markets, ∴ making high energy dense processed foods more available and accessible;
- advertising and marketing, ∴ making high energy dense processed foods more desirable;
- low birth weight infants leads to programming of ill-health → ↑ likelihood of obesity/chronic disease in later life (Barker hypothesis).

See 🕮 Chapter 21, 'Foetal programming', p. 415.

Health consequences of the transition

- Large ↑ in diet-related chronic diseases (non-communicable) in adults (and now in children) → Coronary heart disease, strokes, type II diabetes, cancers, obesity.
- 80% of chronic disease deaths occur in low/middle income countries.
- Over 2/3 of diabetes cases are in the low and middle income countries.
- Over half of the new cases in the world are found in India and China.
- The age-specific burden is relatively much higher at a younger age in poorer countries.
- Increasing prevalence of overweight and obesity in low income/food insecure households—a seemingly paradoxical association. One explanation is dependency on low cost foods that are flour/tuber-based and also high in added sugar, fats/oils.
- Limited variety of food consumed relying on a few 'stomach-filling' high energy foods.
- Evidence of overweight mother/underweight or stunted child in the same household among the poor—a challenging situation for public health.

Preventing obesity

See 🕮 Chapter 18, 'Policy options for preventing obesity', p. 360.

[1] Popkin, B.M. (2001). *J Nutr* **131**, 871S–3S.

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Nutrition in emergencies

'Nutrition in Emergencies' (NIE) has progressed and expanded greatly over the past 20 years with the renewed political interest in tackling hunger and famine. Humanitarian funding demands that activities work to internationally accepted professional standards.[1] Detailed assessment methods, programme design, training manuals,[2] and monitoring and evaluation systems are all widely documented and available at no cost.

Tools exist to fully address undernutrition and the challenge is to increase the knowledge of personnel in emergency prone countries. The 'emergency' tends to relate to context, e.g. war/famine, but with a case fatality rate of 20–30%, undernutrition can be viewed as an emergency.

The undernutrition crisis

Regrettably, climate change, population growth, and rising oil prices will exacerbate the crisis. It is important within emergency contexts to protect nutritional status in all people if excess morbidity and mortality are to be avoided. South Asia is the continent with the highest numbers, and prevalence rates[3] of the acutely malnourished. Sub-Saharan Africa has frequent and high profile 'famines', though the mortality rates tend to be lower than that of South Asia. Reasons behind this include:

- even poorer status of women in South Asia;
- lack of sanitation;
- urbanization;
- higher numbers of natural disasters which can worsen an already poor situation.

Treating undernutrition in emergencies

The case fatality rate for acute malnutrition in a non-emergency setting is 20–30%. In emergencies addressed by the humanitarian community, this is typically reduced to <10%. A significant reduction in mortality can be attained by understanding the factors that lead to death; these principles are incorporated into clinical management protocols for treatment of undernutrition of host governments, UN agencies, and non-governmental agency (NGAs):

- *Hypothermia:* thermogenesis is impaired due to lack of energy. It can be addressed with kangaroo care, provision of blankets at night.
- *Hypoglycaemia:* gluconeogenesis impaired due to lack of energy intake. It can be addressed by feeding through the night.
- *Dehydration:* can be addressed by careful rehydration with appropriate rehydration solution.
- *Infections:* usually present, but undetected in the undernourished due to impaired responses. Addressed by routine administration of broad spectrum antibiotics, as per host government guidelines.

[1] Sphere (2004). *Humanitarian charter and minimum standards in disaster relief.*
[2] UN (2008). *Harmonised training package for nutrition in emergencies.* Available at: www.oneresponse.org/nutrition.
[3] UNICEF (2008) *State of the Worlds Children.*

Common types of malnutrition seen in humanitarian emergencies

The focus of NIE is on acute undernutrition in <5-year-olds and pregnant/lactating women. However, chronic undernutrition is also very likely to be prevalent in emergency contexts. The negative impacts of undernutrition are largely reversible in <2-year-olds, i.e. 9–24 months.

Global acute undernutrition (GAM) is the proxy indicator of the prevalence of undernutrition in a population.

GAM = moderate acute undernutrition (MAM) + severe acute undernutrition (SAM)

To reduce GAM, both preventative and curative methods are required. Undernutrition is also measured as low birth weight (LBW) in newborns, low mid-upper arm circumference (MUAC) in pregnant women (typically <22 cm) and specific micronutrient deficiencies.

The main nutritional problems commonly addressed in emergencies are:

- *Acute undernutrition in pregnant and lactating women.*
- *Micronutrient deficiencies*: vitamin A and zinc should be routinely addressed. Outbreaks of scurvy (vitamin C), beri-beri (thiamine) and pellagra (nicotinic acid) have been documented in the past 20 years (see 📖 Chapter 6, p. 93, for further information).
- *Acute undernutrition in young children- kwashiorkor and marasmus*:
  - *marasmus* (Fig. 20.2) is the name given to uncomplicated starvation.
  - *Kwashiorkor* is the name given when there is the presence of bilateral oedema. The causation of kwashiorkor remains unknown, but it is NOT due to protein deficiency, as was previously believed, and indeed a low protein diet is needed to treat these syndromes.

► Mild cases of marasmus and kwashiorkor are difficult to detect visually and need to be carefully assessed, by taking weight/height, MUAC, and/or checking for bilateral oedema (Box 20.3).

Box 20.3 Marasmus and kwashikor symptoms

Marasmus

- Extremely emaciated
- Fat and muscle tissue greatly reduced
- Prominence of the scapulae, spine and ribs
- Thin, flaccid skin, 'old man's' appearance
- Normal hair
- Frequent infection with minimal signs
- Electrolyte imbalance
- Alert and irritable.

Kwashiorkor

- Bilateral pitting oedema
- Higher mortality

Box 20.3 *(Contd.)*

- Reduced fat and muscle tissue which may be masked by oedema
- Skin lesions: hyper-pigmentation, skin cracked and peeling off (Fig. 20.2)
- Prone to ulceration and infection
- Pale appearance
- Hair colour becomes paler/redder, brittle, thin
- Frequent infections, e.g. URT, otitis media, URI
- Generally apathetic lethargic and miserable when left alone. Irritable when handled.

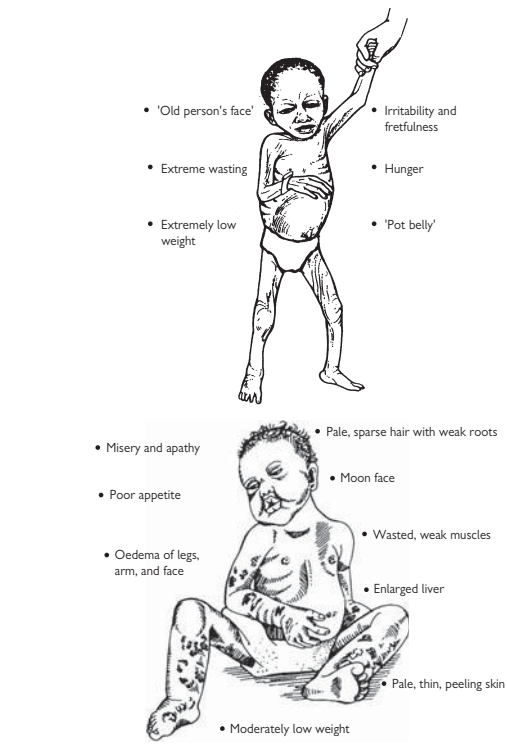

Fig. 20.2 Clinical features of (a) marasmus and (b) kwashiorkor.
Reproduced from *Oxford Handbook of Tropical Medicine*, 2nd edn, Eddison, M., Davidson, R, Wilkinson, R, Pierini, S. p. 595 (Oxford: 2005). With permission from Oxford University Press.

Anthropometric surveys to assess for acute undernutrition

The objective of an anthropometric survey is to quantify the prevalence of global acute undernutrition (GAM) within a population to determine whether an emergency response is needed. GAM is used as a proxy indicator of the health of the whole population.

Cross-sectional anthropometric surveys are implemented using either: simple random sampling, e.g. in a refugee camp situation or stratified cluster sampling, e.g. for a district level survey.

The WHO Child Growth Standards (2006) have been widely adopted and replace the NCHS growth standards (see 📖 Chapter 13, 'Infant growth and development', p. 238; see Table 20.1 for the WHO classification rates for 2003). Weight and height are collected in children aged 6–59 months; or 65–110 cm. MUAC data is collected on children 12–59 months. Presence of bilateral oedema is assessed.

Additional information can also be gathered, e.g. measles vaccination, retrospective mortality, whether the child is registered for the therapeutic or supplementary feeding programme.

Interpretation of anthropometric data

Prevalence of undernutrition must be interpreted in relation to the seasonal patterns of undernutrition.

Table 20.1 WHO classification of rates of GAM*, 2003

Severity	Prevalence of GAM
Acceptable	<5%
Poor	5–9%
Serious	10–14%
Critical	≥15%

*Global acute malnutrition.

For example, 15% wasting during the hungry season in a stable, rural population is not as alarming as a prevalence of 15% wasting post-harvest when conditions are at their best or if the mortality rate is increasing.

For mortality, the following thresholds are used:

- <5 years mortality rate of 2/10,000/day = serious situation.
- <5 years mortality rate of 4/10,000/day = an emergency out of control.

Conceptual framework for malnutrition in emergencies

Emergency contexts can affect nutritional status in many ways → to high rates of undernutrition and preventable mortality. These may be due to epidemics, poor food security or a poor public health environment. Influences such as food price fluctuations, gender equality, climate change, and population growth mean that nutritional emergencies are likely to be commonplace for some time despite the renewed international interest in tackling it. See 📖 this Chapter, 'Causes of global undernutrition', p. 392.

Food security

See 🕮 Chapter 19, ‘Food security’, p. 383.

Tackling acute undernutrition in low-income countries

In recent years there has been a focus on building the evidence base of nutrition activities. In 2008 the *Lancet* 'Maternal and Child Undernutrition series' stated that 'More than 3.5 million mothers and children <5 years die unnecessarily each year due to the underlying cause of undernutrition, and millions more are permanently disabled by the physical and mental effects of a poor dietary intake in the earliest months of life'.

Direct evidence based interventions in low-income countries

The following activities can cost-effectively address undernutrition:[1]

- Promoting breastfeeding via individual and group counselling.
- Behaviour change communication for improved complementary feeding for infants; with food provision in food insecure environments.
- Zinc supplementation.
- Zinc in the treatment of diarrhoea via health service provision.
- Vitamin A fortification or supplementation via health service provision, nutrition programming, national immunization days.
- Salt iodization.
- Hand washing/hygiene interventions.
- The treatment of severe acute malnutrition; community-based management of acute malnutrition (CMAM) see 📖 p. 409.

Of these interventions the most effective to ↓ mortality is the promotion of exclusive breastfeeding and the most cost-effective is vitamin A supplementation.[2] In addition iron, multinutrient, and calcium supplementation in pregnant women are efficacious.

Studies suggest that if these 8 activities are implemented at scale they: 'could together prevent about one-quarter of child deaths under 36 months of age and reduce the prevalence of stunting at 36 months by about one-third'. See *Lancet* 'Maternal and Child Undernutrition series, 2010 for further information.

Traditionally nutrition in emergencies has focused on the treatment of severe and moderate undernutrition. Recently the profile of infant feeding in emergencies (IFE) has been raised, e.g. in 2010 the Haiti earthquake and Pakistan floods both had strong IFE responses.

Some of the most commonplace and longstanding developmental activities lack an adequate evidence base of efficiency and efficacy. These include growth monitoring, supplementary feeding programmes to address moderate acute undernutrition and school feeding.

[1] Bhutta *et al.* (2008). What works for maternal and child undernutrition. *Lancet* 'Maternal and Child Undernutrition series'.
[2] Copenhagen Consensus 2004 and 2008.

Community based management of acute malnutrition: a curative programme

CMAM is a type of therapeutic feeding programme[1] that treats the majority of children in their own homes. This avoids risks associated with infectious diseases in crowded environments. Children with no appetite/complications must be admitted to an inpatient stabilization centre.

Advances in food packaging and food technology → the production of ready to use therapeutic food (RUTFs). RUTF provides all the required nutrition for the child in clean packaging. Children treated at home need regular and frequent follow up by trained personnel and CMAM programmes need support from local community to be effective.

Indirect nutrition interventions in low-income countries

These activities are preventative and designed to address the underlying causes of undernutrition. Without these actions recovered children will fall back into undernutrition. These include:

- Food security and agricultural activities.
- Social protection measures such as cash transfers, cash for work.
- Primary health care.
- Provision of safe drinking water.
- Girls education and support to gender equity.

Food aid

Over 60% of all humanitarian spending goes on the distribution of food aid. Food aid is a bilateral gift from one country to another and is highly vulnerable to political considerations. It is only rarely and in high profile emergencies that food aid requirements are adequately met by the international community. Food aid should be a preventative measure brought in before the onset of a nutritional emergency.

It can be very useful in the early stages of an acute emergency. However, in the longer term its use can become controversial as the provision of large quantities of free food in the marketplace can reduce prices and become a strong disincentive to local marketing and agricultural production.

An emergency food aid basket usually provides a cereal grain, pulses; vitamin A fortified vegetable oil, iodized salt. A full ration provides 2100 kcal/p/day with 10–12% of energy coming from protein.

It can be used in a number of ways[2] in emergencies:

- *General food distribution (GFD)* refers to the free distribution of a combination of food commodities to the affected population as a whole. If the population is cut off from its food supply, or suffers abnormally high rates of malnutrition, food rations should meet all nutritional needs.
- *Food for work (FFW)* activities largely involves providing food aid to unskilled labourers in exchange for work.
- *School feeding* is the provision of foods as a meal or snack at school.
- *Blanket supplementary feeding* for all under fives is used to protect food security.

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WHO (1999). *The treatment of severe malnutrition, a guide for senior health professionals in developing countries.* WHO, Geneva.

Impact assessment of large scale food security programmes (FAO)

www.fao.org/spfs/monitoring-evaluation/e-learning/en/.

Chapter 21

Obesity

Classification and prevalence

Classification

The simplest and most widely used classification for obesity in adults is based on body mass index (BMI; Table 21.1 and see Appendix 2, p. 751). For obesity in children, see 📖 Chapter 14, 'Childhood obesity and weight problems', p. 288.

Table 21.1 Classification of obesity in adults based on BMI*

	BMI (kg/m^2)
Underweight	<18.50
Normal range	18.50–24.99
Overweight/pre-obese	25–29.99
Obesity - class 1	30–34.99
- class 2	35–39.99
- class 3	≥40

*World Health Organization 1998. Obesity. *Preventing and managing the global epidemic. Report of a WHO Consultation on Obesity*. World Health Organization Geneva (revised 2010 from http://apps.who.int/bmi/index.jsp?introPage=intro_3.html).

While BMI is simple and quick to use, it has limitations because it is based simply on the ratio of weight to height and does not take account of body composition. For example, lean but well-muscled individuals may have a BMI above 25 kg/m^2 but not have an excess of body fat. However, this is an issue for a relatively small proportion of the population at the boundaries of the categories and BMI remains practical in most situations and is widely used. If clarification is required, waist circumference can be used to identify individuals at risk (Table 21.2).

BMI cut-offs for Asian populations

There has been debate whether BMI cut-off points should be lower for Asian populations because the proportion of Asian people with a high risk of type 2 diabetes and cardiovascular disease is substantial at BMIs lower than the existing WHO cut-off point for overweight. However, the cut-off point for observed and high risk varies in different Asian populations so WHO recommend using international WHO BMI cut-off points (Table 21.1).

Prevalence

UK

The prevalence of obesity is the UK has been increasing for decades with ~24% of men and 25% of women in England classified at obese (BMI >30 kg/m^2) in 2008 (The Health and Social Care Information Centre 2010)

compared to 6% and 8% respectively in 1980. ↑ risk of obesity is associated with:

- increasing age;
- lower socio-economic groups, especially women;
- ethnicity, higher in Black African, Pakistani, and White.

http://www.foresight.gov.uk/Obesity/14.pdf

Worldwide

Prevalence varies greatly from country to country from <0.1% in South Asia to >75% in urban Samoa. Globally, it is estimated that more than 1 billion adults are overweight and at least 300 million are obese. Increases in prevalence have been observed in North America, UK, Eastern Europe, the Middle East, the Pacific Islands, Australasia, and China, but some of the fastest rates of increase have been observed in urban areas of low and middle income countries where obesity and undernutrition coexist ('double burden'). This rapid increase in obesity has been attributed to the nutrition transition (see Chapter 20, 'Nutrition transition', p. 400).

Table 21.2 Classification of risk of obesity based on waist circumference*

	Waist circumference (cm)
Men	
↑ risk	94–102
Substantially ↑ risk	>102
Women	
↑ Risk	80–88
Substantially ↑ risk	>88

*World Health Organization 1998. *Obesity. Preventing and managing the global epidemic. Report of a WHO Consultation on Obesity*. World Health Organization Geneva.

Contributing causes and clinical consequences

Contributing causes

Obesity results from an excess of dietary energy intake over energy expenditure and thus both an increase in intake and a decrease in expenditure will lead to excess calories being stored as fat and, ultimately, to obesity. The UK Foresight project[1] identified that energy imbalance is determined by a complex multifaceted system of determinants, where no single influence dominates. These include:

Increased energy intake

Food has become more accessible and often cheaper in most parts of the world through improved agricultural practices, industrialization of food processing, and introduction of efficient food transport and storage. In addition, energy dense foods are more available and it has become easier and quicker to obtain, prepare, and eat palatable meals. In spite of this trend, it appears that the average total energy intake of the UK has fallen over the last 30 years, a period during which obesity rates have ↑. There is debate over whether this is due to (1) under-reporting of food intake or (2) an inadequate evaluation of food consumed outside the home. Although the concentrated calories found in a high fat, high sugar diet undoubtedly contribute to an excessive energy intake, this is not the only factor responsible for the present obesity epidemic.

Decreased energy expenditure

There are no nationally representative data for monitoring energy expenditure in the UK. However, evidence points to a rapid decline in levels of activity when evaluated by participation in manual labour, car ownership, availability of labour-saving devices, hours spent watching television, and computer use. This decline mirrors the rise in obesity and is thus considered a major contributory factor[2].

Metabolic factors

There is no evidence to support the concept that a low metabolic rate is the major cause of obesity. However, in a very small number of individuals, endocrine disorders such as Cushing's syndrome and hypothyroidism, Prader–Willi syndrome (see 📖 this Chapter, 'Conditions associated with obesity', p. 440), and congenital leptin deficiency are the cause of obesity.

Genetic factors

Obesity tends to run in families but shared environmental factors (meals and level of activity) probably contribute more to obesity than common genetic factors and the current, rapid increase in obesity prevalence cannot be explained by the gene pool changing so quickly. However, it is likely that some individuals are genetically more susceptible to the effects of an obesogenic environment. Evidence to support this came from a large

[1] Butland, B., Jebb, S., Kopelman, P., *et al.* (2007). *Foresight—tackling obesities: future choices—project report*, 2nd edn. Government Office for Science, London.
[2] Prentice, A.M. and Jebb, S.A. (1995). Obesity in Britain: gluttony or sloth? *Br .Med. J.* **311**, 437–9.

study of twins in the UK[3] that reported that BMI was largely down to genes and only 25% of variation was due to environment.

Foetal programming

The Barker hypothesis proposes that undernutrition during pregnancy may permanently damage the fetus, leading to the programming of ill health, including ↑ susceptibility to obesity in adulthood. Exposure to undernutrition or over-nutrition in early life is a risk factor for disease in adulthood. Low birth weight (<2.5 kg) or thinness at birth predict later risk of CHD and type 2 diabetes. Rapid catch up growth in infancy following fetal restriction ↑ disease risk. WHO estimate that 30 million babies are born with low-birth-weight annually worldwide, mostly in low-income countries, which contributes to their rising prevalence of obesity.

Obesogenic environment

The obesogenicity of an environment has been defined as '*the sum of influences that the surroundings, opportunities, or conditions of life have on promoting obesity in individuals or populations*'.[4]. This includes the physical, economic, political and socio-cultural environments relating to food and physical activity. Environmental influences on diet may involve access to foods from supermarkets, or takeaways and restaurants. Environmental influences on physical activity levels may involve access to cycle lanes, safe walking areas. The Foresight report[5] on tackling obesity identified how the 'obesogenic environment' exposes humans' biological vulnerability to gain weight.

Psychosocial influences

Evidence suggests that psychological factors act as a driver of obesity, including ambivalence. There is a conflict between what people want to eat (e.g. high fat/sugary tasty foods) and their desire to be slim. People who are ambivalent may not perceive obesity as an issue that directly concerns them (optimistic bias). Further information in *Foresight-Tackling Obesities: Future Choices project* report (2007).

[3] Wardle, J., Carnell, S., Haworth, C.M., *et al.* (2008) Evidence for a strong genetic influence on childhood adiposity despite the force of the obesogenic environment. *Am. J. Clin. Nutr.* **87**, 398–404.

[4] Swinburn, B., and Egger, G. (2002). Preventive strategies against weight gain and obesity. *Obes. Rev.* **3**, 289–301.

[5] Jones, A.,. Bentham, G., Foster, C., *et al.* (2007) *Foresight—tackling obesities: future choices—obesogenic environments—evidence review*. Government Office for Science, London.

Clinical consequences

Obesity is associated with a higher risk of death and morbidity (Table 21.3). The life expectancy of men and women with a BMI of >45 kg/m^2 aged 20–30 years is 13 and 8 years lower, respectively, than that of those with a BMI of 24 kg/m^2. It is estimated that approximately 34,100 deaths (~7%) in England in 2004 were attributable to obesity[1].

Metabolic Diabetes type 2 (insulin resistance), hyperlipidaemia, hypertension, stroke, gall stones, breast and colon cancer, infertility (men and women), and polycystic ovary syndrome.

Physical Osteoarthritis, chronic back pain, respiratory problems, ↓ mobility and accidents, sleep apnoea, skin problems.

Psychosocial Depression, low self-esteem, social isolation, poor employment status, impaired relationships.

Table 21.3 Relative risk of developing complications associated with obesity in adults in England[1]

	Men	Women
Diabetes type 2	5.2	12.7
Myocardial infarction	1.5	3.2
Hypertension	2.6	4.2
Stroke	1.3	1.3
Cancer of colon	3.0	2.7

[1] Health and Social Care Information Centre (2010). *Statistics on obesity, physical activity and diet: England 2010.* http://www.ic.nhs.uk/pubs/opad10.

Weight management: overview

Approach to management

Managing obesity can be very challenging but successful weight loss is accompanied by health and social benefits (see Box 21.1). A range of approaches are required to facilitate this including public health initiatives (see 📖 Chapter 18, p. 349) and those working with individuals. These are associated with varying efficacy and the optimum choices are multi-component and which people are willing to engage with and adhere to over time.

Box 21.1 Physical benefits of intentional weight loss

Losing 10 kg is associated with a reduction of:
- >20% total mortality
- 10 mmHg systolic and 20 mmHg diastolic BP
- 50% fasting glucose
- 10% total cholesterol and rise of 8% HDL cholesterol.

The first essential stage of individual management approaches is to assess:
- history of weight gain and previous attempts at reduction;
- family and medical history (is there an underlying cause, e.g. hypothyroidism, are there any co-morbidities, e.g. diabetes);
- evaluation of intake, i.e. reasons for eating, current dietary intake, meal patterns, alcohol and food preferences;
- evaluation of physical activity, i.e. current level of activity, reasons for being active or not, smoking;
- degree of motivation and whether the individual is ready to change (see 📖 Chapter 17 'Communication and counselling skills', p. 340 and Tables 17.4 and 17.5);
- preferences for managing their weight and for support;
- physical measures should include BMI and waist circumference.

Following assessment, a support package can be worked out with the patient's input. This will depend on services available locally and ideally should include behaviour change strategies to optimize eating behaviour and the quality of the person's diet and reduce energy intake and to increase people's physical activity levels or decrease inactivity. In addition, for adults pharmacotherapy and bariatric surgery may also be considered. For children see 📖 Chapter 14, p. 279.

Guidelines and care pathways

- NICE (2006) *Obesity Clinical Guideline 43* (updated January 2010). 🖰 http://www.nice.org.uk/nicemedia/live/11000/30365/30365.pdf
- NHS care pathway for adults with obesity (primary care): 🖰 http://www.dh.gov.uk/prod_consum_dh/groups/dh_digitalassets/@dh/@en/documents/digitalasset/dh_4134412.pdf
- National Obesity Forum obesity care pathway: 🖰 http://www.nationalobesityforum.org.uk/images/stories/care-pathway-toolkit/Obesity_Care_Path.pdf

Management: behaviour change strategies

Strategies to change behaviour relating to weight management include cognitive behavioural therapy and a range of other psychological approaches including cognitive therapy, psychotherapy, relaxation therapy and hypnotherapy. Depending on the type of strategy, these may either be delivered by specifically trained psychologists or adapted to simpler models which can be adopted by other healthcare professionals.

Systematic review[1] indicates that behaviour change strategies (BCS), and specifically cognitive behavioural therapy, are most effective when combined with physical activity and dietary approaches (weighted mean difference –4.9 kg: 95% CI –7.3 to –2.4, compared with exercise/diet alone).

❗ Healthcare professionals who are working with overweight and obese people, but have not had the opportunity for specific training in BCS are advised to learn about the approach which is essentially patient-centred and focused on empowering rather than didactically teaching. See 🕮 Chapter 17, 'Communication and counselling skills', p. 340 and Boxes 21.2 and 21.3.

Box 21.2 Approaches to include when discussing weight management

Healthcare professional style

- Supportive
- Patient-centred
- Respectful of patient-autonomy
- Goal focused
- Facilitative
- Non-judgmental
- Non-persuasive
- Non-confrontational

Examples of issues to explore

- Times of the day when controlling food intake is particularly difficult/less of a problem
- Eating more/being less active when feeling sad/bored/angry, etc.
- Feelings when eaten more/been less active than originally planned
- Feeling of eating/lack of activity being 'out of control'.

[1] Shaw, K.A., O'Rourke, P., Del Mar, C., *et al.* (2005). Psychological interventions for overweight or obesity. *Cochrane Database System. Rev.* **2**, Art. No. CD003818.

Box 21.3 Examples of behaviour strategies for weight reduction

- Shop for food only after eating and buy items from a list
- Eat all food whilst sitting down in one place and at planned times
- Serve food on a smaller plate; if possible, ask another person to serve
- Put down fork between each mouthful
- Chew each mouthful thoroughly
- Concentrate on eating and enjoying food
- Do nothing else while eating (e.g. watching TV)
- Leave table as soon as meal is completed
- Differentiate between hunger and the urge to eat
- Identify situations that might lead to a lapse; plan how to cope with these and consider writing them down
- Self-monitor progress, e.g. by keeping a food and activity diary
- Set realistic goals for improved eating and weight loss
- Plan rewards for goals achieved.

Management: physical activity

Increasing energy expenditure is an effective way of improving health, reducing weight and preventing weight gain (see Box 21.4). Additional benefits associated with regular exercise include ↑ sense of well-being and reduced risk of ill health including diabetes, cardiovascular disease, and some cancers. Even if no weight is lost, exercise is associated with ↓ risk of diabetes and cardiovascular disease.

Systematic review[1] indicates that physical activity combined with dietary approaches is more effective than either intervention alone (weighted mean difference –1.0 kg: 95% CI –1.3 to –0.7, compared with diet alone).

Box 21.4 Recommended physical activity for adults

- Take more exercise even if this doesn't lead to weight loss because it has other benefits such as ↓ risk of type 2 diabetes and cardiovascular disease
- Do at least 30 min of at least moderate-intensity physical activity on 5 or more days per week, in one session or several shorter ones lasting 10 min of more; 45–60 min may be needed to prevent obesity; people who have lost weight may need to do 60–90 min to avoid regaining weight
- Build up to the recommended levels using a managed approach with agreed goals
- Reduce the time spent inactive such as watching TV or using a computer
- Types of activity can include:
  - Activities incorporated into everyday life such as brisk walking, gardening or cycling
  - Supervised exercise programmes
  - Walking a certain number of steps each day, stair climbing or swimming
- Consider each person's current physical fitness and ability.

℞ NICE (2006). *Obesity Clinical Guideline 43* (updated January 2010): http://www.nice.org.uk/nicemedia/live/11000/30365/30365.pdf.

Adverse events

Relatively few side-effects have been reported but range from mild (e.g. minor trauma) to severe (e.g. myocardial infarction) predominantly in individuals with morbid obesity. As the latter have most to gain from losing weight, they require appropriate advice about increasing everyday physical activities, like walking, rather than embarking on an over-taxing exercise programme.

[1] Shaw, K.A., Gennat, H.C., O'Rourke, P., *et al.* (2006). Exercise for overweight or obesity. *Cochrane Database of System. Rev* **4**, Art. No. CD003817.

Make it fun and sociable!

The England-based Walking for Health Initiative promotes activity through a network of organized walks and encourages individuals to take a minimum of 10,000 steps per day monitored using a pedometer.

http://www.whi.org.uk

Management: dietary aspects

A plethora of weight-reducing diets is available to the general public ranging from healthy diets with a modest reduction in energy to questionable, complex or specific food regimes. The goal of all dietary management in obesity should be to help the individual to reduce their energy intake to an acceptable level while consuming a diet that is adequate in all other nutrients, compatible with long-term good health, practical to follow and can be reconciled with their life style (Box 21.5). The approaches described in this section are an outline from which different elements can be combined.

Box 21.5 Recommended dietary advice for adults

- Encourage people to improve their diet even if they do not lose weight, because there can be other health benefits
- Dietary advice should be individualized, tailored to food preferences and allow for flexible approaches to reducing calorie intake
- Do not use unduly restrictive and nutritionally unbalanced diets because they are ineffective in the long term and can be harmful
- In the longer term, people should move towards eating a balanced diet, consistent with other healthy eating advice
- Total energy intake should be less than energy expenditure.

℔ NICE (2006) Obesity Clinical Guideline 43 (updated January 2010): http://www.nice.org.uk/nicemedia/live/11000/30365/30365.pdf

Modified healthy eating or low fat diets

Based on the 'Eatwell Plate' (see 📖 Chapter 2, p. 27), a modest energy reduction of approximately 600 kilocalories per day[1] can be achieved by reducing or eliminating the intake of concentrated calories particularly from fat (see Table 21.4), increasing fruit and vegetable intake to a minimum of five portions per day and maintaining intake of whole grain cereals, lean meat or fish, and low fat dairy products. Reducing energy intake to 1000–1600 kcal (i.e. >600 kcal deficit) may be considered, but this is less likely to be nutritionally complete and ∴ should not be advised for long periods.

The 600 kcal deficit through modified healthy eating is ideal for encouraging gentle weight loss of 0.5–1.0 kg/week accompanied by a long-term change in eating habits and is suitable for well-motivated individuals. Although individuals who are looking for a rapid response may find the rate of weight loss and long-term commitment unacceptable, systematic review[2] has shown that low fat diets produce a significant weight loss up to 36 months (–3.55 kg, 95% CI, –4.54 to –2.55 kg).

[1] NICE (2006). *Obesity Clinical Guideline 43* (updated January 2010): ℔ http://www.nice.org.uk/nicemedia/live/11000/30365/30365.pdf.

[2] Avenell, A., Brown, T.J., McGee, M.A., *et al.* (2004). What are the long-term benefits of weight reducing diets in adults? A systematic review of randomized controlled trials. *J. Hum. Nutr. Dietet.* **17,** 317–35.

Low carbohydrate diets

Low carbohydrate diets, also known as protein-sparing modified fasts, are defined as providing ≤40 g/day carbohydrate. Interest in these regimes has focused on the widespread popularity and apparent success of the Atkins® diet. This advises an initial 2-week reduction in carbohydrate intake to <20 g per day while eating unrestricted amounts of protein, including poultry, fish, eggs, and red meat, and fats such as butter and olive oil. The ongoing weight loss and then maintenance programme advise continued but less stringent carbohydrate restriction.

Meta-analysis[3] of five studies showed that low carbohydrate diets are associated with a similar weight loss at 12 months to low fat, energy restrictive diets. They are also associated with favourable ↓ in triglycerides and ↑ in HDL cholesterol but a less favourable change in total and LDL cholesterol compared with those on a low fat diet. Other studies, but not all, report similar findings and there is a need for further research into the health effects beyond 12 months.

Table 21.4 Concentrated calories—and some alternatives

Fried foods	Grill, roast, or bake without fat or oil
Butter/margarine	Soften and spread thinly or use low fat spreads
Oil/cooking fat	Use non-stick pans and 'dry fry'
Salad dressings	Choose low fat options or use vinegar/mustard
Pastry	Mashed potato or bread crumbs on top of dishes
Full fat milk	Semi-skimmed or skimmed
Double cream	Diet yogurt or fromage frais or custard made from skimmed milk
Sugar/honey	Cultivate a preference for less sweet taste or use artificial sweeteners
Jam/marmalade	Sugar-free fruit spreads or savoury spreads
Confectionery	Fresh fruit or limited amounts of dried fruit
Sugary soft drinks	Water, low calorie alternatives, tea
Alcohol	Limit intake and use low-calorie mixers

[3] Nordmann, A.J., Nordmann, A., Briel, M., *et al.* (2006). Effects of low-carbohydrate vs low-fat diets on weight loss and cardio-vascular risk factors. *Arch. Intern. Med.* **166**, 285–93.

Structured weight loss plans

These regimes are designed to be followed for a limited period of time during which a detailed, prescribed menu is followed. Ideally, this should provide ~600 kcal per day less than the pre-dieting intake and include adequate nutrients. The advantage for some participants is the comfort of having a structured eating pattern especially if this is accompanied by regular weight loss. Some programmes are associated with regular support meetings where participants are weighed and motivated by their leader and fellow weight reducers. Healthcare professionals should include commercial, self-help and community programmes in the options offered to patients but, if chosen, continue to monitor their progress and provide support.

The disadvantages of structured weight loss plans can include:

- additional expense;
- rigidity of some regimes;
- hard to follow, particularly in the context of a busy family or work life;
- designed to be followed for a limited period of time;
- limited contribution to long-term healthier eating practices (although some have associated weight-maintenance programmes).

Examples include Weight Watchers® (℞ www.weightwatchers.co.uk) and Slimming World® (℞ www.slimming-world.co.uk).

Calorie counting

This is based on the simple concept of reducing energy intake to a level below total energy expenditure, i.e. negative energy balance, which is fundamental to achieving weight loss. However, focusing solely on energy content rather than more broadly on food, it is not necessarily compatible with optimum nutritional intake or promoting long-term health. Even so, some people appreciate the flexibility of being able to plan their own intake and vary this from day to day, including 'naughty' items that conventionally are not encouraged on a weight reducing diet: it is possible but not desirable to construct a low calorie diet based on chocolate, crisps, and beer! Lists of the calorie contents of a wide range of foods are available from most bookshops.

Very low calorie diets

Very low calorie diets (VLCDs) are defined as diets providing <1000 kcal/day although some may provide as little as 400 kcal/day. NICE guidance[4] advices that a diet of <1000 kcal/day should be followed for <12 weeks or intermittently, i.e. 2–4 times per week, interspaced with a diet providing 1000–1600 kcal/day if the person is obese and has reached a plateau in their weight loss.

Commercial VLCDs are usually liquid, milk-based drinks or soups, which aim to provide the consumer's total nutrient requirements within a day's limited energy format (Table 21.5). Comparable diets can be made at home from milk but require adequate micronutrient supplementation. A review of studies has shown that VLCDs are effective in bringing about short-term (4–8 weeks) weight loss and, in some but not all patients, this

[4] NICE (2006). *Obesity Clinical Guideline 43* (updated January 2010): ℞ http://www.nice.org.uk/nicemedia/live/11000/30365/30365.pdf.

is maintained over a longer period of time.[5] Concerns have been raised about a reduction in lean body mass associated with rapid weight loss and the absence of re-education about long-term eating habits. VLCDs are contraindicated in individuals who have a history of, or are at risk from cardiac disease, cerebrovascular accident, renal or liver disease, hyperuricaemia, porphyria, or psychiatric disturbances and have been associated with severe adverse effects including death. Diets providing <600 kcal per day should only be undertaken under medical supervision.

Table 21.5 Composition of liquid VLCDs

	Cambridge Weight Plan®[a] (3 servings)	Optifast®[b] (4 servings)	Skimmed milk[c] (3 pints)
Energy (kcal)	415	832	594
Protein (g)	43	70	59
Carbohydrate (g)	42	167	90
Fat (g)	8	33	1.8
Micronutrients	Supplemented	Supplemented	Supplementation required

[a] http://www.cambridgeweightplan.com/

[b] http://www.nestlenutrition.co.uk/healthcare

[c] Calculated from data obtained from Food Standards Agency (2002) *McCance & Widdowson's Composition of Foods*, 6th summary edn. Cambridge: Royal Society of Chemistry, Cambridge.

Meal replacement/meal provision

Similar to the 'complete nutrition' concept of very low calorie diets, meal replacement programmes provide the weight reducer with a range of food items or meals that contribute an intake limited in energy but supplemented with micronutrients. The food items include milk shakes, soups, cereal-type snack bars and pre-prepared meals and some are delivered to the door. Most programmes give the consumer the flexibility to select from interchangeable formats within a total calorie intake and may include one 'normal' but calorie-counted meal. Some also provide lifestyle guidance on increasing physical activity and changing behaviour. However, as individuals are able to purchase these from supermarkets, chemists and via the internet, they frequently do not have input from health-care professionals. The disadvantages of such products include the expense, limited flexibility and limited availability of long-term education to support permanent changes in lifestyle.

Examples include Diet Chef® (http://www.dietchef.co.uk/), Jenny Craig Programme® (http://www.jennycraig.co.uk/), Rosemary Conley Solo Slim® (http://www.rosemaryconley.com/) and Slimfast® (http://www.slimfast.co.uk).

[5] Jebb, S.A., and Goldberg, G.R. (1998). Efficacy of very low-energy diets and meal replacements in the treatment of obesity. *J. Hum. Nutr. Dietet.* **11**, 219–25.

Box 21.6 So, which diet is the most effective?

An RCT[1] and follow up[2] of four commercial weight-loss diets[3] available in the UK showed all resulted in significant ↓ body fat and weight compared to controls after 6 months with no significant difference in loss between the diets. All diets were also associated with favourable changes to cardiovascular risk factors.

This suggests that if a person is able to follow advice to modify their intake over a period of time, they will lose weight. Therefore, it is the adherence to the regime, rather than regime *per se* which is important. A behavioural approach can optimize adherence by facilitating overweight and obese people to identity the diet that will be most effective from them.

[1] Truby, H., Baic, S., deLooy, A.,*et al* (2006). Randomised controlled trial of four commercial weight loss programmes in the UK: initial findings from the BBC "diet trials". *Br. Med. J.* **332**, 1309–14.
[2] Morgan, L.M., Griffin, B.A., Millward, D.J.,*et al.* (2009). Comparison of the effects of four commercially available weight-loss programmes on lipid-based cardiovascular risk factors. *Publ. Hlth Nutr.* **12** 799–807.
[3] Diets included 'Dr Atkins' new diet revolution', 'Slim-Fast plan', 'Weight Watchers pure points programme' and 'Rosemary Conley's eat yourself slim diet and fitness plan'.

Group or individual approaches

Meta-analysis[6] of five studies comparing weight loss following group or individual-based treatments found a significantly greater weight change at 12 months among the group participants. This finding shows the value of group-based approaches, particularly for women participants, but does not necessarily mean that groups are the best weight management strategy for all. Further investigation is needed into how best to support men.

Overall message: keep management patient-centred.

Financial incentives

Losing excess weight is very difficult for most obese people and the health benefits associated are often not immediately apparent. Can other incentives help? Meta-analysis[7] of 11 studies has shown little effect of financial incentives although they may work better if the amount is >1.2% of disposable income, related to changed behaviour rather than weight loss and delivered by a non-psychologist.

Overall message: no convincing evidence to support this approach.

[6] Paul-Ebhohimhen, V. and Avenell A.(2009). A systematic review of the effectiveness of group versus individual treatments for obesity. *Obesity Facts* **2,** 17–24.
[7] Paul-Ebhohimhen, V. and Avenell, A. (2008). Systematic review of the use of financial incentives in treatments for obesity and overweight. *Obesity Rev.* **9,** 355–67.

Pharmacotherapy for obesity[1]

When to prescribe

Drug treatment in obesity is not a 'first line treatment', but can be considered in adults if:

- Part of an overall plan for managing obesity.
- Patient's BMI is ≥30 kg/m^2 or ≥28 kg/m^2 with co-morbidities (e.g. diabetes type 2, hypertension).
- Dietary, exercise and behavioural approaches have started and been evaluated.
- Patients have not met their target weight loss or have reached a plateau after losing weight on dietary, exercise and behavioural approaches.

Before prescribing

The following should be discussed with patients:

- The prescribed drug's potential benefits and limitations.
- How the drug works and possible adverse effects.
- How they will be monitored while taking the drug.
- Agreed goal for losing weight.
- Weight loss that is required to support continued prescription.
- Offer support and counselling on dietary, exercise, and behavioural strategies.

Care required whilst prescribing

- Review weight loss.
- Patients should be monitored to evaluate the effect of drug treatment and to reinforce lifestyle advice and need for adherence.
- Consider micronutrient supplementation (i.e. to provide reference nutrient intake of all vitamins, minerals and trace elements) if concern about adequacy of dietary intake especially in vulnerable groups, e.g. elderly.

Stopping prescription

- Consider withdrawing drug treatment after 3 months if the person does not lose at least 5% of their body weight.
- Less strict goals should be used in diabetes type 2.
- When stopping, offer support to help maintain weight loss because patients' self-confidence and belief in their ability to lose weight may be low.

What to prescribe

One drug, Orlistat (see Box 21.7), is currently authorized in the UK for the treatment of obesity. RCTs show that it is an effective adjunct to dietary treatment and associated with a weight loss of 2–5 kg greater than that of control groups at 2 years. Sibutramine has now been suspended due to concerns over its use (Box 21.8).

[1] NICE (2006). *Obesity Clinical Guideline 43* (updated January 2010): http://www.nice.org.uk/nicemedia/live/11000/30365/30365.pdf.

Box 21.7 Orlistat

Orlistat (Xenical® Roche, Welwyn Garden City) reduces calorie intake by blocking digestive lipases reducing GI absorption of fatty acids, cholesterol, and fat-soluble vitamins and increasing faecal fat excretion. Side-effects include oily rectal discharge (27%) and faecal incontinence (8%). ↓ Blood fat-soluble vitamins has been observed but no clinical deficiencies have been reported.

Box 21.8 Sibutramine

Sibutramine (Reductil® Abbott, Maidenhead) was authorized until 2010 but this is currently suspended in response to evidence that cardiovascular risks associated with the drug outweighed the benefits. Its mode of action is mediated via ↓ energy intake by inducing early satiety through blocking re-uptake of serotonin and noradrenaline in the brain.

Bariatric surgery[1]

When to refer for bariatric surgery

Bariatric surgery is not generally recommended for children or young people. It is recommended as a treatment option for obese adults if:

- patient's BMI is ≥40 kg/m^2 or 35–40 kg/m^2 with co-morbidities (e.g. diabetes type 2, hypertension) ***and;***
- all appropriate non-surgical measures have been tried but have failed to achieve or maintain adequate, clinically beneficial weight loss for >6 months ***and;***
- patient has been receiving or will receive intensive management in a specialist obesity service, is fit for anaesthesia and surgery and commits to long-term follow-up.

Bariatric surgery is also recommended as a first-line option for adults (instead of lifestyle interventions or drug treatment) if patient's BMI is ≥50 kg/m^2 and surgical intervention is considered appropriate.

Before surgery

- A hospital specialist/surgeon should discuss in detail with patient ± family the potential benefits, long-term implications and risks, including complications and peri-operative mortality (see Box 21.9).
- Psychological and clinical factors that could affect adherence to post-operative care, e.g. dietary change, should be fully assessed.

Surgical intervention

- Choice of type of surgery should be made jointly with patient considering:
  - degree of obesity and co-morbidities;
  - evidence on effectiveness and long-term effects;
  - facilities and equipment available;
  - experience of the surgeon who will operate.
- The patient should be managed by a multidisciplinary team (MDT) including a surgeon who:
  - has undertaken a supervised training programme;
  - has specialist experience in bariatric surgery;
  - is willing to submit data for a national clinical audit scheme.
- The MDT should have expertise in:
  - pre- and post-operative assessment, including specialist assessment for eating disorders, dietetic and surgical follow up;
  - providing information on the procedures, including potential weight loss and risks, plastic surgery;
  - management of co-morbidities;
  - psychological support pre- and post-surgery;
  - access to suitable equipment, e.g. scales, theatre tables, and staff trained to use them.

[1] NICE (2006). *Obesity Clinical Guideline 43* (updated January 2010): ℒ http://www.nice.org.uk/nicemedia/live/11000/30365/30365.pdf.

Box 21.9 Contraindications to bariatric surgery

Include myocardial infarction, significant chronic obstructive airways disease or respiratory dysfunction, non-compliance with medical treatment and significant psychological disorders.

After the operation

- Provide regular specialist dietetic input including:
  - information on dietary intake appropriate for the procedure;
  - monitoring of micronutrient status;
  - information on patient support groups;
  - individualized nutritional supplementation, support and guidance for long-term weight loss and weight maintenance.
- Arrange for short- and long-term monitoring of outcomes, complications, impact on quality of life, nutritional status and co-morbidities.
- Surgical revision should only be undertaken in specialist centres with extensive experience because of ↑ complication rate and ↑ mortality.

Types of bariatric surgery[2]

All bariatric surgical procedures have to ↓ patient's GI capacity for ingesting ± absorbing dietary, thus ↓ available energy and promoting negative energy balance and weight loss. There is no evidence[3] at present that one type is superior to another in terms of weight loss or complications and choice depends on the individual patient's needs and surgical experience.

Gastric banding (laparoscopic)

Formation of stomach pouch using a tight band applied via keyhole surgery. Less invasive than gastroplasty or bypass and can be reversed. Band can be adjusted to change size of pouch using fluid injected via subcutaneous port. Associated with weight loss (mean 37 kg in 58% of patients at 5 years). Fewer short-term complications associated with procedure but long term, 35% of bands required removal due to band slippage, pouch dilatation and erosion.

Gastric bypass (Roux-en-Y)

Small section of upper stomach is formed into a pouch and anastomosed on to proximal jejunum, which limits food intake and bypasses most of stomach and duodenum resulting in some malabsorption. Associated with significant weight loss (50–75% of pre-surgery weight lost after 5 years). Complications include anastomotic leak, stricture formation, incisional hernia and dumping syndrome.

[2] Jaunoo, S.S., Southall, P.J. (2010). Bariatric surgery. *Int. J. Surg.* **8,** 86–9.
[3] Colquitt, J.L., Picot, J., Loveman, E. *et al.* (2009). Surgery for obesity. *Cochrane Database of System. Rev.* 2006, **2**, Art. No. CD003641.

Sleeve gastrectomy

Two-stage procedure, firstly dividing stomach vertically to reduce size to 25% whilst retaining intact pylorus. After 6–12 months and following gastric expansion and ↓ weight loss, a gastric bypass or bilio-pancreatic diversion with duodenal switch (BPD-DS) is undertaken. The advantage of the sleeve procedure is that it is safer to undertake in high risk patients, retains the pyloric function and can progress onto the second stage if needed. The advantage of the BPD-DS is that a degree of malabsorption results but biliary and pancreatic secretions still access GI tract. The BPD-DS is reversible, but sleeve is not.

Vertical banded gastroplasty

Stomach is surgically stapled to create ~30-ml pouch that restricts intake of food at any one time. Now rarely undertaken due to high failure rate.

Other

Jejuno-ileal bypass, bilio-pancreatic diversion, and jaw wiring are older procedures that are now rarely undertaken.

Post-surgical dietary management[4]

Optimum intake will depend on the type of surgical procedure undertaken, i.e. capacity-limiting ± malabsorptive. The goal is to support weight loss while achieving a nutritionally adequate intake and minimal food-related symptoms. Individual advice is needed and should be provided by a dietitian with relevant experience who is working within an MDT framework.

- *Immediately post-operatively:* starting usually with sips of clear fluids the morning after the procedure and progressing on to small (15–30 g portions), frequent intake of semisolid foods gradually increasing on to a nutrient-dense diet including puréed meat, fish, yogurt, mashed potato, fruit juice.
- *Short to medium term:* continue on small, nutrient-dense meals with increasing variety determined by trial and error, avoiding concentrated sources of energy or large quantities that may lead to gastrointestinal symptoms. If pouch formed, stop eating when pouch full and drink separately from eating allowing at least 1 h between. Daily multivitamin supplementation is recommended (adequacy of B vitamins, especially B_{12} is a concern). Nausea and vomiting are common—advise reduction of volume of food consumed and chew thoroughly. Dumping syndrome occurs in ~70% of patients following gastric bypass (see 📖 Chapter 26, ‘Gastrectomy and stomach surgery’, p. 574).
- *Long term:* nutritional complications include anaemia (multiple deficiencies including protein, Fe, Cu, Se, folate and vitamin B_{12}), osteoporosis (protein, vitamin D), neurological symptoms (B vitamins especially thiamine) and cardiovascular disease (folate deficiency leading to hyperhomocysteinaemia). Life-long nutritional and metabolic surveillance is required.

[4] Ziegler, O., Sirveaux, M.A., Brunaud, L., and Southall, P.J., Picot, J., Loveman, E., *et al.* (2009). Medical follow up after bariatric surgery: nutritional and drug issues. *Diabetes Metab.* **35,** 544–57.

Alternative treatment for obesity

As conventional treatment of obesity is often unsuccessful in the long term, there is considerable interest in novel and alternative approaches to weight reduction. These include acupuncture, herbs, aromatherapy, and hypnosis. A systematic review[1] of 517 published studies drew no conclusions on the effects of alternative treatments for obesity.

[1] Östman, J., Britton, M., Jonsson, E., *et al.* (ed.) (2004). *Treating and preventing obesity: an evidence-based review*. Wiley-VCH, Weinheim.

Obesity prevention and weight management during life stages

Obesity prevention

See 🕮 Chapter 18, 'Policy options for preventing obesity', p. 360.

Pregnancy

See 🕮 Chapter 12, 'Healthy weight for conception' p. 220, and 'Maternal weight gain', p. 228.

Children and young people

See 🕮 Chapter 14, 'Childhood obesity and weight problems', p. 288.

Role of healthcare professionals in weight management

Weight management, both prevention and treatment, requires a multidisciplinary approach with the individual patient or groups of patients at the center of the partnership. There are a number of health professionals who should be involved at different times (see Box 21.10):

Box 21.10 Health professionals involved in weight management

- Counsellors
- Dentists
- Dietitians
- Exercise professionals
- General practitioners
- Health visitors
- Hospital consultants
- Mental health professionals
- Nurses
- Pharmacists
- Physiotherapists
- Psychologists
- School nurses
- Surgeons.

Registered dietitians are well-placed to play a role in weight management programmes having been trained in nutritional science and its application and communication through changing behaviour. Their contribution may include the following.

- One-to-one sessions with individuals, evaluating their energy intake and expenditure and readiness to embark on necessary lifestyle changes. Patients can expect assistance in identifying practical lifestyle advice (dietary, activity, and motivational) that is tailored to their needs with the dietitian acting as a facilitator or enabler on how to make the necessary behaviour changes.
- Running group programme covering topics such as those in Box 21.11. Sessions can be run for general or specific groups (e.g. men only, over-60s, etc.) and may be open to self- or GP-referred patients.
- Educating other health professionals by running training days for GPs, practice nurses, etc. This might include information about prevalence and effects of obesity, when to refer, weight loss services available locally (NHS, voluntary, and commercial), morbid obesity.
- Obesity prevention through health promotion and nutrition policy (see 📖 Chapter 18, 'Local food and nutrition policy', p. 364).

Box 21.11 Topics for group weight loss programmes

- Do you want to lose weight — are you ready to change?
- Realistic goals
- What is a healthy diet?
- Overcoming temptation
- Stepping up activity
- Supermarket tour—shopping tips to save calories
- Cutting calories in cooking
- Eating out—how to choose sensibly and still enjoy your meal
- Keeping going when things get difficult
- Looking to the future and weight maintenance.

Conditions associated with obesity

In addition to the clinical consequences associated with obesity (Table 21.3), there are a number of conditions that are closely related:

Polycystic ovary syndrome

Approximately 4–10% of women of child-bearing age have polycystic ovary syndrome (PCOS), which is associated with raised androgen levels and an irregular menstrual cycle ± ovarian cysts. Common nutritional features include abdominal weight gain and the characteristics associated with metabolic syndrome (insulin resistance leading to type 2 diabetes, hyperlipidaemia, and hypertension). Reduced fertility and abnormal menstrual cycles are associated with obesity and hyperinsulinaemia and there is ↑ long-term risk of heart disease.

Management[1]

Weight loss in obese women is associated with the resumption of ovulation and ↑ fertility; relatively small losses of ~ 6 kg have been shown to be beneficial. Modification of dietary intake and physical activity is thus considered first-line treatment. In lean women, addressing insulin resistance through modifying dietary intake with ↓ glycaemic index, ↓ glycaemic load and ↓ saturated fat is recommended. Intervention studies and long-term follow up are required to confirm this.

Prader–Willi syndrome

Prader–Willi syndrome (PWS) is a complex, genetic disorder associated with excessive appetite, low muscle tone, emotional instability, immature physical development, and learning disabilities. Although infants with PWS may have feeding difficulties leading to growth faltering, children aged over 1 year often gain weight very rapidly due to hyperphagia. An estimated 3000 individuals in the UK have PWS.

Management[2]

A paediatric dietitian should give individual dietary advice and, ideally, the whole family should be involved. The goal of dietary management is to achieve a nutritionally balanced intake which optimizes height growth with an appropriate energy intake to treat or prevent obesity. PWS-specific growth standards are available. Compliance is often challenging due to an insatiable appetite and sometimes locking fridges and food stores may be required. A number of ethical issues surround the need to protect individuals with PWS as they are incapable of acting independently in relation to food (see 🕮 Chapter 32, 'Mental Capacity Act 2005', p. 687). Bariatric surgery is not recommended for PWS patients due to difficulty in post-operative compliance. More information is available from the Prader–Willi Syndrome Association UK, http://pwsa.co.uk/.

[1] Jeanes, Y.M., Barr, S., Smith, K., *et al.* (2009). Dietary management of women with polycystic ovary syndrome in the United Kingdom: the role of dietitians. *J. Hum. Nutr. Dietet.* **22,** 551–8.
[2] Butler, M.G. (2006). Management of obesity in Prader-Willi syndrome. *Nat Clin Pract Endocrinol Metab* **2,** 592–3.

Chapter 22

Diabetes

Classification and prevalence

Classification

Diabetes is a metabolic disorder characterized by chronic hyperglycaemia with disturbances of carbohydrate, fat, and protein metabolism resulting from defects in insulin secretion, insulin action, or both. See Table 22.1.

- Type 1 diabetes can occur at any age, but usually develops in children or adults aged <40 years (previously referred to as insulin dependent diabetes, IDDM). This occurs as a result of lack of insulin production by the pancreatic β cells. It requires treatment with insulin and dietary management.
- Type 2 diabetes is usually diagnosed in older adults, but is increasingly seen in younger adults and some children (previously referred to as non-insulin dependent diabetes, NIDDM). It is associated with a lack of insulin function as a result of insulin resistance (Box 22.1) with or without insufficient production and is strongly associated with overweight and obesity. Dietary management is required, with or without oral hypoglycaemic agents or insulin.
- Gestational diabetes is hyperglycaemia diagnosed during pregnancy that had not been previously diagnosed. Advice about diet, exercise and body weight is required, and some patients may also need insulin.

Box 22.1 Insulin resistance

There is considerable variation in the cellular response to insulin by different individuals. A lower than normal response is described as insulin resistance. This includes reduced glucose uptake by the skeletal muscles and/or liver, reduced lipolysis in adipose tissue, and altered amino acid metabolism either alone or in combination. As a consequence, blood glucose levels remain high and lead to further stimulation of the pancreas to release more insulin. Insulin resistance increases with overweight and obesity.

Prevalence

Globally, it is estimated that ~170 million individuals had diabetes in 2000 and that this will rise to ~366 million people by 2030. Countries with the highest number of people with diabetes include India, China, USA, Indonesia and Japan.[1] In the UK, ~2.5 million people (4% of population) have been diagnosed with the condition and the majority of these (85–95%) have type 2 diabetes. It is estimated that a further 500,000 individuals may have undiagnosed type 2 diabetes.

The prevalence increases with age (Fig. 22.1) and lower socio-economic status. It is also varies with ethnicity (~×6 times higher in Bangladeshi and Pakistani communities and ~×4 higher in Black Caribbean women compared to white population).[2] Increasing rates of obesity are closely associated with rising prevalence of diabetes.

[1] World Health Organization (2011). http://www.who.int/diabetes/en/.
[2] Congdon, P. (2006). Estimating diabetes prevalence by small area in England. *J. Publ. Health* **28,** 71–81.

The overall incidence of diabetes can be reduced by preventing and reducing overweight and obesity, particularly central (abdominal) obesity. Individuals at risk can reduce this by eating a balanced diet, losing weight, and increasing their physical activity levels.

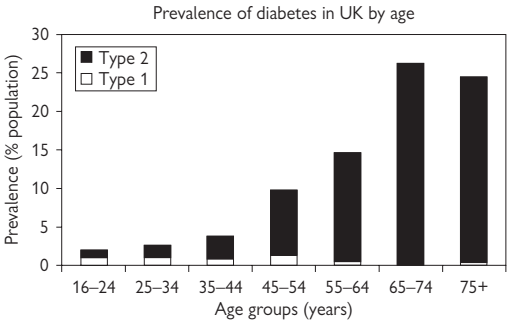

Fig. 22.1 Prevalence of diagnosed diabetes in England age (2006). (Figure drawn from data from the British Heart Foundation Statistics Website, http://www.heartstats.org/datapage.asp?id=1106).

Table 22.1 Values for diagnosing diabetes mellitus and other categories of hyperglycaemia*

	Venous† plasma glucose concentration	
	mmol/l	**mg/dl**
Diabetes		
Fasting *or*	≥7.0	≥126
2 h after 75 g oral glucose load	≥11.1	≥200
Impaired glucose tolerance		
Fasting *and*	<7.0	<126
2 h after 75 g oral glucose load	≥7.8 and <11.1	≥140 and <200
Impaired fasting glycaemia		
Fasting *and*	6.1–6.9	110–125
2 h after 75 g oral glucose load	<7.8	140

*World Health Organization (2006). *Definition and diagnosis of diabetes mellitus and intermediate hyperglycaemia*, Report of a WHO/IDF consultation. World Health Organization, Geneva.

†Venous plasma glucose should be the standard method of measurement. Values for capillary and venous fasting glucose are the same.

Note that since publication of this WHO report, the American Diabetes Association (2010) has recommended that haemoglobin A_{1c} can be used to diagnose diabetes and, although not superior to other tests, is faster and easier.

Contributing causes and clinical consequences

Contributing causes

Type 1 diabetes

Insulin secretion by the pancreatic β cells is reduced following damage mediated by an autoimmune T-cell reaction. Environmental factors, such as viral infection, may be implicated.

Type 2 diabetes

Type 2 diabetes is closely associated with obesity and genetic factors. Approximately 15–25% of first-degree relatives of people with type 2 diabetes develop impaired glucose tolerance or diabetes. However, the substantial contribution made by obesity is particularly important as this is a potentially modifiable risk factor. Excess body fat, stored as lipid in the adipocytes, is associated with ↑ levels of circulating hormones, cytokines, and metabolic fuels (e.g. free fatty acids), which modulate the effect of insulin. Large adipocytes, especially in abdominal fat, are resistant to the lipolytic effects of insulin, which leads to a further release of free fatty acids and ↑ in circulating levels. These changes inhibit the insulin signalling cascade resulting in impaired glucose metabolism in skeletal muscle and stimulated hepatic gluconeogenesis with consequent hyperglycaemia.

Clinical consequences

Diabetes is associated with ↑ risk of serious chronic ill health, disability, and premature mortality. Long-term complications include macrovascular disease, leading to cardiovascular disease and ↑ risk of stroke, and microvascular disease leading to retinopathy, nephropathy, and neuropathy. People with diabetes also have a greater risk of suffering from infections, cataracts, and depression. Many of these complications can be minimized or avoided by earlier diagnosis and more effective treatment (see Box 22.2).

Box 22.2 Benefits of good blood glucose control in type 1 and type 2 diabetes (Diabetes Control and Complications Trial 1993; UK Prospective Diabetes Study 1998)

- New eye disease risk reduced by 76%
- Worsening of existing eye disease reduced by 54%
- Early kidney disease risk reduced by 54%
- More serious kidney problems reduced by 39%
- Nerve damage risk reduced by 60%
- Heart disease risk reduced by 56%
- Stroke risk reduced by 44%
- Kidney disease risk reduced by up to 33%.

Key priorities for management

Type 1 diabetes[1]

See Table 22.2.

Table 22.2 Care of adults, young people, and children with Type 1 diabetes

Adults	Children and young people
• Patient-centred care • Multidisciplinary team approach • Education • Blood glucose control • Arterial risk-factor control • Management of late complications	• Management from diagnosis • Education • Monitoring glycaemic control • Screening for complications and associated conditions • Psychosocial support

Education is a central part of management of type 1 diabetes for adults, children, and young people and dietary aspects play an important role:

- Offer nutritional information from diagnosis onwards
- Provide information that is sensitive to personal needs and culture
- Support individual sessions and education programmes
- Advice provided by professionals with specific and approved training
- Include excess weight, underweight, eating disorders, raised blood pressure, renal failure
- Aim to enable people to make optimal choices about foods they consume
- Facilitate insulin dose changes when taking different quantities of those foods
- Review education needs annually
- Ongoing education with access to information and opportunities for discussion at clinic visits
- Tailor according to maturity, culture, existing knowledge, and wishes of child/young person and family
- Advise on effects of nutritional changes on glycaemic control
- Give support to help optimize weight
- Discuss timing and composition of snacks and problems associated with fasting and feasting
- Multiple daily injections: adjust insulin to carbohydrate intake.

[1] Adapted from NICE (2004). *Type 1 diabetes.* Clinical guideline 15, updated 2010 (separate versions for Adults and for Children and young people). http://guidance.nice.org.uk/CG15/Guidance.

Type 2 diabetes[1]

Adults

- *Patient education:* every person and/or their carer should be offered education at diagnosis and annually.
- Dietary advice should be individualized and provided by a healthcare professional with specific competence in nutrition.
- *Setting target for HbA_{1c}:* generally set at 6.5% for type 2 diabetes, but individuals should be involved in setting own targets and may be >6.5% (see 📖 this Chapter, 'Weight management and monitoring glycaemic control', p. 459). Targets of <6.5%, which require highly intensive management should be avoided.
- *Self-monitoring:* plasma glucose monitoring should only offered within the context of self-management education and this should include interpretation of results and action required.
- *Insulin therapy, if required:* when starting, a structured programme should include frequent self-monitoring, titration of insulin dose to target HbA_{1c}, dietary aspects, self-monitoring, management of hypoglycaemic, support from experienced healthcare professionals and continuing telephone support.

[1] Adapted from NICE (2009). *Type 2 diabetes.* Clinical guideline 87, updated 2010. http://guidance.nice.org.uk/CG87/Guidance.

Goals and principles of dietary management

Goals of dietary management

- To maintain or improve health through the use of appropriate and healthy food choices.
- To achieve and maintain optimal metabolic and physiological outcomes including:
  - reduction of risk for microvascular disease by achieving near normal glycaemia without undue risk of hypoglycaemia;
  - reduction of risk of macrovascular disease including management of body weight, dyslipidaemia, and hypertension.
- To optimize outcomes in diabetic nephropathy and in any concomitant disorder such as coeliac disease or cystic fibrosis.

Dietary advice should be placed within a holistic context of care, which includes supporting patients to manage their diabetes and make decisions; it should also complement other treatment including pharmacological, physical activity, behavioural, and smoking cessation programmes. If it is to be effective, dietary advice must take account of the individual's personal preferences, cultural background, and lifestyle.

Principles of dietary management

People with diabetes do not need to follow a 'special diet' or comply with narrow restrictions and measured portions of food that were considered central to dietary advice in previous years (but see 📖 this Chapter, 'DAFNE,', p. 458). However, eating well is essential for good diabetes control and can contribute to improved well-being and improved long-term health outcomes. The optimum healthy choice of food for people with diabetes is the same as for the general population (see 📖 Chapter 2, 'The Eatwell Plate', p. 27) and ideally should be low in fat, sugar, and salt, include plenty of fruit and vegetables, and base meals on starchy foods such as bread, potatoes, and rice (Table 22.3). Patients with newly diagnosed diabetes should have access to dietary advice; those with type 1 diabetes should be referred to a dietitian for individual advice while those with type 2 diabetes should be referred to a structured education programme (discussed later in this chapter) or seen by a dietitian. After this time, further dietetic intervention should be offered when required, e.g. at annual review or when deemed appropriate by another healthcare professional or the person with diabetes.

Ten steps to healthy eating in diabetes[1]

The composition of optimum diet above must be translated into practical advice which gives patients guidance about what they should eat. This should be facilitated on an individual basis by a registered dietitian. The following guidelines provide a rough starting point:

- Eat three meals a day and avoid skipping meals. Spread breakfast, lunch and evening meal across the day.

[1] Adapted from Diabetes UK (August 2010) (℞ www.diabetes.org.uk).

- Include starchy carbohydrate foods such as bread, potatoes, rice, pasta, and chapattis at each meal. The better choices include pasta, wholegrain and granary bread, oats, new potatoes and yam.
- Reduce intake of fat, especially saturated fat. Eat less butter, margarine, cheese, and fatty meat and instead choose low fat dairy foods, lean meat and fish. Replace fried foods with grilled, steamed, or oven baked items. Use small quantities of mono-unsaturated oil, e.g. olive oil or rapeseed oil.
- *Eat more fruit and vegetables:* aim for at least five portions per day.
- Include more beans and pulses, for example kidney beans, butter beans, chick peas and lentils.
- *Oily fish:* aim for at least two portions per week. These could include mackerel, sardines, salmon, and pilchards.
- Reduce sugar and sugary foods. Following a strict sugar-free diet is not necessary—sugar can be used as an ingredient in foods, e.g. in wholegrain breakfast cereals. Sugary drinks can be replaced by sugar-free or diet alternatives.
- Cut down on salt by limiting the amount of processed foods consumed as well as added table salt. Herbs and spices can be used as an alternative.
- Drink alcohol in moderation which is a maximum of two units per day for women and three units per day for men. Be aware that the alcohol content of drinks has gone up (for information on alcohol units, see 📖 Chapter 9, 'Alcohol', p. 186). Avoid drinking alcohol on an empty stomach as it may contribute to hypoglycaemia.
- Diabetic food products offer no health benefits, often contain high levels of energy, can have a laxative effect and are expensive.

Role of glycaemic index in diabetes

The glycaemic index (GI) aims to quantify the blood sugar response after eating specific foods. This has potential significance in diabetes where fluctuations or rapid increases in blood sugar are undesirable. However, the issue is complex and GI values vary considerably depending on the exact nature of the food (e.g. method of processing or cooking, degree of ripeness, or strain of plant), and whether they are eaten alone or accompanying a meal providing mixed macronutrients. At present, it is recommended that using GI values to provide a broad guide to a food's glycaemic effect may be a helpful adjunct to other dietary issues, but that it should not be relied upon as the most important feature of food-related advice (Table 22.4; see 📖 Chapter 5, 'Glycaemic index', p. 77).

The potential benefits from ↓ intake of high GI foods and replacing them with ↑ low GI food include:

- Helping to maintain a more even blood glucose level, thus ↓ both hypoglycaemia and high blood sugar levels sometimes observed after meals.
- Greater satiety, thus potentially helping people to avoid excessive intake.
- Improvements in lipid profile (↑ HDL cholesterol, i.e. good), which is associated with ↓ cardiac risk.

Table 22.3 Composition of the optimum diet in diabetes*

Protein	≤1 g/kg body weight
Total fat	<35% of energy intake
Saturated and transunsaturated fat	<10% of energy intake
n-6 polyunsaturated fat	<10% of energy intake
n-3 polyunsaturated fat	Eat fish, especially oily fish, once or twice weekly. Supplements not recommended
cis-monounsaturate fat	10–20% of energy intake‡
Total carbohydrate†	45–60% of energy intake‡
Sugar	Up to 10% of energy provided eaten in context of healthy diet
Fibre	No quantitative recommendation. Soluble fibre has beneficial effect on glycaemic and lipid metabolism. Insoluble fibre also has health benefits
Vitamins and anti-oxidants	Encourage foods naturally rich in vitamins and antioxidants. Supplements are not recommended
Salt	≤6 g NaCl per day

* Adapted from Connor, H., Annan, F., Bunn, E., *et al.* (2003). The implementation of nutritional advice for people with diabetes. *Diabet Med.* **20,** 786–807.

† See 🕮 'Role of glycaemic index in diabetes', p. 449 and Chapter 5, 'Carbohydrate', p. 72.

‡ 60–70% of energy intake should come from combined monounsaturated fat and carbohydrate.

Table 22.4 Guide to the glycaemic effect of various foods

Lower GI	Apples, oranges, pears, peaches, pasta, oats and oat products, pulses, peas, beans, legumes, sweet potato
Moderate GI	White basmati rice, new potatoes, cous cous, pitta bread, Shredded Wheat, Weetabix, honey, jam
Higher GI	White rice, white and wholemeal bread, baked and mashed potatoes, cornflakes, glucose

Further information is available from http://www.glycemicindex.com/

Diet and insulin

The hypoglycaemic action of injected insulin should be balanced against the glycaemic effects of consuming carbohydrate. Ideally, the insulin prescribed should be selected on the basis of the compatibility of its timing and mode of action with each patient's lifestyle and meal habits, rather than the other way round.

The availability of short-, intermediate-, and long-acting insulin and the ability to administer these via a pen-like injection device increases the flexibility of insulin regimes to meet most meal patterns. However, individuals with very erratic lifestyles and eating habits will benefit from more regular intake of food, i.e. three meals daily and no meal-skipping, although this does not need to follow a rigid, clock-watching meal pattern. Rapid-acting analogue insulin starts to work within 5–15 minutes of injection and ∴ eating some carbohydrate within this time period is necessary (see Tables 22.5 and 22.6; see also Box 22.5). All patients taking insulin, but particularly those taking analogue insulin, must be aware of the possibility of hypoglycaemia (see 📖 this Chapter, 'Hypoglycaemia', p. 457).

As insulin is anabolic, increasing the dose to control the glycaemic effects of excessive food intake may lead to weight gain and potentially to a cycle of worsening blood sugar control. It is important, ∴ that dietary advice to people taking insulin does not simply focus on the short-term glycaemic effects of food and the need to balance the effects of insulin, but also incorporates other aspects of the diet described above.

Table 22.5 Examples of analogue insulins

Non-proprietary name	Proprietary name
Insulin aspart	NovoRapid® Novo Nordisk, Crawley
Insulin detemir	Levemir® Novo Nordisk, Crawley
Insulin glargine	Lantus® Sanofi-Aventis, Guildford
Insulin lispro	Humalog® Lilly, Basingstoke

Table 22.6 Examples of commonly used insulins*

Proprietary name (manufacturer)	Source	Form†	Injection‡	Effect§		
				Onset	Peak	End
Rapid-acting analogue						
Humalog (Lilly)	Analogue	CDV	–15 to +5	0.1	0.2–2	4
NovoRapid (Novo Nordisk)	Analogue	CD	–15 to +5	0.1	0.2–3	4.5
Short-acting insulins						
Actrapid (Novo Nordisk)	Human	V	–30	0.1	0.5–2.5	8
Humulin S (Lilly)	Human	CV	–45 to –20	0.1	0.5–2.5	7
Medium and long-acting insulins						
Hypurin Bovine PZI (Wockhardt UK)	Beef	V	–30	4	10–20	36
Insulatard (Novo Nordisk)	Human	CDV	–30	0.1	1–12	24
Insuman Basal (Sanofi–Aventis)	Human	CDV	–60 to –45	0.1	1.5–4	20
Mixed insulins						
Humulin M3 (Lilly)	Human	CV	–45 to –20	0.1	1–8	22
Hypurin Porcine 30/70 (Wockhardt UK)	Pork	CV	–30 to –15	0.1	4–12	24
Mixtard 30 (Novo Nordisk)	Human	CDV	–30	0.1	0.5–8	24
Analogue mixture						
Humalog Mix 25 (Lilly)	Analogue	CD	–15 to +5	0.2	0.5–2.5	22
NovoMix 30 (Novo Nordisk)	Analogue	CD	–15 to +5	0.1	0.5–4	24
Long-acting analogues						
Lantus (Sanofi-Aventis)	Analogue	CD	1 x daily	0.1	—	24
Levemir (Novo Nordisk)	Analogue	CD	1–2 x daily	0.1	—	24

*© Diabetes UK. This information has been reproduced from Balance, July–August issue 2005: p 44–45, with the kind permission, for first edition, of Diabetes UK. Updated from British National Formulary, 2010.

†C, Cartridge; D, pre-filled disposable injection device; V, vial.

‡Optimum time (minutes) of injection in relation to food or, for medium-and long-acting insulin, in relation to bedtime.

§Approximate time (h) after injection for effect on blood sugar.

Box 22.5 Dietary management of adults treated with insulin analogues (IA)[1]

Dietitians should play an integral role in determining the appropriate insulin regimen in order to assist the patient to achieve optimal glycaemic control.

Meals

- Consider post-prandial injection of rapid-acting IA if person eats meal with unpredictable amount of CHO, e.g. buffet, carvery, etc.
- Consider postprandial injection of rapid-acting IA if person eats meal that may → ↓ post-prandial blood glucose, i.e. low GI or high fat meal, especially if pre-meal blood glucose is <6 mmol/l.
- If pre-meal blood glucose is >6 mmol/l, then consider pre-prandial injection of rapid-acting insulin analogues (IA) even if meal includes slowly absorbed CHO.
- If meal is low GI or high fat, consider dividing bolus of rapid-acting IA to ↓ risk of delayed hypoglycaemia.
- If meal is low GI, consider ↓ dose of rapid-acting IA. This may → postprandial hyperglycaemia.
- Dose of insulin should be adjusted primarily on the CHO content of the meal.
- If blood sugar ↑ by >2–3 mmol/l after meal, review insulin to CHO ratio.

Snacks

- For patients using glargine, supper snack is usually unnecessary. Use blood glucose and preference to determine if supper snack is needed after a 1–2-week settling period.
- If large amounts of alcohol are consumed, a supper snack may be needed to prevent hypoglycaemia even if glargine is used.
- People treated with rapid-acting IA and glargine may require extra bolus of rapid acting IA if substantial snacks, i.e. >15 g CHO, consumed between meals.
- Between meal snacks are generally considered unnecessary for people treated with fast acting analogues. Advice to add snacks should be based on blood glucose and preference.

Exercise

- Dose of rapid-acting IA may need to be ↓ for exercise, i.e. as for soluble insulin. This will depend on changes in CHO intake, strenuousness and duration of exercise, and time between injections and exercise.
- Blood glucose before and after exercise should guide adjustments in dose of rapid-acting IA.
- Do not give insulin bolus if extra CHO has been taken to compensate for the effect of exercise.

Insulin pumps

- Timing of bolus should be determined by meal components (CHO, fat, protein, GI), quantity consumed and timing.
- Bolus may be given as (depending on type of pump):
  - *standard (or 'normal'):* complete dose is delivered immediately. Best for meals where food is mostly CHO (unless low GI, e.g. porridge);

Box 22.5 (*Contd.*)

- *delayed:* given 15–30 min later. Best for meals with ↑ fat, ↑ protein, ↓ GI or where CHO content cannot be anticipated.
- *split:* delivered in ≥2 doses. The first given just before eating and next 15–60 min later. Best for meals with mixture of CHO, fat, and protein.
- *extended or square wave:* delivered over extended period up to 4 h. Best for meals with ↑ fat, ↑ protein, ↓ GI or multi-course meals eaten over period of time.

[1] Vaughan, L. (2005). *Suggested good practice for dietitians involved in the dietetic management of adults with type 1 diabetes treated with insulin analogues.* Diabetes Management and Education Group of the British Dietetic Association, London.

Diet and oral hypoglycaemic drugs

In type 2 diabetes, if haemoglobin A_{1c} remains >6.5% (or agreed individual target) in spite of life style intervention, i.e. facilitated dietary and physical activity advice, oral hypoglycaemic drugs (Table 22.7) should be considered. NICE guidelines[1] provide algorithms describing an evidence-based process.

Table 22.7 Hypoglycaemic drugs

Oral	
Biguanide	Metformin (e.g. Glucophage)
Sulphonylurea	Glibenclamide (e.g. Daonil), gliclazide (e.g. Diamicron), glipizide (e.g. Glibenese)
DPP-4 inhibitors	Sitagliptin (e.g. Januvia)
Thiazolidinediones	Rosiglitazone (e.g. Avandia)
Prandial glucose regulator	Repaglinide (e.g. Prandin)
Alpha glucosidase inhibitor	Acarbose (e.g. Glucobay)
Non-oral hypoglycaemic agents (i.e. for injection)	
Incretin mimetic	Exenatide (e.g. Byetta)

Taking this medication does not change the goals or principles of the optimum diet described above, but patients should be aware of the potential for weight gain if prescribed sulphonylurea or thiazolidinediones, and of the small possibility of hypoglycaemia.

[1] NICE (2009). *Type 2 diabetes.* Clinical guideline 87, updated 2010. Available at: http://guidance.nice.org.uk/CG87/Guidance.

Hypoglycaemia

Causes

- Too much insulin or oral hypoglycaemic medication.
- Missed or delayed meal or snack.
- Meal or snack providing insufficient carbohydrate.
- Strenuous or prolonged physical activity.
- Consuming too much alcohol or drinking on an empty stomach.
- Occasionally, no obvious cause.

Symptoms

Symptoms vary between individuals and with the severity of the hypoglycaemia:

- Hunger.
- Trembling or feeling shaky.
- Sweating.
- Anxiety or irritability.
- Pallor.
- Fast pulse or palpitations.
- Tingling lips.
- Blurred vision.

Signs of a more severe hypoglycaemia:

- Difficulty in concentrating.
- Vagueness or confusion.
- Irrational behaviour.

Treatment

Acute hypoglycaemia should be treated with 10–20 g of glucose orally in conscious patients, i.e. glass of fizzy drink containing glucose or fruit juice, >3 glucose tablets, 5 sweets, e.g. jelly babies. If this is not available, regular sugar (sucrose) or a sugary drink can be given, although this will not be effective in patients taking the α-glucosidase inhibitor, acarbose. If unconscious, no oral intake should be given, inject glucagon if available and trained to do so or call for ambulance.

On recovery, a further 10–20 g of slower-acting carbohydrate (see Box 22.7) should be given unless the next meal or snack is due, in which case this should be eaten as usual.

If episodes of hypoglycaemia are frequent, treatment regime in context of current intake should be reviewed.

Box 22.7 Suitable snacks after initial recovery from 'hypos' (hypoglycaemia)

- Sandwich—ideally made from wholegrain bread
- Bowl of wholegrain or oat-based cereal
- Banana and glass of semi-skimmed milk
- Oat cakes.

Structured education in diabetes

DAFNE

'Dose Adjustment For Normal Eating' is a structured training programme in intensive insulin therapy and self-management. The programme is delivered to small groups (6–8 people) over five consecutive days by specifically-trained healthcare professionals with a follow-up session about 8 weeks later. The principles are to teach flexible insulin adjustment to match carbohydrate in a free diet on a meal-by-meal basis with an emphasis on self-management and independence from the diabetes care team. Nutrition education sessions cover how to identify macronutrients in the diet and estimate carbohydrate portions, weight control, and healthy eating. This intensive programme started in Germany in the 1980s and was introduced into limited centres in the UK in 1999. Analysis of data[1] indicates improved glycaemic control and quality of life and the programme is being expanded to more areas in the UK.

Further information http://www.dafne.uk.com.

DESMOND

The 'Diabetes Education and Self-Management for Ongoing and Newly Diagnosed' programme is a similar, multicentred, structured educational approach, but designed for people with type 2 diabetes. Evaluation[2] has indicated that the programme is associated with improved weight loss and smoking cessation but not improvements in HbA_{1c}.

Other

Several other structured education programmes have been developed, e.g. ASPIRE, BERTIE, DEAL, SNACCS, and X-PERT. Not all have been evaluated effectively, but a systematic review[3] of those examined by randomized controlled trials in type 2 diabetes has concluded that structured programmes were associated with a significantly greater improvement in HbA_{1c} in patients with high baseline values, i.e. >8%, but not overall.

[1] Speight, J., Amiel, S.A., Bradley, C., *et al.* (2010). Long-term biomedical and psychosocial outcomes following DAFNE (Dose Adjustment For Normal Eating) structured education to promote intensive insulin therapy in adults with sub-optimally controlled type 1 diabetes. *Diabetes Res. Clin. Pract.* **89,** 22–9.

[2] Davies, M.R., Heller, S., Skinner, T.C., *et al.* (2008). Effectiveness of the diabetes education and self management for ongoing and newly diagnosed (DESMOND) programme for people with newly diagnosed type 2 diabetes: cluster randomised controlled trial. *Br. Med. J.* **336,** 491–5.

[3] Duke, S.A., Colagiuri, S., Colagiuri, R., *et al.* (2009). Individual patient education for people with type 2 diabetes mellitus. *Cochrane Database of System. Rev.* CD005268.pub2.

Weight management and monitoring glycaemic control

Weight management

More than 80% of people diagnosed with type 2 diabetes are overweight. Weight management in both type 1 and type 2 diabetes is important to help to reduce insulin resistance, control blood glucose levels, and lower the risk of long-term complications. Although preventing weight gain and/or reducing excess body weight can be very challenging, it is central to optimizing diabetes care and is a cornerstone in the dietary management of diabetes. NICE guidelines for type 2 diabetes advise a loss of 5–10% of body weight as an initial target but that smaller losses can still be beneficial. Ideally weight loss targets should be individualized in the context of current body mass index, physical exercise. A multifaceted approach utilizing dietary and physical activity advice, drug therapy, and behavioural support is required.

Monitoring of glycaemic control

Glycosylated haemoglobin (HbA_{1c}) reflects blood glucose levels over the preceding 3 months. Values <6.5% are considered a desirable target for most patients with type 1 and type 2 diabetes, and are associated with a reduced risk of complications. People with severe risk of hypoglycaemia should aim for a target of <7.5%.

Conversion of HbA_{1c} from % to mmol/mol (which will be used from 2011 in the UK)

- 6.0% → 42 mmol/mol.
- 6.5% → 48 mmol/mol.
- 7.0% → 53 mmol/mol.
- 7.5% → 58 mmol/mol.
- 8.0% → 64 mmol/mol.

Diabetes in pregnancy, children, and young people

Diabetes in pregnancy

Women with diabetes represent approximately 2–5% of all pregnancies in England and Wales and comprise:

- Gestational diabetes ~87.5%
- Type 1 diabetes ~8.5%
- Type 2 diabetes ~2.0%

The prevalence of type 1 and type 2 diabetes is increasing, especially in people from African, Black Caribbean, South Asian, Middle Eastern and Chinese origins. A body mass index >30 kg/m^2 is an independent risk factor for developing gestational diabetes.

Gestational diabetes (GDM) is usually an asymptomatic form of intolerance of carbohydrate leading to hyperglycaemia with onset or first recognition in pregnancy. In most cases it resolves spontaneously after delivery, but it is associated with adverse pregnancy outcomes including large-for-gestational-age babies, congenital abnormalities, and the long-term risk of type 2 diabetes in the mother.

Most cases are treated with nutritional management alone although some women also require medication (oral hypoglycaemics or insulin—see NICE guidelines[1]). A meta-analysis[2] of intervention studies indicates that treatment (in most cases, diet ± insulin) was associated with fewer perinatal complications. However, the analysis did not explicitly consider the dietary aspects of the interventions. In the absence of specific evidence about diet, current guidance[1] is:

Pre-conception

- Women with diabetes who are planning pregnancy should be offered individual dietary advice.
- When planning to become pregnant, women with diabetes and a body mass index >27 kg/m^2 should be offered advice about how to lose weight (see 🕮 Chapter 21, 'Weight management: overview', p. 418, and NICE health guidance[3]).
- When planning to become pregnant, women with diabetes should be advised to take folic acid supplements (5 mg/day) until 12 weeks gestation.

Gestational diabetes

- Women with GDM should be offered information about the role of diet, body weight and exercise and the risk of the baby developing obesity ± diabetes in later life.
- Women with GDM should be advised to choose dietary CHO from low glycaemic index sources (see 🕮 Chapter 5, 'Carbohydrate', p. 72), protein from lower fat foods (i.e. leaner meat, low fat dairy products, pulses) and oily fish (see 🕮 Chapter 12, 'Eating fish in pregnancy', p. 223)

[1] NICE (2009). *Diabetes in pregnancy*. Clinical guideline 63. Available at: http://guidance.nice.org.uk/CG63/Guidance.

[2] Horvath, K., Koch, K., Jeitler, K., *et al.* (2010). Effects of treatment in women with gestational diabetes mellitus: systematic review and meta-analysis. *Br. Med. J.* **340,** c1395.

and use polyunsaturated or monounsaturated fats in preference to saturated fat.
- Women with GDM with pre-pregnancy BMI >27 kg/m^2 should be advised to restrict energy intake to ≤25 kcal/kg/day and to take moderate exercise (≥30 min/day). See also 🕮 Chapter 12, 'Maternal weight gain', p. 228.

Post-natal care

- Women with insulin treated pre-existing diabetes should be informed that they are at risk of hypoglycaemia in the post-natal period, especially when breastfeeding and should be advised to have a meal or snack available before or during feeds.
- Women diagnosed with GDM should be offered lifestyle advise (weight control, diet and exercise) and offered a fasting plasma glucose measurement at 6-week post-natal check and annually afterwards.

Diabetes in children and young people

Children and young people with diabetes should be offered a care package by a multidisciplinary paediatric diabetes care team who operate both in the hospital and the community and who liaise closely with primary healthcare and school. The team should include a dietitian with expertise in childhood diabetes. Among the dietary management issues that must be considered on an individual basis are:
- the need to provide the nutritional requirements associated with growth as well as the optimum diet for diabetes;
- the psycho-social aspects of dietary intervention in a young person.

See 🕮 this Chapter, 'Key priorities for management', p. 446, and NICE guidelines[3,4] for type 1 diabetes.

[3] NICE (2010). *Weight management before, during and after pregnancy*. Public health guideline 27. Available at: http://guidance.nice.org.uk/PH27/Guidance/pdf/English.
[4] NICE (2004). *Type 1 diabetes*. Clinical guideline 15, updated 2010 (separate version for Children and young people). Available at: http://guidance.nice.org.uk/CG15/Guidance.

Metabolic syndrome

Closely associated with diabetes, metabolic syndrome is also known as syndrome X and the insulin resistance syndrome. It comprises interrelated factors including obesity (particularly central), insulin resistance, glucose intolerance/diabetes, hyperlipidaemia, and hypertension, all of which predispose to cardiovascular disease. See Table 22.8 for diagnostic criteria.

Tables 22.8 Consensus diagnostic criteria for metabolic syndrome*

Central obesity (i.e. waist circumference above ethnicity-specific cut-off, see Table 22.9) and at least one of the following:	
Raised triglycerides	≥1.7 mmol/l (150 mg/dl) or specific treatment for this lipid abnormality
Reduced HDL cholesterol	♂ <1.03 mmol// (40 mg/dl) ♀ <1.29 mmol/l (50 mg/dl) or specific treatment for this lipid abnormality
Raised blood pressure	Systolic ≥130 mmHg *or* Diastolic ≥85 mmHg
Raised fasting plasma glucose	≥5.6 mmol/l (100 mg/dl) or previously diagnosed type 2 diabetes.

* Alberti, K.G.M.M., Zimmet, P., and Shaw, J.(2006). Metabolic syndrome—a new world wide definition. A consensus statement from the International Diabetes Federation. *Diabet Med* **23**, 469–80.

Table 22.9 Country/ethnic specific values for identifying central obesity using waist circumference*

Country/ethnic group	Waist circumference (cm)	
	Male	Female
Europids	≥94	≥80
South Asians	≥90	≥80
Chinese	≥90	≥80
Japanese††	≥90	≥80
Ethnic South and Central Americans	Use South Asian data†	
Sub-Saharan Africans	Use European data†	
Eastern Mediterranean and Middle Eastern (Arab) populations	Use European data†	

* Alberti, K.G.M.M., *et al.* (2007). International Diabetes Federation: a consensus on type 2 diabetes prevention. *Diabet Med* **24,** 451–63.

† Until specific data available.

†† Lack of consensus about values in Japanese: These values show best agreement with cardiovascular and diabetes risk. Visceral fat correlates best with 85 cm in men and 90 cm in women.

The individual components of metabolic syndrome can be treated by a healthy diet based on 'The Eatwell Plate' (see 📖 Chapter 2, p. 27) and incorporating specific advice relevant to the factors present (e.g. obesity, diabetes, or dyslipidaemia). The fundamental message is that, to avoid this syndrome, people should avoid excessive weight gain and stay physically active.

Further information on diabetes

- DAFNE programme. Available at: http://www.dafne.uk.com.
- Diabetes UK. Available at: http://www.diabetes.org.uk.
- Glycaemic index information, University of Sydney. Available at: http://www.glycemicindex.com/.

Alberti, K.G.M.M., Zimmet, P., and Shaw, J. (2007). International Diabetes Federation: a consensus on type 2 diabetes prevention. *Diabet. Med.* **24**, 451–63.

Davies, M.R., Heller, S., Skinner, T.C., *et al.* (2008). Effectiveness of the diabetes education and self management for ongoing and newly diagnosed (DESMOND) programme for people with newly diagnosed type 2 diabetes: cluster randomised controlled trial. *Br. Med. J.* **336**, 491–5.

Duke, S.A., Colagiuri, S., Colagiuri, R. (2009). Individual patient education for people with type 2 diabetes mellitus. *Cochrane Database of System. Rev.* CD005268.pub2.

Horvath, K., Koch, K., Jeitler, K. (2010). Effects of treatment in women with gestational diabetes mellitus: systematic review and meta-analysis. *Br. Med. J.* **340**, c1395.

NICE (2004). *Type 1 diabetes.* Clinical guideline 15, updated 2010 (separate versions for Adults and for Children and young people). Available at: http://guidance.nice.org.uk/CG15/Guidance.

NICE (2009). *Type 2 diabetes.* Clinical guideline 87, updated 2010. http://guidance.nice.org.uk/CG87/Guidance.

NICE (2010) *Weight management before, during and after pregnancy.* Public health guideline 27. Available at: http://guidance.nice.org.uk/PH27/Guidance/pdf/English.

Speight, J., Amiel, S.A., Bradley, C., *et al.* (2010). Long-term biomedical and psychosocial outcomes following DAFNE (dose adjustment for normal eating) structured education to promote intensive insulin therapy in adults with sub-optimally controlled Type 1 diabetes. *Diabetes Res. Clin. Pract.* **89**, 22–9.

World Health Organization (2006). *Definition and diagnosis of diabetes mellitus and intermediate hyperglycaemia. Report of a WHO/IDF consultation.* World Health Organization, Geneva.

Chapter 23

Cardiovascular disease

Classification, prevalence, and contributing causes

Classification

Cardiovascular disease (CVD) includes the following.

- *Coronary heart disease:* narrowing of the lumen of arteries supplying blood to the heart muscle as a result of atheromatous plaque on the arterial walls. This limits the blood supply to the heart muscle causing pain (angina) and breathlessness on exertion. Damaged plaque leads to a clotting response, which may result in a thrombus detaching from the artery wall and occluding the lumen with subsequent heart muscle death (myocardial infarction).
- *Cerebral infarction:* thrombus occlusion of an artery supplying blood to the brain leading to irreversible damage to brain tissue (stroke or transient ischaemic attack). The thrombus may arise from atheromatous plaque from within the brain or another blood vessel and the risk of occlusion is ↑ in narrowed arteries.
- *Peripheral arterial disease:* atheromatous plaque leads to narrowing of peripheral blood vessels, most commonly in the legs. This results in poor blood supply and pain on exertion (claudication).

Prevalence

Globally, CVD is the greatest cause of death, ~17 million (29%) per year. The prevalence is increasing in countries adopting a Western diet and lifestyle (see Box 23.1 for contributing factors), and with rates rising rapidly in developing countries in parallel with obesity, urbanization, and longevity (Fig 23.1). The greatest ↑ in CVD deaths in men between 1992 and 2002 were reported from Ukraine, the Russian Federation and Kazakhstan; over the same period, the greatest ↓ were reported from the Czech Republic, Finland and the UK.

The CVD mortality rate has fallen in the UK since peak levels in the early 1970s, because of improvements in prevention, diagnosis, and treatment and ↓ prevalence of smoking, although CVD remains the most common cause of death (Table 23.1).

CVD prevalence is higher in men than women before the menopause and increases with age. It is higher in Scotland and the north of England compared to the south of England and is higher than average in lower socio-economic groups and South Asians.

Table 23.1 Deaths and morbidity from circulatory system disease and coronary heart disease (CHD) in the UK*

	Number (%)		
	Total	**Male**	**Female**
Circulatory system deaths, all	193 287 (33)	93 050	100 237
CHD deaths, ≤75 years	29 158 (15)	21 613	7 545
Coronary heart disease[1]	2 267 923 (3.6%)		

*Data from British Heart Foundation Statistics Website http://www.heartstats.org.

[1]Prevalence in England, Scotland and Wales.

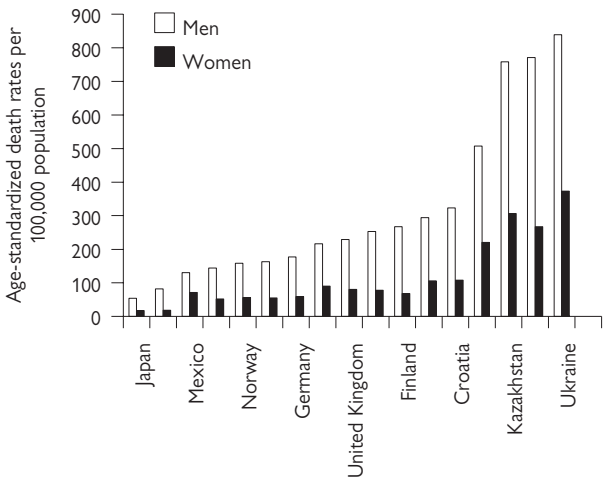

Fig. 23.1 Age-standardized death rates per 100,000 population from coronary heart disease in men and women in 2000 in selected countries. ICD codes 410–14 (8th and 9th revision), 120–5 (10th revision). Data from British Heart Foundation (2007) Coronary heart disease statistics http://www.heartstats.org/uploads/documents%5C48160_text_05_06_07.pdf (accessed 21 August 2010).

Box 23.1 Contributing causes

Confirmed risk factors/markers include

- Increasing age
- Male gender
- Females (post-menopause)
- ↓ Socio-economic status
- Ethnic background
- Physical inactivity*
- Smoking*
- Diabetes
- Obesity*
- High blood pressure*
- ↑ Serum total cholesterol*
- ↑ Serum LDL cholesterol*
- ↓ Serum HDL cholesterol*
- ↑ Serum triglycerides*

Other probable risk factors/markers include

- Other lipid-related factors (not cholesterol)*
- Vascular endothelial dysfunction
- Oxidative stress*
- Coagulation factors, e.g. fibrinogen
- Inflammatory markers, e.g. C-reactive protein
- Blood homocysteine*
- Maternal/fetal undernutrition
- Vitamin D deficiency*

* Factors that are or may be potentially amenable to modification by intervention.

Cardioprotective diet

Primary prevention

Dietary advice for cardiovascular risk is associated with health benefits in healthy adults.[1] Nutritional recommendations for the general population, including those with CVD risk factors, are given in Table 23.2. These are compatible with the UK food based guidelines, the Eatwell Plate (see 📖 Chapter 2, p. 27) and can be summarized as follows.[2]

- Eat oily fish at least once per week, e.g. sardines, salmon, fresh tuna.
- Eat five or more portions of fruit and vegetables per day.
- Reduce amount of all fat eaten, e.g. select lean meat and lower fat dairy products, use less oil and fat in cooking, reduce use of full fat spreads, eat less fried food and high fat foods such as cakes, biscuits, pastries, and savoury snacks.
- Choose oils/spreads that are higher in monounsaturates and lower in saturates, e.g. olive oil and rapeseed oil.
- Reduce salt intake by using less at table, in cooking, and salty foods.
- Eat more starchy foods, e.g. bread, potatoes, pasta, rice, etc.
- Drink alcohol sensibly, e.g. 2–3 units/day for women and 3–4 units/day for men.

Secondary prevention

Systematic review[3] of randomized controlled trials of dietary advice to individuals with CVD shows that dietary strategies save lives and improve health.

- For those with CVD, i.e. following an MI or with angina:
  - reduce saturated fat intake and replace totally or partially with unsaturated fats.
- Those with CVD who have had an MI, should follow the advice above and:
  - follow Mediterranean dietary advice, which includes ↑ *n*-3 fats, fruit and vegetables, wholegrains, pulses, nuts, and fresh foods, ↓ saturated fats and processed food;
  - aim for 2–3 large portions of oily fish per week or equivalent (0.5–1.0 g/day omega-3 fats from fish oil, preferably fish body oil, rather than cod liver oil). See Table 23.3.

Ideally, this advice should be provided by registered dietitians but where access is limited, their workload should be prioritized to:

- optimize the advice provided by other health professionals, e.g. cardiac rehabilitation nurses, practice nurses, etc.;
- support individuals who are struggling with dietary advice or who have other medical conditions, e.g. diabetes;
- provide high quality written information to all people with CVD.

[1] Brunner, Rees, K., Ward, K., *et al.* (2009). Dietary advice for reducing cardiovascular risk. *Cochrane Database of Systematic Reviews.* CD002128.pub3.

[2] Stanner, S. (2005). *Cardiovascular disease diet, nutrition and emerging risk factors.* Blackwell Publishing, Oxford.

[3] Mead, A., Atkinson, G., Albin, D., *et al.* (2006). Dietetic guidelines on food and nutrition in the secondary prevention of cardiovascular disease – evidence from systematic reviews of randomized controlled trails (second update, January 2006). *J. Hum. Nutr. Dietet.* **19,** 401–19.

Table 23.2 Nutritional recommendations for the general population*

Nutrient	Recommendation
Fat	
Total fat	Reduce to 35% food energy
Saturated fat	Reduce to 11% food energy
Monounsaturated fat	Increase to 13% food energy
n-6 polyunsaturates	Maintain at current 6.5% food energy; concern if intake >10% food energy
n-3 polyunsaturates	Increase intake to 0.45 g/day
Trans fatty acids	Reduce to <2% energy intake
Carbohydrates	
Starch, intrinsic and milk sugars	Increase to 39% food energy
Non-milk intrinsic sugars	Restrict to 11% food energy
Non-starch polysaccharides	Increase to 18 g/day (adults)
Sodium	Reduce salt intake from 9 to 6 g/day
Potassium	Increase intake to 3.5 g/day

*Stanner, S. (2005). *Cardiovascular disease: diet, nutrition and emerging risk factors*. Blackwell Publishing, Oxford.

Considering that the prevalence of CVD is higher in lower socio-economic groups and in certain ethnic groups and geographical regions, the advice given must be tailored to the target individual or group's needs so that it is practical and culturally appropriate.

Individuals who have either suffered recently from an MI or other CVD event or have a close family member who has, are often more amenable to acting on dietary advice.

Other lifestyle interventions

Dietary advice should be given in conjunction with guidance/support about other aspects of life including:

- promotion of physical activity, e.g. 30–60 minute session of moderately intense aerobic activity preferably daily or at least 3–4 times per week;[1] exercise training should be prescribed on an individual basis after clinical evaluation with a general recommendation of ≥150 minutes per week (ideally 3–4 h) including weight/resistance training twice weekly;[1]
- smoking cessation;
- stress management.

[1] Piepoli, M.F., Corrà, U., Benzer, W., *et al.* (2010). Secondary prevention through cardiac rehabilitation: from knowledge to implementation. A position paper from the Cardiac Rehabilitation Section of the European Association of Cardiovascular Prevention and Rehabilitation. *Eur. J. Cardiovasc. Prev. Rehabil.* **17**, 1–17.

Table 23.3 Oily fish providing n-3 fatty acids*

• Mackerel, fresh or frozen	Very high source[a]
• Kippers, fresh or frozen	
• Pilchards, canned in tomato sauce	
• Tuna or trout, fresh or frozen	
• Sprats or salmon, fresh or frozen	
• Mackerel, smoked or canned	
• Sardines, fresh or frozen	
• Herring, pickled, fresh or frozen	
• Sild or skippers, canned	
• Salmon, canned in brine or in pasta dishes or smoked salmon	
• Crab, fresh	
• Herring, canned	
• Swordfish[b]	
• Salmon fishcakes or potato-topped pies	
• Salmon fish paté	
• Tuna canned in oil	
• Crab canned in brine	
• Eel, fresh or jellied	
• Fish paste, e.g. crab, salmon, sardine	
• Cod or haddock, fresh or frozen	
• Fish cakes or fish fingers (white)	
• Tuna, canned in brine	Low source

* UK Heart Health and Thoracic Dietitians Specialist Group (2007). *Heart disease and omega-3 fats*. BDA.

[a] Fish are ranked in order of omega-3 content in average portion. Those above the dotted line are considered high sources, providing ≥1 g omega-3 per average serving.

[b] Eat swordfish, shark and marlin only once per week.

For advice about eating fish for women who are pregnant or trying to conceive, see 📖 Chapter 12, p. 217.

Familial hypercholesterolaemia

Diagnosis

A definite diagnosis of familial hypercholesterolaemia (FH) should be made if a person has plasma cholesterol concentration described in Table 23.4 and tendon xanthomas or evidence of these signs in 1° or 2° relative or who have DNA evidence of LDL-receptor mutation, familial defective apo B-100 or a PCSK9 mutation.[1]

Table 23.4 Plasma cholesterol levels used as diagnostic criteria in FH[1]

	Total cholesterol (mmol/l)	LDL cholesterol (mmol/l)
Child / young person	>6.7	>4.0
Adult	>7.5	>4.9

Management

Lifestyle advice should be a component of management but not an alternative to lipid-modifying drug therapy (see 📖 p. 475). In addition to discouraging smoking, patients with FH should be offered the following advice:[1]

- Dietary advice provided by an appropriately trained healthcare professional, e.g. registered dietitian.
- A ↓ fat diet compatible with the 'Eatwell Plate' (see 📖 Chapter 2, p. 27), which includes ≤30% energy from fat and ≤10% energy from saturated fat.
- Monounsaturated and polyunsaturated fats should be used to replace saturated fat.
- Dietary cholesterol intake should be limited to ≤300 mg/day.
- Eat at least five portions of fruit and vegetables each day.
- Eat at least two portions of fish per week, including at least one oily fish (see Table 23.3). For women who are pregnant or trying to conceive, see 📖 Chapter 12, p. 217.
- Advise people who wish to take food products containing stanols and sterols that they need to be taken consistently to be effective (see 📖 Chapter 8, p. 165, and Table 8.5).
- Do not routinely advise people to take omega-3 supplements.
- Alcohol intake should be limited to ≤3–4 units per day for men and ≤2–3 units per day and binge drinking of alcohol avoided.
- Weight management advice should be offered to those who are overweight or obese (see 📖 Chapter 21, p. 411).
- For people with FH who have already had a myocardial infarction (MI), dietary advice should be in line with guidance on secondary prevention, see 📖 this chapter, p. 470.
- Advise >30 min of physical activity/day. This should be of moderate intensity on at least 5 days per week. If not able to undertake moderate intensity activity because of co-morbidities or other reasons, a person should be encouraged to exercise at their maximum safe capacity. Accumulated bouts of activity of ≥10 min are as effective as longer sessions.

Lipid-modifying drug therapy[1]

Statins should be prescribed as initial treatment in adults and in children and young people with FH. Ezetimibe is an option if statins are contraindicated or not tolerated. Treatment with bile acid sequestrants can be considered if both statins and ezetimibe are contraindicated or not tolerated but should be decided by a specialist in FH. If bile acid sequestrants are prescribed for long-term treatment, supplementation with fat-soluble vitamins (A, D, & K) and folate should be considered.

[1] NICE (2008). *Identification and management of familial hypercholesterolaemia.* Clinical guideline 71. http://guidance.nice.org.uk/CG71/Guidance.

Heart failure

Heart failure results when damage to the heart leads to reduced efficiency in pumping blood around the body with the consequent symptoms of breathlessness, fatigue and fluid retention. Medical treatment, including the prescription of ACE (angiotensin-converting enzyme) inhibitors and diuretics, may be supported by dietary management.

- *Restricting dietary sodium:* current guidelines[1] recommend avoiding excessive salt intake but recognize that evidence is required to support this. Limiting sodium intake will theoretically help maximize the effects of diuretics and thus moderate the workload on the heart by reducing the circulating volume. Low sodium diets can be very unpalatable so a compromise between avoiding an excessive salt intake while maintaining an adequate nutritional intake is required. A 'no added salt' diet should exclude high sources of dietary sodium by avoiding salt added at table (use just a pinch in cooking), stock cubes, meat and vegetable extracts, cured meat, tinned fish and meat, tinned and packet soup, salted nuts and crisps, soy sauce, monosodium glutamate (see 🕮 Chapter 28, 'Low sodium diets for ascites and oedema', p. 624).
- *Fluid restriction:* patients with severe heart failure should be advised to restrict their fluid intake to 1.5–2.0 l/day. Patients with mild or moderate heart failure are unlikely to benefit from fluid restriction.
- *Obesity:* patients with excess body weight (BMI >30 kg/m^2) should be advised to reduce slowly in order to reduce progression of heart failure, ↓ symptoms and improve well-being. In moderate to severe heart failure routine weight loss advice is inappropriate because unintentional weight and anorexia is common.
- *Nutritional adequacy*: in more advanced cases, appetite can be very poor and food intake limited by symptoms. Patients should be regularly screened for under nutrition (see 🕮 Chapter 25, 'Nutrition screening', p. 502). Changes in body weight may indicate fluid retention so a holistic assessment taking into account their food intake is required. Ensuring a nutritionally adequate intake by encouraging small, frequent, nutrient-dense meals may help maintain body weight. This may conflict with the principles of the cardioprotective diet so advice must be given holistically to take into account the likely prognosis.
- *Alcohol:* consuming alcohol is associated with a negative effect of cardiac muscle contraction; ↑ blood pressure and risk of arrhythmias. Patients should be advised to limit their intake of alcohol to 10–20 g alcohol (approximately 1–2 units, see 🕮 Chapter 9, 'Alcohol', p. 186). If patients have alcohol-related cardiomyopathy, they should be advised to abstain from alcohol completely.

[1] Dickstein, K., Cohen-Solal, A., Filippatos, G., *et al.* (2008). ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure. *Eur. J. Heart Fail.* **10,** 933–89.

Refsum's disease

Refsum's disease (RDis) is a rare autosomal recessive disorder of lipid metabolism where the presence of a defective enzyme, phytanoyl-coenzyme A hydroxylase, results in the accumulation of phytanic acid leading to neurological symptoms.

Treatment is based on restricting the dietary intake of phytanic acid from a usual intake of 50–100mg/day to <10mg/day and minimizing release of endogenous phytanic acid.

- *Rich sources of phytanic acid*: avoid in RDis, e.g. beef, lamb, meat from ruminant animals (e.g. venison), dairy products (including cows' and goats'), fish and fish oils, baked products with unknown sources of fat.
- *Foods containing little phytanic acid*: (or in bound form): acceptable in RDis, e.g. poultry, pork, fruit, vegetables, sea-food with very low fat content, e.g. crab and prawns, cereal products (unless prepared with dairy or fish oil), eggs, soya milk, vegetable oils, and margarine made exclusively from vegetable oils.
- *Nutritional adequacy of the diet:* must be checked to ensure sufficient energy and all other nutrients are provided. A low energy diet will lead to weight loss and the accompanying lipolysis will mobilize endogenous phytanic acid. If necessary, supplements should be provided during periods of intercurrent illness to ensure an adequate intake is maintained.
- *Caffeine:* high intakes should be avoided as they are associated with hepatic lipolysis and phytanic acid release.

Adherence to the diet is associated with sustained reductions in serum phytanic acid and few acute complications that are associated with untreated RDis. It is recommended[1] that patients are reviewed 6-monthly and that dietary restrictions should be followed for life.

[1] Baldwin, E.J., Gibberd, F.B., Harley, C., *et al.* (2010). The effectiveness of long-term dietary therapy in the treatment of adult Refsum disease. *J Neurol Neurosurg Psychiatry* doi:10.1136/jnnp.2008.161059.

Stroke/cerebrovascular accident

Stroke or cerebrovascular accident (CVA) is the cause of approximately 11% of deaths in the UK, the second most common cause of dementia, and the most important single cause of severe disability in people living in their own homes. Approximately 150 000 people suffer from a stroke each year in the UK and the incidence increases with ↑ age.

Causes:

- 80% cerebral infarction;
- 15% primary intracerebral or subarachnoid haemorrhage;
- 5% cause uncertain.

Nutrition has two key roles to play:

- Public health, i.e. *prevention*—a healthy, well-balanced diet can help reduce risk (Table 23.5).
- Clinical, i.e. *treatment*—an appropriate, modified diet may be required to help maintain adequate nutritional intake.

Nutritional advice in stroke prevention

Table 23.5 Prevention of CVA

Risk factor	Nutritional link
Hypertension (major risk)	Associated with obesity, ↓ physical activity alcohol, ↑ Na^+ intake, ↓ K^+ intake
Hyperhomocysteinaemia	Associated with ↓ fruit and vegetable intake
Oxidative stress	Improved by dietary antioxidants
Endothelial dysfunction	Improved by *n*–3 fatty acids

Although these risk factors are associated with ↑ stroke, there is little evidence at present from intervention studies to indicate that addressing them reduces risk. This is likely to be a consequence of partly the difficulty in undertaking long-term dietary interventions and the limited mechanistic understanding of micronutrient effects. In light of this, a cardio-protective diet may be the best recommendation. The key points relating to stroke are:

Dietary advice

- Reduce weight in obesity. A reduction of 3–9% in body weight is associated with a 3 mmHg reduction in systolic and diastolic BP. Ideally this should be accompanied by an increase in regular, low intensity activity such as walking.
- Avoid binge drinking of alcohol, i.e. ≥6 units of alcohol on one day. A J-shaped curve of ischaemic CVA against alcohol intake suggests that there may be benefits from consuming up to 1–2 units of alcohol/day. See 🕮 Chapter 9, 'Alcohol', p. 186).
- Reduce excessive salt intake. Cutting salt intake from 9–6 g/day is estimated to reduce systolic BP by 3 mmHg. This could be achieved by avoiding adding salt to food at the table, using just a pinch of salt in

cooking, and limiting processed foods including stock cubes, meat and vegetable extracts, cured meat, tinned fish and meat, tinned and packet soup, salted nuts and crisps, soy sauce, monosodium glutamate.

- Increase fruit and vegetable intake. A minimum of five portions per day, including green leafy vegetables, will help increase potassium and folate intake (to counter homocysteinaemia) and provide the antioxidants, vitamin C, carotenoids, and flavonoids. There is no evidence that supplements with B vitamins including folate are beneficial.
- There is conflicting evidence over the use of antioxidant supplements and ∴ food sources are recommended in preference. In addition to the fruit and vegetables, vitamin E can be obtained from vegetable oil including olive oil and flavonoids from tea.
- Eat oily fish once a week to provide *n*–3 fatty acids. This has an anti-thrombogenic effect but without exacerbating bleeding tendency. There is some evidence that a moderate intake from food may be better than high dose supplements.
- In people with diabetes, ensure good blood sugar control.

Changes in food intake typically associated with older people, including a reduced food intake due to bereavement, difficulty in shopping, cooking, eating, or lack of appetite, often lead to a reduced intake of fruit and vegetables and ↑ consumption of nutrient-poor, processed foods. Support may be required to counter this with an adequate, well-balanced diet.

Nutritional support following stroke

Anyone with a suspected stroke should be admitted to a specialist stroke unit. Following brain imaging, possibly thrombolysis and early mobilisation, all patients should be screened for swallowing function and malnutrition (Fig 23.2).

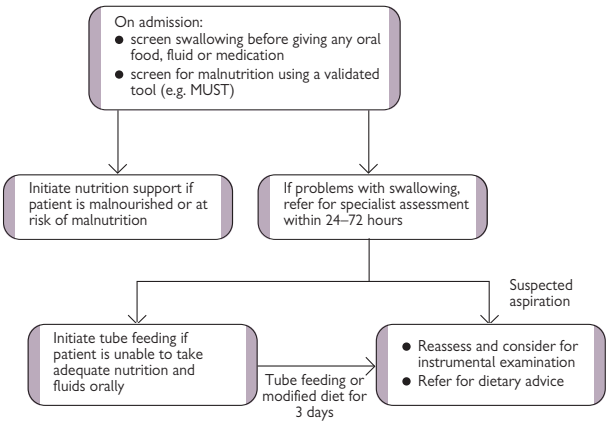

Fig. 23.2 Swallow and nutrition screening for patients admitted to hospital with stroke. From NICE (2008) Stroke. Clinical guideline 68. http://guidance.nice.org.uk/CG68/Guidance.

Dysphagia

Dysphagia (discomfort, difficulty, or pain when swallowing) is common following stroke. Swallowing has four stages:

- *Preparation:* transfer of food into the mouth, sealed with lips.
- *Oral:* chewing, mixing with saliva, bolus formation, and transfer toward pharynx.
- *Pharyngeal:* complex stage where bolus is involuntarily transferred towards oesophagus with simultaneous closure of larynx and pause in respiration.
- *Oesophageal:* transfer to stomach by peristalsis and gravity.

❗ Impaired swallow leads to high risk from aspirating food or liquid into the respiratory tract.

Texture modification

If swallowing is impaired, modifying the texture of food may facilitate safe oral intake. A speech and language therapist should assess what each patient is capable of swallowing safely using standardized descriptive categories, see Table 23.6.

Tube feeding

Patients with dysphagia who are unable to achieve their nutritional requirements orally and those who are unconscious may require tube feeding. This should commence within 24 h of admission and a gastrostomy or nasal bridle ❗ should be considered if the person cannot tolerate a nasal tube. If feeding is likely to be required for >4 weeks, a percutaneous enteroscopic gastrostomy (PEG) should be considered. Although inserting a PEG is an invasive procedure, and ethical consideration must be given to the associated risks and ongoing management issues, feeding via a PEG has a much lower risk of aspiration and is associated with better clinical outcome in some studies.

Longer term

The patient's nutritional status and ability to swallow should be regularly monitored and adjustments made to their nutritional management accordingly. Dysphagia frequently resolves in the first 6 months following a CVA although it may persist. Switching to overnight tube feeding to encourage oral intake during the day may facilitate a return to normality. Patients may continue to need assistance with meals and, most of all, time to maximize their ability to eat an adequate diet. Neurological damage after CVA varies with the following losses of function that may impact on eating:

- altered levels of consciousness in 30–40% of patients;
- difficulty in swallowing in 30%;
- motor weakness in 50–80%;
- slurred speech, 30%;
- dysphagia/aphasia in 30%;
- visual field defects in 7%.

As a result, inadequate intake and consequent undernutrition is a common problem, especially in altered states of consciousness, and becomes significantly worse as hospital stay continues and is associated with ↑ morbidity and reduced survival. Evidence from feeding studies has shown that nutritional status, length of hospital stay, and mortality can be influenced by nutrition intervention.

Table 23.6 Dysphagia diet food texture descriptors*

(B)	THIN PURÉE DYSPHAGIA DIET Food has been puréed or has thin purée texture; does not require chewing; smooth throughout with no 'bits'; moist but all fluid is within food and does not separate out; not sticky; no hard pieces, crust or skin formed during cooking/standing; no garnish; no jelly or ice cream unless advised suitable for individual. Food of this texture does not hold its shape on plate; spreads out if spilled; cannot be piped or moulded; cannot be eated with a fork; a light disposable plastic teaspoon must stand upright if placed in food with head fully covered.
(C)	THICK PURÉE DYSPHAGIA DIET Food has been puréed or has thick purée texture; does not require chewing; smooth throughout with no 'bits'; may have 'fine' textured quality providing bolus remains cohesive in mouth; moist but all fluid is within food and does not separate out; not sticky; not rubbery; no hard pieces, crust or skin formed during cooking/standing; no garnish; no jelly or ice cream unless advised suitable for individual. Food of this texture holds its shape on plate; does not spread out if spilled; can be piped or moulded; can be eaten with a fork.
(D)	PRE-MASHED DYSPHAGIA DIET Food is soft, tender, moist and needs little chewing; mashed with fork before serving; requires very thick, smooth gravy/sauce/custard but no loose fluid; not hard, tough, chewy, stringy, dry, crispy, crunchy or crumbly; no pips, seeds, husks, skin, bone, gristle; no long-shaped foods e.g. sausage; no round-shaped food e.g. grapes; no sticky food e.g. cheese; no floppy food e.g. lettuce; no juicy foods where liquid separates e.g. water melon.
(E)	FORK MASHABLE DYSPHAGIA DIET Food is soft, tender, moist and needs some chewing; it can be mashed with a fork; usually requires very thick, smooth gravy/sauce/custard but no loose fluid; not hard, tough, chewy, stringy, dry, crispy, crunchy, or crumbly; no pips, seeds, husks, skin, bone, gristle; no long-shaped foods e.g. sausage; no round-shaped food e.g. grapes; no sticky food e.g. cheese; no floppy food e.g., lettuce; no juicy foods where liquid separates e.g. water melon.

*Adaped from NPSA (2011). Dysphagia diet food texture descriptors.
http://www.bda.uk.com/publications/statements/NationalDescriptorsTextureModificationAdults.pdf
Please note that there is no texture A.

Box 23.3 Thickened fluid for dysphagia*

Stage 1: Can be drunk through a straw or from a cup is advised/ preferred. Leaves thin coat on back of spoon.
Stage 2: Cannot be drunk through a straw. Can be drunk from a cup. Leaves thick coat on back of spoon.
Stage 3: Cannot be drunk through a straw or from a cup. Needs to be taken by spoon.

*Adapted from Nutrition and Diet Resources (2010) Easier swallowing: fluid thickening guide http://www.ndr-uk.org/downloads/pdf/consistency/100505FluidThickening.pdf

Nutritional requirements

Energy and protein requirements (Tables 23.7 and 23.8) may be ↑ due to hypermetabolism, which persists for 4–8 weeks, and in frail elderly people who have pre-existing undernutrition.

Table 23.7 Estimating energy requirements*

1	Estimate basal metabolic rate using the Schofield equation[a] (see 🕮 Appendix 4, p. 772)	
	Either:	
2a	Add to BMR a stress factor: (calculated as % of BMR) for specific clinical condition:	
	Condition	**Stress factor** (%)
	Cerebral vascular accident	5
	Cerebral haemorrhage	30
	Or	
2b	Add to BMR 400–1,000 kcal/day if ↑ in body weight (lean or fat) is desired *or* subtract 400–1,000 kcal/day if ↓ in body fat is desired and patient is not metabolically stressed	
3	Add to BMR an activity factor (calculated as % of BMR)	
	Activity level	**Activity factor (%)**
	Patient in bed and immobile	10
	Patient in bed but able to move and sit up	15–20
	Patient mobile on ward	25
	Patient living in the community	Use PAL[b]

* Todorovic, V.E., *et al.* (2007). *A Pocket Guide to Clinical Nutrition*. British Dietetic Association, pp. 3.1–3.3a.

[a] Requirements should be based on actual body weight except in obesity (if BMI 30–50 kg/m^2 use 75% value; if BMI >50 kg/m^2 use 65% value).

[b] PAL – physical activity level (see 🕮 Chapter 5 'Energy balance', p. 80).

Table 23.8 Estimating protein requirements*

Protein	Nitrogen (g/kg/day)†	
	Mean	Range
Normal	0.17	0.14–0.20
Hypermetabolic (+5 to +25%)	0.20	0.17–0.25

*Requirements should be based on actual body weight except in obesity (if BMI 30–50 kg/m^2 use 75% value; if BMI >50 kg/m^2 use 65% value).

†1 g nitrogen ≡ 6.25 g protein; 1 g protein ≡ 0.16 g nitrogen.

Free radical damage may play a role in brain damage after infarction/reperfusion. This suggests that antioxidant therapy may have a role to play although current evidence is unclear and insufficient to support supplementation. Although a well-balanced, antioxidant-rich diet may have potential value, nutrient intake may be compromised in dysphagia. It is important to remember that foods of altered texture, e.g. puréed items, may contain less energy and ↓ quantities of macronutrients than un-puréed food because of the dilution effect of adding fluid to facilitate blending. In addition, the blending process breaks down plant cell walls and exposes antioxidants and other micronutrients to potential oxidation thus reducing bio-availability. There is no evidence available at present to support antioxidant supplementation post-stroke and this cannot be recommended. However, attention should be given to the adequacy of micronutrient intake from food.

Further information

NICE (2008) *Stroke*. Clinical guideline 68. http://guidance.nice.org.uk/CG68/Guidance.
Gariballa, S. (2004). *Nutrition and stroke*. Blackwell Publishing, Oxford.
British Dietetic Association (2009). *National descriptors for texture modification in adults*. BDA, Birmingham.
The Stroke Association http://www.stroke.org.uk/index.html.

Hypertension

Hypertension (HT) is defined as persistent systolic blood pressure (SBP) above 140mm Hg, diastolic blood pressure (DBP) above 90mm Hg, or when levels below this are maintained by antihypertensive medication.

It is a significant health problem in the UK with an estimated prevalence of 39% and 31%, respectively, for men and women in England. Prevalence increases with age and is higher in Black and south Asian communities.

HT is a major risk factor for cardiovascular disease (especially stroke, angina, myocardial infarction, heart failure, and left ventricular failure), renal disease, and retinopathy.

Factors contributing to HT include:

- obesity, especially if central adiposity;
- insulin resistance;
- diabetes mellitus;
- low levels of physical activity;
- psychosocial stress;
- high salt intake;
- high alcohol intake, especially if regular heavy or binge drinking;
- increasing age.

Management

Lifestyle advice should be offered as the first line of treatment. This should include the promotion of a healthy diet (see 📖 p. 470 and Box 23.3), regular exercise (~30 min/day at least 5 times per week) and smoking cessation. Those with diabetes, hyperlipidaemia or previous coronary heart disease should receive additional advice.

Dietary intervention

- Reduction of excess body weight. Losing 3–9% body weight is associated with 3 mmHg reduction in systolic blood pressure (SBP) and diastolic blood pressure (DBP).
- Limit alcohol intake to maximum of 21 or 14 units per week for men and women, respectively. Structured intervention to reduce excess intake is associated with a mean reduction of 3–4 mmHg SBP and DBP with a third of patients achieving >10 mmHg reduction in SBP.
- Limit salt intake to <6 g/day (2.4 g or 100 mmol Na^+) from current UK average intakes of ~9 g/day. This reduction does not mean consuming 6 g of 'added salt' (i.e. heaped teaspoonful)! Most people consume a substantial proportion of salt from prepared food and this should be included with the 6 g limit. This could be achieved by not adding any salt to food at the table, using just a pinch of salt in cooking, and limiting processed foods including stock cubes, meat and vegetable extracts, cured meat, tinned fish and meat, tinned and packet soup, salted nuts and crisps, soy sauce, monosodium glutamate. Some people use potassium-containing salt substitutes but others find them metallic-tasting and unpleasant. Experimenting with herbs, spices, lemon juice and garlic etc can help promote flavour and enjoyment in eating. There may be additional health benefits anticipated from lower

salt intakes ~3g/day. Although this may be an ideal goal to aim at, for many people it will require major and possibly unacceptable changes to their usual eating habits.
- Limit caffeine consumption to <5 cups of tea/coffee per day.
- Eat more fruit and vegetables. Five portions per day should be the minimum target.
- Calcium, potassium, and magnesium supplements are not recommended.
- Overall, a cardioprotective diet is appropriate for individuals with HT (see 📖 this chapter 'Cardioprotective diet', p. 470).

Box 23.3 Dietary Approaches to Stop Hypertension

The principles described above are incorporated into the dietary approaches to stop hypertension (DASH) diet (↓ salt, ↓ fat, ↑ fruit and vegetables), which has been developed and evaluated in the USA. Adherence is associated with improved health outcomes including d hypertension.

🖱 http://dashdiet.org

Further information

Bibbins-Domingo, K., Chertow, G.M., Coxson, P.G., *et al.* (2010). Projected effect of dietary salt reductions on future cardiovascular disease. *N. Engl. J. Med.* **362**, 509–99.

Strazzullo, P., D'Elia, L., Kandala, N.B., *et al.* (2009). Salt intake, stroke, and cardiovascular disease: meta-analysis of prospective studies. *Br. Med. J.* **24**, 339.

🖱 British Hypertension Society http://www.bhsoc.org/

🖱 Blood Pressure Association http://www.bpassoc.org.uk/Home

🖱 Consensus Action on Salt and Health http://www.actiononsalt.org.uk/

Peripheral arterial disease

Peripheral arterial disease (PAD) is caused by atheromatous plaque leading to narrowing of peripheral blood vessels, most commonly in the legs. This results in poor blood supply and pain on exertion (claudication) and may lead to ulceration, limb loss and ↑ risk of death from myocardial infarction and stroke. Poor quality of life and depression are associated.

- *Prevalence:* although reported as poorly diagnosed, it is estimated that ~12% of the adult population may be affected and ~20% of those over 70 years.
- *Risk factors:* include ↑ age, smoking, diabetes mellitus, hyperlipidaemia and hypertension.

Nutrition

- *Prevention:* epidemiology[1] indicates that ↑ intake of antioxidants (vitamins A, C and E), vitamin B_6, folate, fibre and omega-3 fatty acids have a protective effect independent of other cardiovascular risk factors.
- *Folate:* patients with PAD have significantly ↑ total plasma homocysteine levels compared with controls in meta-analysis[2] of 14 studies. This is associated with ↓ folate intake but, at present, there is limited evidence from intervention studies[2, 3] that folate supplementation has any clinical benefit.
- *Vitamin D:* low serum vitamin D levels are associated with ↑ prevalence of PAD[4] and may explain differences in risk between ethnic groups. To date, no intervention studies have examined the effects of vitamin D supplementation.
- *Cardioprotective diet:* in view of ↑ PAD risk associated with hyperlipidaemia and hypertension, it is reasonable to recommend a cardioprotective diet (see 🕮 this chapter, p. 470) which is associated with ↑ endothelial function. No studies to date have examined the effects of a holistic cardioprotective dietary approach, rather than the effects of single elements, for managing PAD.

[1] Lane, J.S., Magno, C.P., Lane, K.T., *et al.* (2008). Nutrition impacts the prevalence of peripheral arterial disease in the United States. *J. Vasc. Surg.* **48,** 897–904.
[2] Khandanpour, N.,, Loke, Y.K., Meyer, F.J., *et al.* (2009). Homocysteine and peripheral arterial disease: Systematic review and meta-analysis. *Eur. J. Vasc. Endovasc. Surg.* **38,** 316–22.
[3] Khandanpour, N., Armon, M.P., Jennings, B., *et al.* (2009). Randomized controlled trial of folate supplementation in patients with peripheral vascular disease. *Br. J. Surg.* **96,** 990–8.
[4] Melamed, M.L., Muntner, P., Michos, E.D., *et al.* (2008). Serum 25-hydroxyvitamin D levels and the prevalence of peripheral arterial disease. *Arterioscler. Thromb. Vasc. Biol.* **28,** 1179–85.

Chapter 24

Cancer and leukaemia

Cancer: introduction and dietary guidelines to minimize risk

Cancer is a major cause of morbidity with almost 300,000 new cases diagnosed in the UK each year and one in four deaths cancer-related. Nutrition plays an important role in both the aetiology and treatment of many different cancers.

Incidence

See Tables 24.1 and 24.2

Table 24.1 Most common cancers in UK (excluding non-melanoma skin; 2007 data from Office for National Statistics)

Men		Women	
Type	**Number (%)**	**Type**	**Number (%)**
Prostate	30 201 (25)	Breast	38 048 (31)
Lung	17 961 (15)	Colorectal	13 876 (11)
Colorectal	17 851 (14)	Lung	13 561 (11)
Total	123 131 (100)	Total	122 196 (100)

Table 24.2 Most common cancers in the world (excluding non-melanoma skin). (2002 data from Cancer Mondial. Available at: http://www-dep.iarc.fr/)

Men		Women	
Type	**Number (%)**	**Type**	**Number (%)**
Lung	965 000 (17)	Breast	1 151 000 (23)
Prostate	679 000 (12)	Cervix	493 000 (10)
Stomach	603 000 (10)	Large bowel	473 000 (9)
Large bowel	550 000 (9)	Lung	387 000 (8)
Total	5 802 000 (100)	Total	5 061 000 (100)

Aetiology

It is estimated that dietary factors are responsible for ~25% of cancer deaths in the UK. Only deaths related to use of tobacco are higher (~30%). The strength of evidence linking specific elements of the diet to different cancers varies (Table 24.3).

The link between food and cancer causation is very complex because the variation in what people eat and the interrelationships between different foods, nutrients, lifestyle, and environmental factors make unraveling the connections very difficult. However, a number of currently ongoing, large epidemiological studies may contribute more information and although some links have only been tentatively established at present (see Table 24.3) there is sufficient information to make dietary recommendations.

Dietary guidelines to minimize cancer risk

The UK 'Eatwell Plate' guideline (see 📖 Chapter 2, p. 27) are compatible with those made by the World Cancer Research Fund.[1]

Key recommendations for all population groups include the following:

- Choose a diet high in wholegrains, fruit and vegetables, and low in fat and sugar.
- Increase intake of fish and white meat, reducing red and processed meats.
- Maintain a healthy weight and be physically active.
- Drink alcohol in moderation, if at all.
- Select foods low in salt.
- Vitamin and mineral supplements should not be taken to protect against cancer.

Table 24.3 Strength of evidence linking specific elements of the diet to different cancers

Level of evidence	Decrease risk		Increase risk	
	Dietary element	**Type of cancer**	**Dietary element**	**Type of cancer**
Convincing			Obesity	Oesophagus, bowel, breast, endometrium, kidney
Probable	Fruit and vegetables	Upper GI tract, stomach, bowel	Very hot drinks. Salt and salt-preserved foods	Upper GI tract, Stomach
			Preserved and red meat	Bowel
Insufficient	Fibre, soya, fish, *n*–3 fats, vitamins, minerals, non-nutrient plant constituents	—	Animal fats, heterocyclic amines, polycyclic aromatic hydrocarbons, nitrosamines, acrylamide	—

Key, T.J., Schatzkin, A., Willett, W.C., *et al.* (2004). Diet, nutrition and the prevention of cancer. *Publ. Health Nutr.* **7**, 187–200.

Further information: The European Prospective Investigation into Cancer and Nutrition (EPIC) Project. Available at: http://epic.iarc.fr/index.php.

[1] Reproduced with kind permission from the World Cancer Research Fund website (℞ www.wcrf.org).

Effects of cancer on nutritional status

Whilst the guidelines in 🕮 'Cancer: introduction and dietary guidelines to minimize risk', this chapter, p. 488, are appropriate for healthy adults, those who have been diagnosed with cancer may require different and more specific dietary advice because of the negative nutritional effects of cancer or treatment. Factors that may lead to a deterioration in nutritional status are listed in Box 24.1.

Box 24.1 Factors that may lead to a deterioration in nutritional status

- Reduced intake due to:
  - physical symptoms e.g. anorexia, nausea, vomiting, diarrhoea, constipation, taste changes, mucositis, dry mouth, fatigue and pain
  - psychological symptoms e.g. anxiety and depression
  - psychosocial issues e.g. reduced access to food due to poor mobility or financial concerns
  - nil by mouth (investigations and treatment).
- Reduced digestion/absorption:
  - ± specific to site of cancer
- Altered metabolism (see 🕮 'Cancer cachexia', this chapter, p. 492):
  - ↑ protein catabolism
  - ↓ muscle protein synthesis
  - ↑ gluconeogenesis ± ↑ insulin resistance
  - ↑ lipolysis and ↑ fatty acid and glycerol turnover
  - ↑ metabolic rate 2° to tumour growth
  - secondary infections
  - loss or gain of weight
- Increased losses:
  - vomiting
  - diarrhoea
  - fistulae
- Effects of treatment:
  - surgery (see 🕮 Chapter 25 'Surgery', p. 552)
  - chemotherapy (see 🕮 this Chapter, 'Chemotherapy', p. 490);
  - radiotherapy (see 🕮 this Chapter, 'Radiotherapy', p. 491).

Chemotherapy

The nutrition-related side-effects associated with chemotherapy depend on the regime and dose prescribed. Chemotherapy regimes often comprise cycles of cytotoxic drugs repeated at regular intervals over several months. Fatigue, anorexia, and an increased risk of infection are common in people receiving chemotherapy. Eating well during the 'good times' between doses may help to maintain nutritional status, even if intake falls during and immediately after the infusion period.

Examples of common side-effects related to specific cytotoxic drugs

- *Capecitabine:* nausea, vomiting, mucositis and diarrhoea.
- *Cisplatin:* severe nausea and vomiting.
- *Doxorubicin:* severe mucositis (dose-dependent), nausea and vomiting.
- *5-fluorouracil (5FU):* severe mucositis plus taste alterations, nausea, vomiting, and diarrhoea.
- *Vinblastine:* mild to moderate nausea, vomiting, and mucositis.

For further information see Cancer Research UK. Available at: http://www.cancerhelp.org.uk/about-cancer/treatment/chemotherapy/index.htm.

Radiotherapy

The nutrition-related side-effects associated with radiotherapy depend on site of the cancer and dose prescribed.

- General effects can include anorexia, nausea, tiredness, and altered mood. If the course of radiation includes a prolonged programme of regular sessions requiring daily hospital visits, assistance with the practicalities of food shopping and preparation may be required.
- Local effects arise from the impact of the radiation on healthy tissue near the cancer site and can cause soreness. This may have little nutritional impact if it is a discrete, non-systemic area, e.g. limb. However, severe problems can arise with radiotherapy to:
  - *Head and neck:* includes cancer of mouth, tongue, salivary glands, nasopharynx, tonsil, larynx, neck, and face. Damage to the mucosal cells breaches the surface potentially leading to infection, inflammation, and severe pain. A dry mouth, taste changes, loss of taste, and difficulty swallowing may also occur and the combined effects often severely limit food intake leading to a rapid decline in nutritional status. Regular nutritional assessment is required; the suggestions in the Box 24.2 may help. Artificial nutrition support is often indicated in these patients. Gastrostomy tubes should ideally be placed prior to treatment commencing.
  - *Abdominal/pelvic area*: includes cancer of the cervix, colon, pancreas, prostate, and rectum. Irradiation damages the gastrointestinal (GI) mucosa leading to a reduction in epithelial surface and impaired absorption of nutrients. Diarrhoea, bloating, and cramping pains may result and, in severe cases, ulceration, strictures, and perforation. Reducing dietary fibre, fat and lactose may help some patients but should ***not*** be advised routinely to all. Individual advice from a dietitian will help identify the dietary components that exacerbate symptoms and facilitate the construction of a nutritional adequate intake compatible with avoiding these.
  - *Lungs:* irradiation to some parts of the lungs can result in a sore throat and swallowing difficulties. Patients who are receiving continuous hyperfractionated accelerated radiotherapy (CHART) are at greater risk, particularly as symptoms may not occur until after the treatment has been completed and the patient has been discharged home.

Cancer cachexia

A high proportion of patients with cancer develop cachexia. It is a complex multifactorial metabolic syndrome. Cytokines, e.g. interleukin-1, interleukin-6 and TNF-α, are released by the body in response to the tumour resulting in systemic inflammation. Metabolic changes, including an increase in protein and fat breakdown, decrease in protein synthesis and altered resting energy expenditure, result in weight loss. In addition, common symptoms experienced by those with cachexia such as anorexia, fatigue, and low mood all contribute to a reduced oral intake. Cachexia plays an important role in the development of malnutrition in people with cancer.

Mucositis

Prevalence: ~40% of all cancer patients; ~75% of those receiving high-dose chemotherapy, e.g. prior to bone marrow transplantation; ~100% in patients receiving radiotherapy for head and neck cancer.

Symptoms: occur 7–14 days after treatment starts. Can affect any mucus membrane, but the most common site is in the mouth. This starts with soreness of the mouth lining, which may develop into ulcers affecting tongue and lips, and leading to painful eating and, in severe cases, preclude swallowing even liquids. Mucositis in the GI tract is usually associated with diarrhoea and may include blood in stools, bloating, and abdominal pain.

Consequences: impaired nutritional intake will contribute to cachexia whilst damage to mucosal integrity allows bacterial access which can lead to sepsis, especially when immunity is impaired.

Management: MOUTH—good oral hygiene should include brushing teeth with soft brush twice daily, flossing teeth daily, and using mouth wash four times daily; avoid tobacco and alcohol and irritating foods (see Box 24.2); ensure adequate fluid intake (>1.2 L/day); sucking ice cubes may alleviate pain. In severe cases, medication (e.g. palfermin), laser treatment of ulcers and nutrients delivered by an alternative route may be required; GI TRACT—diarrhoea will require additional fluid intake either orally or, in severe cases, intravenously. Loperamide and octreotide may also be prescribed.

Nutritional management in cancer

Nutritional management can play an important role during cancer treatment (Box 24.2). The goal of treatment will be:

- *Curative:* to eradicate the disease.
- *Palliative:* to relieve symptoms and maximize quality of life (see 📖 Chapter 35, 'Palliative care', p. 715).

Nutritional assessment

The effect of cancer on nutritional status varies depending on the site of the tumour, the stage of the disease, treatment, and potential complicating factors. Nutritional screening should be implemented locally to identify those who are in need of dietetic advice and at risk of malnutrition. Each patient should then be assessed, including an evaluation of:

- anthropometrics (see 📖 Chapter 4, 'Nutrition assessment', p. 33);
- biochemical parameters;
- clinical diagnosis and condition (including symptoms);
- nutritional intake and dietary preferences;
- energy and nutrient requirements (see 📖 Chapter 25, 'Estimating requirements in disease states', p. 540, and Appendix 6, p. 777);
- treatment received and proposed.

Nutrition support

The first step of nutritional management should be the discussion, and where possible the correction, of factors contributing to a reduced intake. Nutritional support should be given by ensuring that the patient has access to suitable food and fluids when they are able to eat orally. Intake should be supplemented using ordinary food, proprietary products or a combination of both (see 📖 Chapter 25, 'Treatment of undernutrition', p. 512). If an adequate oral intake cannot be achieved and it is clinically appropriate (see 📖 Chapter 35, 'Palliative care', p. 715), artificial nutrition support preferably via the gut (see 📖 Chapter 25, 'Enteral feeding: introduction', p. 516) or directly into the blood (see 📖 Chapter 25, 'Parenteral nutrition', p. 536) should be undertaken.

There is strong evidence to suggest that prevention of weight loss can reduce treatment complications and the length of time spent in hospital, as well as improving the patient's quality of life. There is no conclusive evidence to support the theory that restricting the nutritional intake of patients with cancer reduces tumour growth.

Box 24.2 Suggestions to alleviate nutrition-related side-effects

- *Anorexia:* try to maximize intake by taking high-energy/high-protein foods and fluids; gentle exercise/fresh air before meals may promote appetite; smaller more frequent meals may help.
- *Taste changes:* try food chilled; eat more of the foods that still taste good; if red meat doesn't taste right, try poultry, fish, eggs instead; stronger flavours like ginger, lemon or spices may help; maintain regular mouth care.
- *Dry mouth:* sip cool drinks; try using a drinking straw; suck ice chips; sharp tastes like grapefruit or lemon may stimulate saliva; serve meals with sauce or gravy.
- *Nausea and vomiting:* avoid off-putting smells; plain foods in small quantities may be better tolerated; avoid lying down after eating (a gentle walk may help); sip drinks throughout day, but wait for 15 min after eating before taking more fluid; try ginger flavours, mints, and plain biscuits.
- *Mucositis (sore mouth ± oesophagus):* try soft, smooth foods with plenty of sauce; avoid spicy and salty foods and sharp, citrus tastes; chilled or warm foods may be less painful than hot; coarse/crumbly food like toast, crackers, and pastry may be better avoided.
- *Diarrhoea:* avoid irritating foods that exacerbate, e.g. pulses, onions, strong spices; consider reducing fibre-rich foods (including whole grains, fruit and vegetable intake if intake is already high); ensure fluid intake is adequate and try and keep eating even if small quantities of smooth foods; reducing fat intake and milk products is often suggested: this may have a negative effect on nutrient intake so individual advice from a dietitian is required.
- *Constipation:* this may arise 2° to cancer, treatment or analgesia, or simply from a poor intake and inactivity; eating more and gentle exercise may help; increase fluid intake ~2 L/day; gradually increasing intake of fruit, vegetables, and wholegrain cereals may help if tolerated; prunes and prune juice; a little hot water on waking may stimulate the bowel.
- *Fatigue:* ignore the clock and eat when feel more awake; a variety of well-balanced snacks can be as nutritious as a main meal; ask for help with food preparation or use ready meals or home-delivered food.

Medication to assist

- *Artificial saliva:* may alleviate a dry mouth. Available as a spray, liquid, gel, or lozenges and needs to be used frequently and before eating. Sprays containing mucin may be most effective but as this is derived from pigs, may not be acceptable to some.
- *Anti-nausea drugs:* includes metoclopramide, prochlorperazine, and domperidone. Available as tablets, capsules, and intravenous and intramuscular injections. In severe cases, administration via syringe driver may help.
- *Anti-diarrhoea drugs:* include codeine phosphate, loperamide and co-phenotrope.

Other dietary approaches to cancer treatment

There is a considerable amount of information about food and cancer available to the general public and some includes dietary advice that is based on limited or questionable scientific evidence (Box 24.3). Whilst some is compatible with what is generally regarded as a healthy, well-balanced diet, other advice is not. These include regimes recommending intakes that are nutritionally inadequate and/or unpalatable or advocating complex eating patterns that require either the sourcing of unusual or hard-to-buy items or complicated preparation regimes. Focusing on food can be a valuable and positive activity for a patient and his/her carers, and alternative approaches should not automatically be regarded as 'bad'. However, it is essential that unproven benefits are balanced against any potential nutritional inadequacy and negative emotional consequences, e.g. denial of favourite foods, guilt at non-compliance, and despair if cure does not occur.

Patients who are interested in following complementary or alternative dietary regimes should be encouraged to discuss this with their doctor and dietitian so that the potential benefits and/or detrimental effects can be evaluated with respect to their individual needs. Consideration can be given to combining such approaches with conventional nutritional support and healthcare professionals should help provide patients with appropriate information so that they can make an informed decision.

Box 24.3 Examples of popular dietary approaches to cancer

Bristol approach to healthy eating

Developed from the old 'Bristol Diet' at Penny Brohn Cancer Care. Promotes healthy eating and recommends a diet that is based primarily on plant foods including vegetables, fruits, whole grains, pulses, nuts, seeds, herbs, and spices. A smaller quantity of animal products is recommended alongside the plant foods whilst refined grains, 'damaged fats', processed meat and alcohol are discouraged. Organic food, maintaining a healthy weight and enjoying meals are encouraged.

Comment: this advice includes key aspects of World Cancer Research Fund guidelines and can be nutritionally adequate for people who are eating well. Those with a poor appetite may not achieve an adequate energy or protein intake. The scientific evidence for discouraging dairy intake is limited and not universally accepted.

Gerson therapy

Based on stimulating the immune system to rid the body of cancer-related toxins. The diet is a very strict low salt one that includes large quantities of organic fruit and vegetables taken in the form of juice. Associated with other treatments known as 'metabolic therapy'.

Comment: the diet is expensive and potentially nutritionally inadequate. Coffee enemas have been associated with infection and inflammation of the bowel. Recent review by the USA National Cancer Institute reported that no good quality evidence has been published to support its use.[1]

Macrobiotics

A philosophical approach to life that includes balancing yin and yang elements. The dietary element is based on predominantly vegetarian, high carbohydrate, low fat food with regular consumption of soya and sea vegetables.

Comment: the high phytoestrogen content of the diet may offer some benefits but there is no firm scientific evidence to confirm this. A low energy and protein density is a concern in patients with a poor appetite.

Plant programme

Advises the elimination of all dietary dairy produce, reducing meat intake and replacing it with soya-based foods, pulses and cereals because of a perceived causal relationship with cancer, particularly with those that are hormone-mediated, e.g. breast and prostate.

Comment: published epidemiological studies do not support the proposed link. Milk and other dairy products provide useful nutrients in a palatable form and, although avoidance may not comprise intake in relatively well patients, it may be detrimental in those who are eating poorly.

[1] http://www.cancer.gov/cancertopics/pdq/cam/gerson/HealthProfessional/page2.

Leukaemia

Leukaemia can be acute (e.g. acute lymphoblastic or acute myeloid) or chronic (e.g. chronic myeloid or chronic lymphocytic). The type will determine the management and prognosis. Patients with leukaemia tend to have a better nutritional status at diagnosis than those with solid tumours. However, this can deteriorate rapidly in response to treatment including chemo- and radiotherapy (see 📖 this Chapter, 'Chemotherapy', p. 490 and 'Radiotherapy', p. 491). Nutritional support is often required to optimize well-being and nutritional status.

Bone marrow transplantation

Bone marrow transplantation (BMT) (or stem cell transplantation) is used to treat patients with leukaemia, lymphoma, and other haematological disorders including aplastic anaemia, some solid tumours, and some inherited metabolic disorders. Grafts may be autologous (i.e. from the patient themselves, stem cells collected whilst in remission) or allogenic (i.e. from a donor). Although most patients embarking on this course of treatment are relatively well-nourished, there are nutritional implications. Research suggests that energy requirements increase to 130–150% of predicted basal requirements and that protein needs are met by 1.4–1.5 g/kg/day.

The side effects of BMT treatment, which can impact on nutritional status include:

- Mucositis of gastrointestinal tract usually occurs within 7–14 days of high dose chemo- or radiotherapy before BMT and continues for 2–3 weeks afterwards. Leads to ↓ oral intake due to nausea, vomiting, pain of eating, and malabsorption and is worse in donor grafts where a greater degree of immunosuppression is required than for autologous grafts. Feeding via the gut, either orally or via a tube, should be attempted, but if this is unsuccessful, parenteral nutrition should be instigated. There is insufficient evidence to confirm that either enteral or parenteral nutrition is better in BMT patients.
- Immunosuppression is a necessary prerequisite for successful grafting of new bone marrow tissue. Following chemotherapy the neutrophil (white blood cell) count drops. The unwanted side-effect is ↑ susceptibility to infection. Patients are nursed in isolation and protected from potential sources of pathogens. In some units this includes the provision of specific food safety advice during neutropenia (see Table 24.4).
- Graft vs. host disease (GVHD) arises between 10 and 20 days after BMT from the response of the graft tissue to the leukaemia (a positive outcome) and host cells (a negative complication). This may affect different organs including the intestine which can lead to severe abdominal pain and malabsorption. Dietary manipulation may be required to maximize absorptive function in patients who are able to eat. Parenteral nutrition may be required in severe cases.

Studies have shown that the immunomodulating effects of omega-3 fatty acids may have benefits after BMT by reducing GVHD and the potentially

fatal complication, veno-occlusive disease.[1,2]. These findings have not been replicated by other researchers and further studies are needed.

NB. Intravenous lipid, once controversial, has been shown to be safe in parenteral regimes, is a useful source of energy and essential fatty acids, and is not associated with an increase in bacterial or fungal infections. Parenteral glutamine supplementation has also been associated with a shorter hospital stay and a reduction in +ve blood cultures in BMT patients.

Food safety advice during neutropenia

Ideally, patients having a BMT should eat a well-balanced and nourishing diet provided by food with a minimum risk of bacterial or fungal contamination. Sterile diets that were implemented 25 years ago are no longer used because they compromise nutrient intake. Although practice varies between centers, all patients at risk of neutropenia should be advised on general food safety.[3] This should include advice on:

- Shopping.
- Storage.
- Food preparation.
- Cooking.
- Eating out.

For those patients with neutropenia, their neutrophil count should determine the level of additional dietary restrictions (Table 24.4). Once their neutrophil count increases, patients should continue to follow the general food safety advice for at least 6 months after discharge from hospital.

[1] Takatsuka, H., Takemoto, Y., Iwata, N., *et al.* (2001). Oral eicosapentaenoic acid for complications of bone marrow transplantation. *Bone Marrow Transplant.* **28,** 769–74.

[2] Takatsuka, H., Takemoto, Y., Yamada, S., *et al.* 9(2002). Oral eicosapentaenoic acid for acute colonic graft-versus-host disease after bone marrow transplantation. *Drugs Exp. Clin. Res.* **28,** 121–5.

[3] For further information on food hygiene, see resources available from the Food Standards Agency. Available at: http://www.food.gov.uk/safereating/hyg/.

Table 24.4 Specific food safety advice for neutropenia

Neutrophil count of 0.5 – 2.0 x 10^9/L	
Not allowed	**Allowed**
Soft ripened and blue veined cheese, e.g. Brie, goat's cheese, paneer and Stilton	Vacuum packed pasteurized hard cheese, e.g. Cheddar and Edam Processed cheese, e.g. cheese spread
Raw or lightly cooked shellfish	Well cooked shellfish
Raw or undercooked meat, poultry or fish	Well cooked meat, poultry or fish Vacuum-packed cold meats such as turkey or ham; tinned meat and fish
Raw or undercooked eggs including foods containing them, e.g. homemade mayonnaise or ice cream	Hard boiled eggs, shop-bought ice cream and mayonnaise
Prebiotics, live or bioproducts	Pasteurized yoghurts
Paté	Pasteurized paté and paste in tins or jars that do not need to be refrigerated
All unpasteurized dairy products	Any pasteurized and UHT milk and cheese products
Neutrophil count <0.5 x 10^9/L	
In addition to the advice above:	
Raw unpeeled fruit, vegetables and salad items; raw dried fruit; damaged or over-ripe fruit or vegetables; unpasteurized or freshly squeezed fruit or vegetable juice or smoothies	Good quality fruit and vegetables that are well cooked or peeled; UHT or long-life fruit juices; pasteurized smoothies; tinned fruit; cooked dried fruit
Uncooked herbs, spices and pepper	Cooked herbs, spices and pepper
Non-drinking water, bottled mineral or spring water, water from wells, water from coolers or water fountains	Freshly run tap water, filtered, sterilized or carbonated water
Unpasteurized or 'farm fresh' honey and honeycomb	Pasteurized or heat-treated honey
Unnecessarily large packets; food items from pick and mix; universal jars; deli counter foods	Ideally packets should be for personal use only

Adapted from *Dietary Advice for Patients with Neutropenia* produced by the London Haematology Dietitians Group. Reproduced with permission.

Chapter 25

Nutrition support

Nutrition screening

This should be routinely undertaken to identify individuals who are at risk from under- or overnutrition, and should be carried out by an appropriately trained and skilled person, but not necessarily a nutrition specialist. It differs from nutritional assessment, which is undertaken by a nutrition-trained healthcare professional, usually a registered dietitian, and which gives a more detailed nutritional profile of an individual.

Who and when to screen (Box 25.1)

- *All hospital inpatients:* on admission and repeated weekly or as per protocol.
- *All hospital outpatients:* on their first clinic visit and repeated when there is clinical concern.
- *All people in care homes:* on admission and repeated when there is clinical concern.
- *All people on GP lists:* on initial registration and repeated when there is clinical concern.

Hospital departments who see groups of patients who are low risk may opt out of screening following an explicit decision made in conjunction with nutrition experts.

Box 25.1 What clinical concern would trigger repeat screening?

- Unintentional weight loss
- Fragile skin
- Poor wound healing
- Wasted muscles
- Apathy with ↓ intake
- Prolonged intercurrent illness
- Poor appetite
- Altered taste sensation
- Impaired swallowing
- Altered bowel habit
- Loose fitting clothes.

How to screen

Many different nutrition screening tools are available. The tool selected for use must:

- include the essential variables (see Box 25.2);
- be appropriate for the patient population;
- have validity confirmed by peer reviewed publication;
- have suitable cut-off points to maximize sensitivity and specificity (minimizing false positive and false negative results);
- provide outcomes that can be acted on when appropriate (link to care plans);
- reflect local needs and overcome resistance to implementation;
- be associated with a staff training programme.

Box 25.2 All screening must include evaluation of

- Body mass index (BMI) see 🕮 Appendix 2, p. 756
- Percentage unintentional weight loss
- Length of time that nutritional intake unintentionally reduced
- Likelihood of future impaired nutrient intake

See BMI ready reckoner for adults inside front cover

🖰 http://www.nice.org.uk/nicemedia/live/10978/29979/29979.pdf

Malnutrition universal screening tool

The malnutrition universal screening tool (MUST), developed by the multidisciplinary British Association for Parenteral and Enteral Nutrition (BAPEN), is considered the most scientifically robust, practical, and versatile nutrition screening tool for adults (Box 25.3 and Box 25.4). It has been designed to detect undernutrition (malnutrition) and over-nutrition (overweight/obesity).

Box 25.3 Use of Malnutrition Universal Screening Tool (MUST)

In different care settings including
- Hospital inpatients and outpatients
- Care homes
- GP surgeries and health centres
- Community

With different groups of adult patients, including but not exclusively
- Elderly
- Surgical
- Medical
- Orthopaedic
- Those requiring intensive care
- Mental health care
- Pregnancy and lactation (with adaptation)

By different healthcare professionals
- Nurses
- Doctors
- Dietitians
- Health-care assistants
- Students.

Box 25.4 The five MUST steps

1 Calculate BMI from weight and height (see 📖 Chapter 4, 'Anthropometry', p. 50). If height cannot be measured, see 📖 p. 506.
- BMI >20 = 0 (>30 = obese)
- 18.5–0 = 1
- <18.5 = 2

2 Determine unplanned weight loss (%) in past 3–6 months.
- <5% = 0
- 5–10% = 1
- >10% = 2

3 Consider the effect of acute disease.
- If patient is acutely ill and there has been or is likely to be no nutritional intake for >5 days, score 2

4 Add scores from 1, 2, and 3 together to give overall risk of malnutrition. Total score
- 0 indicates low risk
- 1 indicates medium risk
- ≥2 indicates high risk

5 Initiate appropriate nutritional management.
Using local management guidelines (Table 25.1), prepare appropriate care plan.

More information is available at 🖱 www.bapen.org.uk

Table 25.1 Malnutrition universal screening tool nutritional management guidelines

Low risk (score 0)	***Routine clinical care***
	Repeat screening (hospital, weekly; care home, monthly; community, annually for special groups, e.g. those >75 years).
Medium risk (score 1)	***Observe***
	Document dietary intake for 3 days if subject in hospital or care home. If improved or adequate intake, little clinical concern; if no improvement, clinical concern: follow local policy.
	Repeat screening (hospital, weekly; care home, at least monthly; community, at least every 2–3 months).
High risk (score ≥2)	***Treat***[1]
	Refer to dietitian, nutritional support team or implement local policy.
	Improve and increase overall nutritional intake.
	Monitor and review care plan (hospital, weekly; care home, monthly; community, monthly).
All risk categories	Treat underlying condition and provide help and advice on food choices, eating and drinking when necessary.
	Record malnutrition risk category.
	Record need for special diets and follow local policy.

[1] Unless detrimental or no benefit is expected from nutritional support e.g. imminent death

If height cannot be measured

Where height cannot be measured, use recently documented or reported height if it appears realistic. If this is not possible, a surrogate measure can be used, e.g. ulnar length, knee height, or demi-span. Ulnar length is the easiest to obtain in bed-bound patients and be measured as follows:

- The forearm is placed diagonally across the chest with fingers pointing towards the shoulder and palm inwards.
- The distance is measured between the central and most prominent part of the styloid process (bony knobble on outer wrist, little finger side) and the centre tip of the olecranon process (elbow). See Fig. 25.1.
- Estimated height ($_{e}$Ht) is calculated:
  - men <65 years: $_{e}$Ht (cm) = 79.2 + 3.60 x ulnar length (cm);
  - men ≥65 years: $_{e}$Ht (cm) = 86.3 + 3.15 x ulnar length (cm);
  - women <65 years: $_{e}$Ht (cm) = 95.6 + 2.77 x ulnar length (cm);
  - women ≥65 years: $_{e}$Ht (cm) = 80.4 + 3.25 x ulnar length (cm);
- Note that the calculated values may be inaccurate and particularly in Asian women, may produce an overestimate of height.

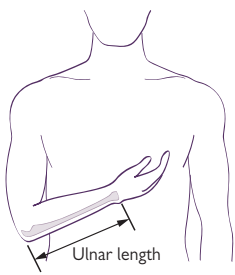

Fig. 25.1 Measurement of ulnar length.

Further information

MUST charts, guidelines, and explanatory booklet can be downloaded from http://www.bapen.org.uk/must_tool.html.

Undernutrition

Undernutrition (often referred to as malnutrition) arises as a consequence of an inadequate intake of energy and macronutrients. In some individuals it may also be associated with frank or subclinical micronutrient deficiencies.

Classification

There is no single, universally accepted definition or classification of undernutrition although a pattern of less than optimum body weight or loss of body weight is the main feature.

Body mass index (BMI) Cut-off values between 18.5 and 20.0kg/m^2 are most often used to identify risk of undernutrition in adults (Table 25.2). However, the use of BMI has its limitations. It cannot be used in children where height may be stunted as a result of poor nutrition, in the very elderly where a true height may be difficult to measure, or where unusual body morphology invalidates the ratio of weight to height.

$$\text{BMI} = \text{weight (kg)}/\text{height}^2\ (\text{m}^2)$$

See 🕮 Chapter 4, 'Anthropometry', p. 50, and inside front cover.

Mid–upper arm circumference (MUAC) Can be used if BMI cannot be calculated due to the absence of an accurate height measurement or because true weight is obscured by fluid retention (Table 25.3). (See 🕮 Chapter 4, 'Anthropometry', p. 50).

Standard deviation score (*Z*-score) Calculated from reference population data and used to determine risk of undernutrition in children (Table 25.4). No values for height are required and it is independent of age making it useful in field situations.

$$\text{Z-score} = (\text{patient's weight} - \text{median weight for population})/\text{SD value for population}$$

See 🕮 Chapter 4, 'Anthropometry', p. 50.

Malnourished patients are not always thin. They may be overweight or obese, but have suffered recent, unplanned weight loss.

Classifying undernutrition is concerned with establishing risk. None of the methods described above are foolproof but they do provide simple and reproducible means of undertaking this. The consequences of failing to identify and treat undernutrition are potentially serious and ∴ caution should be used when interpreting results. (For routine nutrition screening, see 🕮 this Chapter, 'Malnutrition universal screening tool', p. 504.)

Prevalence of malnutrition

The prevalence of undernutrition varies with the population, age group, presence and severity of disease, health, care setting, and the method used to identify undernutrition. Values cited frequently include:

- 10% of individuals with cancer or chronic disease living in the community;
- up to 50% of individuals living in care homes;
- 40–70% of patients admitted to hospital.

These figures show that undernutrition is not a rare event and so health-care staff working in all settings should be aware of this and the need to instigate and implement screening, prevention, and treatment policies.

Table 25.2 Categories of BMI for identifying undernutrition in adults

WHO classification of BMI (kg/m^2)	Interpretation
<18.50 (underweight)	Chronic undernutrition probable
18.50–24.99 (healthy/normal weight)	Chronic undernutrition possible if BMI <20
≥25.00 (overweight)	Chronic undernutrition unlikely (low risk)

Table 25.3 Classification of undernutrition in adults using MUAC*

MUAC (cm)	Classification
Men	
≥22.5	Low risk of undernutrition
<22.5	Possible risk of undernutrition
Women	
≥17.7	Low risk of undernutrition
<17.7	Possible risk of undernutrition

*Based on 5th percentiles from Bishop *et al.* (1981). Norms for nutritional assessment of American adults by upper arm anthropometry. *Am. J. Clin. Nutr.* **34,** 2530–9.

Table 25.4 Classification of undernutrition in adults and children using *Z*-scores*

Z-score	Type and degree of undernutrition (ICD code)
–1 to +1	No undernutrition
–1 to –2	Mild undernutrition (E44.0)
–2 to –3	Moderate undernutrition (E44.1)
<–3	Severe undernutrition (E43)

*Adapted from Stratton, R. J., Green, C.J., and Elia, M. (2003). *Disease-related malnutrition:* Reproduced with permission CABI publishing.

Contributing causes

In most cases, the causes of undernutrition are multifactorial (Fig. 25.2). An awareness of some specific contributory factors is a valuable first step in prevention. The following is just a brief summary.

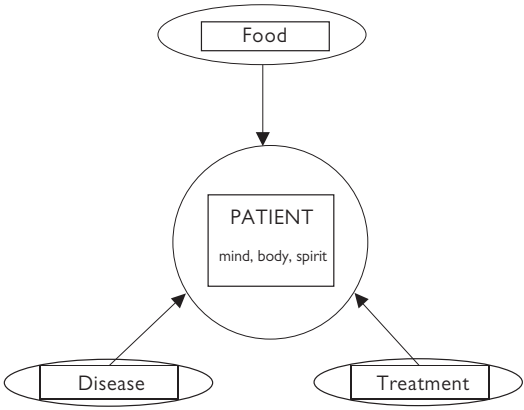

Fig. 25.2 To identify key causative factors of undernutrition, a holistic view of the patient is required.

Reduced nutritional intake

- Inadequate food availability (quantitative or qualitative):
  - patients nursed in isolation where meal trays may be left outside a single room or where they cannot be reached;
  - repeated deliberate starvation, e.g. nil by mouth for multiple tests or treatment;
  - slow motor co-ordination requiring feeding assistance;
  - culturally inappropriate meals, e.g. providing non-halal or non-kosher food for Muslims or Jews (see 🕮 Chapter 16, p. 305).
  - poor quality or unappetizing food.
- Anorexia (loss of appetite):
  - effects of disease, e.g. cancer, infection, inflammation;
  - nausea and vomiting;
  - psychological issues, e.g. depression, anxiety, loneliness;
  - effects of treatment, e.g. chemotherapy.
- Eating problems:
  - poor dentition;
  - changes in taste and smell;
  - dry or painful mouth;
  - breathlessness;
  - disordered swallowing.

Reduced nutrient absorption

- Insufficient GI secretions, including bile and all digestive enzymes, e.g. lack of pancreatic enzymes.
- Damage to absorptive GI surface, e.g. Crohn's disease.

- Gastrointestinal resection ± fistulae.
- Complication of drug therapy.

Increased requirements

- Disease-related hypermetabolism, e.g. liver cirrhosis, some cancers.
- Infection.
- Treatment-related, e.g. post-surgery.
- ↑ Losses, e.g. via gastrointestinal (GI) tract, urine, skin, breath, or surgical drains.
- ↑ Activity, voluntary and involuntary, e.g. Parkinson's disease.

Consequences

The effects of undernutrition vary from subclinical with no obvious clinical impairment to death, and are dependent on the type, length, and degree of nutritional inadequacy and the age and nutritional and health status of the individual.

Survival in the total abstinence from any nutrient intake (water only) is:

- ~55–75 days in lean adults;
- ~32 days newborn infant;
- ~5 days pre-term infant.

In addition to a significant ↑ risk of mortality, undernutrition is associated with greater morbidity:

- Weight loss (predominantly fat and muscle).
- Impaired muscle function:
  - skeletal muscle—poor mobility, ↑ risk of falls;
  - respiratory—↑ risk of chest infection, ↓ reduced exercise capacity, delayed ventilator weaning;
  - cardiac—bradycardia, hypotension, ↓ cardiac output;
  - GI tract—↓ gut wall integrity increasing potential for micro-organism access.
- Reduced immune function:
  - ↓ phagocytosis, ↓ chemotaxis, ↓ intracellular bacterial destruction, ↓ T lymphocytes;
  - ↑ rates of infection;
  - poor response to vaccination.
- Impaired synthesis of new protein:
  - poor wound healing, ↑ risk of ulceration;
  - delayed recovery from surgery;
  - growth faltering or cessation in children;
  - ↓ fertility in women and men.
- Psychological impairment:
  - depression, anorexia, ↓ motivation;
  - ↓ quality of life;
  - intellectual impairment if malnourished in infancy.
- Increased economic cost:
  - ↑ complications;
  - ↑ length of stay in hospital and intensive care unit;
  - ↑ re-admission rates following discharge;
  - longer rehabilitation;
  - ↑ pharmaceutical cost;
  - ↑ visits to GP.

Treatment of undernutrition

Why bother treating undernourished patients? There is good evidence that nutritional support can increase energy and protein intake, improve body weight and attenuate weight loss, improve functional outcomes (muscle strength, walking distances, activity levels, mental health) and clinical outcomes (mortality, complications, length of hospital stay) in both hospital and community settings.

The following numbered sections correspond to the numbered stages in Figure 25.3:

1. Assessment

On diagnosis of undernutrition, a full nutritional assessment should be undertaken to identify contributing causes (see 📖 this Chapter 'Undernutrition', p. 508) and provide a basis for treatment.

After assessment, the following steps can be taken. Although they are suggested in a sequential path, it may be appropriate to undertake a number simultaneously and utilize points that are relevant to the individual being treated.

2. Food access

After assessment, it may become apparent that some relatively simple, non-technical measures are needed to help the undernourished individual access suitable food. Examples:

- arranging support through appropriate carers, e.g. shopping, cooking, company whilst eating;
- locating/repairing dentures;
- providing appropriate cutlery, dishes, utensils, etc. Seek expertise from occupational therapy;
- modifying texture of foods provided (see 📖 Chapter 23, 'Cardiovascular disease', p. 465);
- requesting suitable meals, e.g. vegetarian, halal, kosher (see Tables 16.1–16.3).

3. Supplementation using food

The modification and/or supplementation of food and drink using ordinary food items can substantially increase energy and nutrient intake in many patients. This is a relatively straight forward step and should be tried before more complex interventions are initiated. The patient's nutritional status must be monitored regularly. Examples:

- ensuring three or four meals each day;
- offering nutritious snacks between meals, e.g. small sandwiches, cheese and biscuits, yogurt;
- limiting drinks or foods that provide little energy or nutrients, e.g. low calorie drinks, salads, clear soups;
- replacing all low fat items with higher fat alternatives (e.g. full cream milk);
- increasing energy/protein density of meals and drinks by fortifying, e.g. adding butter, margarine, olive oil, grated cheese to mashed potato or savoury sauces or adding sugar, honey, jam, milk powder, cream to desserts, milky drinks, etc.

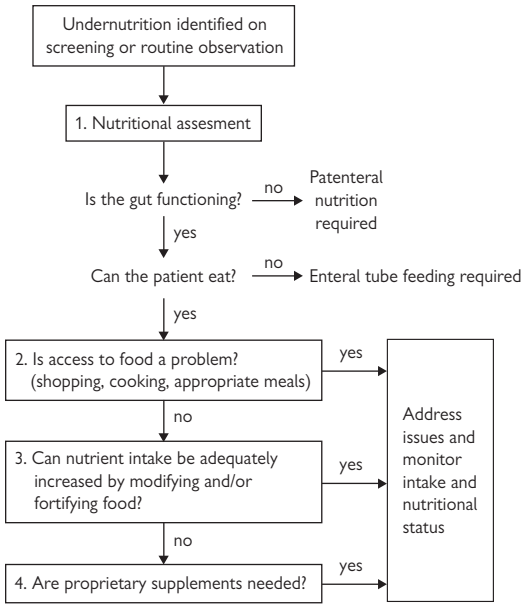

Fig. 25.3 Algorithm showing pathway of nutrition support for undernourished patients.

- Care must be taken to ensure micronutrient intake is adequate when using fortifying food with energy or protein only;
- 'treats' like cake, biscuits, chocolate, crisps, etc., can provide valuable additional calories but should not replace meals providing protein and range of other nutrients;
- alcohol, as an aperitif or added to milky drinks, can stimulate the appetite and provide some extra energy.

Advantages include flexibility, palatability, the non-medicalization of eating, and lower cost. Success depends on the patient being able to consume enough food and a dedicated team of carers and/or health professionals.

Disadvantages include the requirement of a high level of motivation and effort ± culinary skills in patient, carers, and health professionals, the limited availability of appropriate ingredients in institutional food production, the difficulty in measuring ↑ intake, and potential need for additional micronutrient supplementation.

4. Supplementation using proprietary oral nutrition supplements

Many ready-to-use oral nutrition supplements, often called sip feeds, are available that can contribute to a nutritionally well-balanced intake. These may be used in conjunction with the food fortification described above

or used to make up a deficit if an individual cannot eat sufficient food. Some products are prescribable for patients who are undernourished (Table 25.5; see also 🕮 Chapter 38, 'Drug nutrient interactions and prescription of nutritional products', p. 737).

Table 25.5 Examples of proprietary oral nutrition supplements available on prescription

Type	Brand name (manufacturer)
Milk-based drinks	Complan® Shake (Complan Foods)
	Fresubin® Original (Fresenius Kabi)
	Resource® Shake (Nestlé)
Juice-based drinks	Ensure® Plus Juice (Abbott)
	Fortijuice® (Nutricia Clinical)
Savoury drinks	Ensure® Plus savoury (Abbott)
	Vitasavoury® (Vitaflo)
Desserts	Clinutren® Dessert (Nestlé)
	Forticreme® Complete (Nutricia Clinical)

These products can also be bought without a prescription, but are relatively expensive. Non-prescribable powdered supplements, which need to be mixed with milk or water, provide comparable nutrition and are a cheaper alternative and easier to transport, e.g. Build Up® (Nestlé).

Advantages include known composition, most provide well-balanced intake of energy, macro- and micro-nutrients (but see caution at the end of this section), availability of ready-to-use form requiring little or no preparation, range of products and flavours, no cost to patient if prescribed.

Disadvantages include the 'quick fix' of readily dispensed products without full evaluation of patient's needs, flavour-fatigue after prolonged use, prescribing cost, medicalization of nutritional intake may further discourage eating, and (ready-to-use only) bulky/heavy and require storage space for patients at home.

Care must be taken to ensure micronutrient intake is adequate when using products in a supplementary role as they may not provide sufficient to meet requirements.

Next step

If an undernourished patient is unable to achieve an adequate intake orally using the above suggestions and is considered to need additional support, feeding artificially via the gut (see 🕮 this Chapter, p. 516) or directly into the blood (see 🕮 this Chapter, p. 536) will be required.

Box 25.5 Ethical issues when starting or stopping nutritional support

- Consent must be obtained from the patient if he or she is competent
- Act in the patient's best interest if he or she is not competent to give consent
- People receiving nutritional support, and their carers, must be kept informed about their treatment. They should also have access to appropriate information and be given the opportunity to discuss diagnosis and treatment options
- Provision of nutrition support is not always appropriate
- Decisions on withholding or withdrawing of nutrition support require a consideration of both ethical and legal principles (both at common law and statute including the Human Rights Act 1998).

See 🕮 Chapter 35, 'Palliative care', p. 715 and 🕮 Chapter 32, 'Mental Capacity Act 2005', p. 687.

Enteral feeding: introduction

'Enteral' refers to the GI tract so theoretically, 'enteral feeding' encompasses all nutrition assimilated via the gut, including eating and drinking. However, in clinical practice the term is usually used to describe the administration of nutritional feed into the gut through a tube including via nasogastric, nasojejunal, gastrostomy, and jejunostomy routes. Wherever possible, nutrition should be provided by enteral feeding.

Box 25.6 Advantages of enteral over intravenous feeding

- More physiological than feeding synthetic nutrients into blood
- Absorbed nutrients transported via portal circulation directly to liver to support synthesis and metabolic regulation
- Promotes integrity of GI tract mucosa
- Reduces bacterial translocation, e.g. bacteria migrating from gut lumen into circulation, so associated with lower risk of sepsis and multi-organ failure
- Stimulates gall bladder emptying so reducing risk of gallstone formation
- Provides (usually) all dietary constituents including some conditionally essential, e.g. glutamine, which may not be added to intravenous formulae
- Provides (usually) dietary fibre which stimulates colonocytes and short chain fatty acid production, optimizing bowel function
- Microbiologically safer than intravenous feeding
- Avoids complications associated with intravenous access including pneumothorax, catheter embolism, etc.
- Cheaper
- Easier (usually) for staff, carers, and patients to manage.

Most complications associated with enteral feeding, e.g. diarrhoea, tube dislodgement, can be overcome ± managed by experienced administration and following correct clinical procedures (see elsewhere in this Chapter).

In order to undertake enteral feeding, the patient must have some GI tract function.

❗ Absolute contraindications include:

- obstruction;
- prolonged ileus;
- severe GI tract bleeding (while bleeding is active and patient is haemodynamically unstable).

The enteral routes available are described in the following section.

Routes for enteral feeding

Nasogastric feeding

Why feed via the nose?

- Nutrients can be delivered into the gut more easily through a tube passed via the nose, rather than by mouth, because the tube can be fixed more securely and is less likely to dislodge.
- A nasal tube is less likely to disturb eating, if the patient is able to, and so will help facilitate transfer back on to oral nutrition.

Nasogastric (NG) feeding is indicated when:

- the patient is unable to take sufficient nutrition orally, e.g. severe anorexia, dysphagia, mouth/jaw injury, or it is unsafe to do so;
- the GI tract is functioning normally;
- nutrition support is predicted to be required for <28 days;
- nutrition support likely to be required for >28 days but gastrostomy contraindicated.

Tubes and placement

- Fine bore tubes (French gauge 6–9) should be used in preference. Wide bore tubes or 'Ryles' tubes (French gauge 10–18) are associated with ↑ complications, e.g. nasal/oesophageal ulceration and ↓ patient comfort.
- External tube diameter (Table 25.6) is important for patient comfort; internal diameter influences the flow of feed but varies for a given French gauge—contact tube suppliers for details.
- NG tubes should be placed by healthcare professionals who have relevant training and appropriate skills. A risk assessment should be carried out before insertion and local procedures should be followed.
- Tube should be passed via nose with the help of integral guide wire (do not re-insert once removed); if possible, ask the patient to sit rather than lie during insertion; swallowing sips of iced water as tube is inserted helps (providing patient is safe to do so, see 🕮 Chapter 23, 'Cardiovascular disease', p. 465).
- Correct tube position must be confirmed (see Fig. 25.4):
  - after initial insertion;
  - before administering each feed and after breaks in feeding;
  - at least once daily during continuous feeding;
  - following episodes of vomiting, retching and coughing;
  - if evidence of tube displacement, i.e. tape is loose or visual tube is longer;
  - before administering medication.
- Confirm tube position by:
  - check aspirate using pH sticks or paper (do not use blue litmus paper); pH<5 is indicative of gastric placement in adults;
  - X-ray is recommended but should not be used routinely;
  - do not check placement by injection of air and auscultation ('whoosh' test), absence of respiratory distress, watching for air bubbling from tube or observing feeding tube aspirate.

For what to feed, see 🕮 this Chapter, 'Enteral feeding regimes', p. 524.

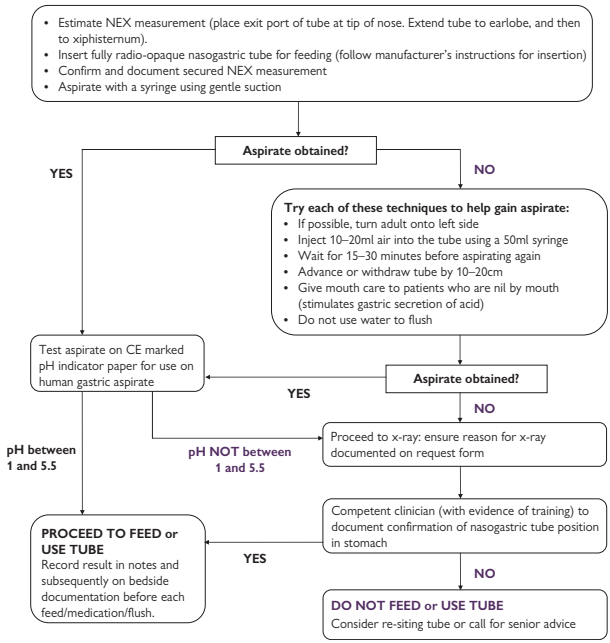

Fig. 25.4 Confirming the correct placement of nasogastric feeding tubes in adults. Reproduced with the kind permission of the National Patient Safety Agency (2011). http://www.nrls.npsa.nhs.uk/alerts/?entryid45=129640.

Table 25.6 External diameters corresponding to different French gauges

French gauge	External diameter (mm)	French gauge	External Diameter (mm)
5	1.7	14	4.7
6	2.0	16	5.3
8	2.7	18	6.0
10	3.3	22	7.3
12	4.0	24	8.0

Nasojejunal feeding

Nasojejunal (NJ) feeding enables nutrition to be infused into the gut distally to the pyloric sphincter. It is the route of choice when feeding is required for <28 days and the upper GI tract is either dysfunctional or inaccessible, but the GI tract is both functional and accessible distal to the stomach. Systematic review found no advantages to providing enteral feed post-pylorically rather than into the stomach and found that the difficulty in tube placement may lead to a delay in the initiation of feeding.[1]

Advantages:

- reduces risk of aspirating feed due to gastroparesis;
- facilitates early post-operative feeding.

Disadvantages:

- placement of tube is more difficult;
- tube may migrate into stomach.

Tubes and placement

- Fine bore tubes of adequate length (100–120 cm) are required.
- Double or triple lumen tubes are available to facilitate simultaneous feeding (post-pylorus), aspiration (gastric) ± pressure regulation.
- To ensure placement in the jejunum, NJ tubes usually require endoscopic insertion, guidance radiologically or by gastric ECG. Recently, successful post-pyloric placement using 'blind' insufflation of 200ml of air[2] or electromagnetic imaging[3] has been reported. Placement beyond the ligament of Treitz, i.e. distal section, will reduce migration into stomach.
- For unguided placement, spiral-ended tubes have a higher rate of post-pyloric placement than straight-ended tubes.
- Initial placement should be confirmed by an abdominal X-ray (unless tube placed radiologically).

For what to feed, see 🕮 this Chapter, 'Enteral feeding regimes', p. 524.

Gastrostomy feeding

A feeding gastrostomy is an artificial route made between the stomach and outside the body for feeding when the GI tract between mouth and stomach is inaccessible or unsafe or when long term enteral tube feeding is anticipated. Although this can be made surgically if the patient is undergoing an upper abdominal operation, it is more commonly formed as a percutaneous endoscopic gastrostomy (PEG) or radiologically-inserted gastrostomy (RIG).

[1] Marik, P.E. and Zaloga, G.P. (2003). Gastric versus post-pyloric feeding a systematic review. *Crit. Care* **7**, R46–51.

[2] Lee, A.J., Eve, R., and Bennett, M.J. (2006). Evaluation of a technique for blind placement of post-pyloric feeding tubes in intensive care: application in patients with gastric ileus. *Intens. Care Med.* **32**, 553–6.

[3] Windle, E.M., Beddau, D., Hall, E. *et al.* (2010). Implementation of an electromagnetic imaging system to facilitate nasogastric and post-pyloric feeding tube placement in patients with and without critical illness. *J. Hum. Nutr. Dietet.* **23**, 61–8.

Percutaneous endoscopic gastrostomy

Indication

- Patients requiring enteral nutrition support >28 days. Insertion is minimally invasive and ↑ comfort than an NG tube.

Contraindications

- Pharyngeal/oesophageal tumour (depending on tumour position).
- Portal hypertension.
- Peritoneal dialysis.
- Coagulopathy.

Advantages

- ↓ Inadvertent removal of tube so fewer disruptions to feeding.
- ↓ Reflux and aspiration of feed (as tube cannot migrate into the oesophagus) so overnight feeding may be safer.
- Cosmetically more appealing, especially for patients who are 'out and about'.
- Discreet fixation devices are available that facilitate tube detachment and are more practical for active patients, especially children.
- Easily removed when no longer needed, either endoscopically or by cutting (internal fixation device passes out through GI tract).

Complications

- Tube blockage (see 🕮 this Chapter, 'Complications of enteral feeding', p. 530).
- Tube displacement. Reasonable care is needed to avoid inadvertently removing PEG. Tube length and position of external markers should be checked daily to ensure tube has not migrated into/from the stomach. Those held by an internal balloon device may dislodge if balloon deflates—this should be checked weekly by withdrawing and replacing water contained within. If the tube is inadvertently removed, the tract will remain patent for ~48 h. A *temporary* Foley catheter can be inserted if the tract is mature (but not if newly formed) until the PEG is replaced.
- Peristomal infection/abscess. This can be ↓ by giving prophylactic antibiotics at insertion and undertaking good clinical practice, e.g. bathe, dry carefully, and check daily for redness, signs of gastric leak.
- 'Buried bumper syndrome'. This may occur if the internal fixation plate becomes buried within the abdominal wall due to overgrowth. This can be prevented by releasing the tube and rotating it by 360° every week (except in those with jejunal extension). PEGs held by an internal balloon are not prone to 'buried bumper syndrome' ∴ do not need to be rotated.
- Feed aspiration leading to pneumonia. Regular patient monitoring is required, especially in more vulnerable patients.

Practicalities

PEGs can be inserted as a day-care procedure providing appropriate after-care is available. Feeding can commence 4 h after insertion if there are no apparent complications (see 📖 this Chapter, 'Complications of enteral feeding', p. 530). Ongoing supplies of feed and consumable items, 'plastics', required for delivery and PEG care need to be organized for patients in the community along with education for them and their carers about how to manage feeding with maximum care and minimum stress.

The most common diagnosis for PEG insertion in adults in the UK is cerebrovascular accident, particularly in patients aged >64 years. A high mortality rate in first 30 days after insertion has been reported; this is likely to reflect the patient population, rather than PEG-related complications, but it is a reminder that the decision to insert a PEG should be taken holistically and consider ***all*** aspects of the patient's care and prognosis.

Other types of gastrostomy

- A percutaneous radiological gastrostomy (PRG; radiologically inserted gastrostomy, RIG) may be inserted under X-ray guidance and is more suitable for patients with compromised ventilation if endoscopy sedation is undesirable.
  - Insertion success rate is > PEGs.
  - Longevity of tubes < PEGs (need to plan replacements).
  - Associated with some post-procedural pain.
  - Different after care to PEGs: all except pig-tail type require daily 360° rotation (or follow local guidance).
- A surgical gastrostomy may be placed if an endoscopic insertion is not possible and the patient is undergoing surgery for other reasons. Complications include haemorrhage, skin excoriation from leaking gastric fluid, wound dehiscence, and intraperitoneal leakage of gastric contents.

❗ Patients with a gastrostomy may be hospital inpatients, but many manage their own nutrition in the community, either alone or with the help of carers. Advice on management of both feeding and gastrostomy care, and practical assistance is vital for this nutrition support to be completely successful and not become another burden for the chronically ill person. Dietitians and nutrition nurses with expertise in this area have a vital role to play and their valuable input cannot be overemphasized.

For what to feed, see 📖 this Chapter, 'Enteral feeding regimens', p. 524.

Further information

🖱 http://www.nice.org.uk/nicemedia/live/10978/29979/29979.pdf.

Slater, R. (2009). Percutaneous endoscopic gastrostomy feeding: indications and management. *Br. J. Nurs.* **18**, 1036–43.

Jejunostomy feeding

A feeding jejunostomy is an artificial route made between the jejunum and outside the body; occasionally access will be made into the duodenum (duodenostomy). It is used when the GI tract between mouth and jejunum is inaccessible, unsafe or malfunctioning and when long-term enteral tube feeding is anticipated.

The indications are similar to those for gastrostomy, i.e. nutritional support required for >28 days, but where post-pyloric feeding is required, e.g. in gastric stasis. Contraindications are the same.

Percutaneous endoscopic jejunostomy

This may be placed in patients with a high risk of aspiration pneumonia where a PEG is considered unsuitable. The insertion technique is more demanding. As an alternative, a conversion kit can be attached to an existing PEG to deliver feed into the jejunum although some risks remain.

Surgical jejunostomy

A number of different techniques are available including Witzel and Roux-en-Y procedures and the insertion of a needle catheter jejunostomy, which is the most common and usually placed during abdominal surgery.

For what to feed, see 🕮 this Chapter, 'Enteral feeding regimens', p. 524.

Enteral feeding regimens

- The patient's nutritional requirements should be estimated (see elsewhere in this Chapter). Energy and protein are very important and usually take priority, but fluid, electrolytes, macro- and micronutrients, and fibre also need consideration.
- The type and amount of feed that will provide the requirements should be calculated—a dietitian will be best placed to advise.
  - Most standard feeds = 100 kcal and 4 g protein/100 ml.
  - Most high energy feeds = 150–200 kcal and 6 g protein/100 ml.
- Method and rate of administration should be determined to ensure all feed is given in a way best suited to the patient's preference, convenience and medication. Options include:
  - *continuous infusion* over 16–24 h ± regulated by a pump. This should be used for patients in intensive care and for those who have difficulty tolerating large quantities of feed. If insulin is required, it is safe and more practical to feed over 24 h;
  - *intermittent infusion* over 8–20 h ± regulated by pump. Providing 'rest' periods may facilitate patient activity, eating, and sleeping; alternatively, feeding can be undertaken overnight. Stopping feeding for >4 h is associated with ↓ gastric pH and ↑ antibacterial effect;
  - *bolus feeding* of 100–500 ml given by gravity feed or syringed in over 10–30 min x 4–10 times daily. Time-consuming and may lead to abdominal symptoms especially in sick patients but is more physiological, e.g. resembles 'meal pattern', so can work well for stable patients.
- Initial administration of feeding needs special consideration:
  - There is little evidence that starter regimens, i.e. diluted feeds or very slow rates of initial delivery, are associated with improved feed tolerance, even with hypertonic feeds, but they will result in delayed administration of the full feed volume. Dilution of ready-to-feed enteral preparations also introduces a risk of contamination.
  - If the patient has been eating relatively normally prior to starting enteral feeding and is reasonably well, then the full strength feed should be initiated at 50% of the target volume that will meet requirements. This should be increased to the target volume over the first 24–48 h according to metabolic and GI tolerance.
  - If the patient has been consumed little or no nutrition by mouth or enterally for more than 5 days, feeding should be maintained at 50% of requirements for 2 days. Feeding should then be increased to the target volume if clinical and biochemical monitoring indicates no refeeding problems (❗ see 🕮 this Chapter, 'Refeeding syndrome', p. 544). Patients who are very sick or have GI symptoms may tolerate feeding better if this approach is taken.

  - A balance is required between a cautious approach to avoid refeeding syndrome and feed intolerance and further deterioration of nutritional status due to delay in reaching the target nutrition support. This can be minimized by regular monitoring (see 📖 this Chapter, 'Monitoring enteral feeding', p. 526) and adjusting feeding accordingly.
- It is not necessary to check gastric aspirates in stable patients who tolerate feeding well. For acutely ill patients, i.e. feeding on intensive care or high dependency units, see 📖 this Chapter, 'Critical care', p. 550.

Monitoring enteral feeding

- Monitoring the patient on a regular basis will minimize the risk of developing complications and help ensure that the patient's nutritional requirements are met and contribute to the best use of resources.
- Categories of variables for monitoring include:
  - nutritional;
  - anthropometric;
  - clinical;
  - laboratory.
- Deciding which variables and how often to monitor them depends on the clinical and nutritional status of the patient, the disease process, the duration of feeding and the patient's location. Obviously, a stable patient who has been fed via a PEG for 18 months and lives at home will require <monitoring than a patient receiving NG feeding on an intensive care unit. The guidance in Tables 25.7 and 25.8, drawn from the National Institute for Health and Clinical Excellence, is an outline that should be adapted to the needs of individual patients.

Table 25.7 Guidelines for laboratory monitoring of enteral feeding in hospital inpatients*

Variable	Frequency
Na^+, K^+, urea, creatinine	Baseline, daily until stable, then 1–2 times/week
Blood glucose	Baseline, 1–2 daily (or more if needed) until stable, then weekly
Mg^{++}, PO_4	Baseline, daily if risk of refeeding, 3 times/week until stable, then weekly
LFTs, INR	Baseline, twice/week until stable, then weekly
Ca^{++}, albumin	Baseline, then weekly
C reactive protein	Baseline, then 2–3 times/week until stable
Zn^{++}, Cu^{++}	Baseline, then every 2–4 weeks depending on results
Full blood count, MCV	Baseline, 1–2 times/week until stable, then weekly
Fe^{++}, ferritin	Baseline, then every 3–6 months
Folate, B_{12}	Baseline, then every 2–4 weeks

Monitoring of Mn, 25 OH vitamin D and bone density is rarely needed in patients receiving enteral feeding unless there is cause for concern.

*Adapted from NICE (2006). *Nutritional support in adults.* Clinical guideline 32
http://www.nice.org.uk/nicemedia/live/10978/29979/29979.pdf

Table 25.8 Guidelines for nutritional, anthropometric and clinical monitoring of enteral feeding in hospital inpatients*

Variable	Frequency
Nutrient intake[c]	Daily
Volume of feed delivered[c]	Daily
Fluid balance chart	Daily
Weight[c]	Daily if concerns about flid balance, otherwise weekly reducing to monthly
BMI[c]	Start of feeding and then monthly
Mid-arm circumference[c]	Monthly if weight cannot be obtained or is difficult to interpret
Triceps skin-fold	Monthly if weight cannot be obtained or is difficult to interpret
Nausea/vomiting[c]	Daily initially reducing to twice weekly
Diarrhoea[c]	Daily initially reducing to twice weekly
Constipation[c]	Daily initially reducing to twice weekly
Abdominal distension	As necessary
Nasally inserted tubes	
Tube placement[c]	Before each feed begins
Nasal erosion[c]	Daily
Tube fixation[c]	Daily
Tube integrity[c]	Daily
Gastrostomy or jejunostomy tubes	
Stoma site[c]	Daily
Tube position[c]	Daily
Tube insertion and rotation[c]	Weekly
Balloon water volume[c]	Weekly
General clinical condition[c]	Daily
Temperature	Daily initially and then as needed
Blood pressure	Daily initially and then as needed
Drug therapy[c]	Daily initially reducing to monthly when stable
Are goals being met?[c]	Daily initially, reducing to twice weekly and then progressively to 3–6 months unless clinical condition changes

(continued)

Table 25.8 (*Contd.*)

Variable	Frequency
Are goals still appropriate?[c]	Daily initially, reducing to twice weekly and then progressively to 3–6 months unless clinical condition changes

[*] Adapted from NICE (2006) Nutritional support in adults. Clinical guideline 32

http://www.nice.org.uk/nicemedia/live/10978/29979/29979.pdf

[c] Variables that should be monitored in patients on long-term enteral feeding in the community, but see p. 528 for frequency.

Monitoring enteral feeding in stable patients in the community

- Who by? Monitoring should be undertaken by a healthcare professional with relevant training and skills. Some observations may be made by the patient and/or their carers and where possible, training and support should be provided for them, especially in understanding when to report observations and who to.
- Frequency? Every 3–6 months or more frequently if condition changes.
- Which variables? See Table 25.7, variables marked with[c]. Laboratory tests are rarely needed in stable patients where feeding is successfully established.

Complications of enteral feeding

Tube blockage (nasogastric, nasojejunal, percutaneous endoscopic gastrostomy)

Prevention

- Flush tube with water every 6 h and at start and end of rest periods. Tap water is suitable, unless patient is immune-compromised or fed post-pylorically or standard of tap water is unsafe (then use cooled boiled or sterile water). Use 30–50 ml water in a 50-ml syringe.
- Medication in liquid form can be very sticky so tube should be flushed with water before and after drug administration. Crushed tablets given by feeding tube may lead to blockages, and this mode of administration is generally outside the drug's product license, although may be preferred to sticky solutions in some units (see 🕮 this Chapter, 'Enteral feeding and drugs', p. 534).
- Low pH (associated with gastric aspirates) encourages protein precipitation. Flush tube with water after each aspiration.
- Use a pump when feeding at low volume rates.

Unblocking

- Never use guide wire—it may pierce tube and injure patient.
- Ensure tube is un-kinked and feel external part of tube to identify any lumps. If located, these may be dispersed by squeezing tube gently.
- Use a 50-ml syringe of cold water to apply push/pull pressure.
- Progress on to warm water, again using push/pull technique. Leave water in tube for ~30 min and repeat.
- Progress on to fizzy water and repeat as above.
- Leave tube filled with water for up to 4 h and repeat.
- Numerous other agents, including cola and cranberry juice, are anecdotally recommended, but none are licensed in UK for unblocking tubes and the low pH of some may lead to coagulation of the feed and worsen the problem. Limited evidence suggests the most effective are water, pancreatic enzymes, and a commercial product, Corflo Clog Zapper (Merck Serono Ltd, Feltham UK) containing papain and amylase.

Aspiration

- ↑ Risk with ↑ age, ↑ debility, dementia, disordered swallow, sedation, supine position, ventilation, and low nursing levels.
- Occurs in 6–12% neurological patients and ~30% with tracheostomy.
- Feed may be aspirated into respiratory tract without obvious vomiting.
- Signs include dyspnoea, cyanosis, tachycardia and hypotension.
- Can lead to pneumonia with associated ↑ morbidity and mortality.
- Prevent by encouraging patients to lie in semi-recumbent position, elevating bed by 30–45° (unless haemodynamically unstable), use iso-osmotic feeds (optimize gastric emptying), reduce overnight feeding.
- Review mode/rate of feeding: continuous infusion may inhibit gastric emptying so changing to bolus feeding may ↓ aspiration risk in some patients. However, in gastroparesis, i.e. ventilated patients, bolus feeding may ↑ gastric volume and potential ↑ risk.

- Pro-motility drugs, e.g. metoclopramide, may be of benefit in ventilated patients with gastroparesis. In others, limited benefit and side effects have been reported so this medication is not generally recommended to prevent tube migration.[1]
- To manage gastric residual volume, see Bankhead *et al.* (2009).[2]
- Consider placing tube post-pylorically (see 📖 this Chapter, 'Routes for enteral feeding', p. 518).

Treat by stopping feed; try to aspirate feed from lungs; prescribe antibiotics if infection confirmed.

Diarrhoea

- Prevalence ~2–95% of patients receiving enteral tube feeds, depending on patient group and definition of diarrhoea.
- Characteristics of faecal output, e.g. frequency and consistency, are useful in identifying potential abnormality and action needed:
  - frequent liquid stools will compromise absorption, cause patient discomfort and present difficulties in care so will require intervention;
  - occasional semi-formed motions may be tolerated by patient and carers.
- Diarrhoea can have multiple causes and enteral feed should not automatically be blamed and stopped (Table 25.9).

Microbiological contamination

- Enteral feeds provide an ideal environment for bacteria to multiply in; the consequences of contamination are potentially very serious.
- All equipment for enteral feeding should be used only once. If bags and giving sets are used, they should be replaced every 24 h and feeding tubes replaced according to the manufacturer's guidelines.
- The maximum safe hang-time, i.e. duration of administration, will depend on the feed, patient and location of delivery. All unused feed should be discarded at the end the maximum feed time and replaced with fresh feed (see Table 25.10).
- Non-sterile feeds, e.g. re-constituted powder, should be prepared with strict attention to hygiene, covered and stored in a refrigerator for <24 h. Unused feed must be discarded.
- The reservoir of feed (bag, bottle, carton) must not be hung below the level of the patient's stomach.
- If the pump is inadvertently reversed, leading to the stomach contents being infused up into the feed reservoir, feeding should be stopped, all the feed and equipment must be discarded, and feeding re-started with new feed, bag, giving set, etc.
- Tap water from a main supply, i.e. drinking water, is suitable for tube flushing, unless patient is immune-compromised or fed post-pylorically. If the tap water comes from a tank or is of uncertain quality, use cooled boiled or sterile water.

[1] Silva, C.D., Saconato, H., Atallah, A.N., *et al.* (2009). Metoclopramide for migration of naso-enteral tube. *Cochrane Database of System. Rev.* CD003353.
[2] Bankhead, R., Boulata, J., Brantley, S., *et al.* (2009). Enteral nutrition practice recommendations. *J Parent. Enteral Nutr.* **33**, 122–67.

Table 25.9 Factors associated with causing diarrhoea and potential solutions

Causes	Potential solutions
Medication, especially antibiotics and sorbitol	Seek expertise from pharmacist to review and modify if possible
↓ Fibre in gut lumen and consequent effect on colonocytes	Consider changing to a fibre-providing feed, e.g. Ensure® Plus Fibre (Abbott), Isosource® Energy Fibre (Nestlé)
↓ or ↑ change in bowel microflora	Send stool specimen, e.g. to check for *Clostridium difficile*, *Escherichia coli,* and treat accordingly Consider probiotics orally or administered by syringe and flush (do not add to feed or post-pylorus)[1]
Rate of continuous feeding	Change to bolus regimen if feeding intragastrically, OR ↓ rate if feeding intraduodenally
Hyperosmolar feed	If using high-energy feed, consider changing to standard 1 kcal/ml feed and increasing volume. If using peptide or elemental feed, review osmolarity between brands
Contaminated feed	Ensure good clinical practice; see above
Constipation leading to overflow	Check if colon impacted with faeces; if so, prescribe suppository. Change to fibre feed, ensure adequate hydration, encourage mobility if patient able
Malabsorption (rather than directly feed-related)	Especially in patients with pancreatic or small bowel disease. Differentiate from feed-related diarrhoea by history, visual examination of faecal output (i.e. fatty globules) and spot faecal fat. If present, change feed to semi-elemental feed, e.g. Peptisorb® (Nutricia Clinical), Survimed® Fresenius Kabi. Prescribe pancreatic enzyme replacement therapy (see 📖 Chapter 27, 'Pancreatic enzyme replacement therapy', p. 616).

[1] Whelan, K., *et al.* (2010). Safety of probiotics in patients receiving nutritional support: a systematic review of case reports, randomized controlled trials, and nonrandomized trials. *Am. J. Clin. Nutr.* **91,** 687–703.

Table 25.10 Recommended maximum hang-time for enteral feeds*

Sterile feed in a closed system	24 h
Sterile feed in an open system at home	12 h
Sterile feed in an open system in hospital	8 h
Sterile feed in an open system in hospital (neonates)	4 h
Non-sterile feed, i.e. re-constituted from powder	4 h
Non-sterile feed, i.e. with additives	4 h

* Adapted from Bankhead, R., Boullata, J., Brantley, S., *et al.* (2009). Enteral nutrition practice recommendations. *J. Parent. Enter. Nutr.* **33**, 122–67.

Enteral feeding and drugs

Patients receiving enteral feeding are often unable to take medication orally and ∴ it may need to be administered via the feeding tube. Using this route, or crushing tablets and opening tablets is generally outside the drug's product license meaning that the prescriber and practitioner accept liability for any adverse effects resulting from their administration. In each case, advice should be sought from a pharmacist and the following considered.

- If the patient can still take the drug orally (and that is the licenced route), this is best.
- Review all medication—is it all still needed?
- Does the tube deliver the drug distal to the site of absorption?
- ❗ Drugs have a notorious reputation for blocking feeding tubes, especially sticky liquids and antacids, so care must be taken at each dosing.
- Should the drug usually be given before/after or with food? This may mean that the feed has to be stopped; the feeding regimen should be amended to take this into consideration so that the patient still receives the total volume prescribed.
- Drugs should not be added to the feed but should be introduced into the tube using a 50-ml syringe (smaller syringes ↑ pressure in tube and may cause it to split).
- Each drug should be administered separately, unless advised by a pharmacist, followed by 10 ml water. A gap may be required between different drugs.
- Wherever possible, prescribe medication in liquid form or as soluble tablets. Crushing tablets and opening capsules should be considered a last resort.
- Soluble tablets should be dissolved in 10–15 ml water before administration.
- Liquids should be diluted with an equal volume of water and mixed well before administration.
- Tablets that have to be crushed should be ground finely using a pestle and mortar or tablet-crusher. Mix with 10–15 ml water to syringe into tube. Rinse crusher and syringe-in rinse water to ensure full drug dose is given.
- Do not crush or chew tablets or capsules that are enterically-coated, modified, or slow release.
- Staff should wash their hands and wear gloves to minimize exposure to the drugs. Cytotoxic medication and hormones should not be crushed due to the risk associated with staff exposure.

More information, including patient information leaflet, is available at http://www.bapen.org.uk/res_drugs.html.

Parenteral nutrition

Parenteral nutrition (PN) refers to the administration of nutrients via the intravenous route. It is required when a patient has intestinal failure to a degree that prevents adequate absorption of nutrients via the GI tract. Complications associated with the access route and the nutrient formulation frequently occurs with PN, so careful patient selection and monitoring are essential.

Indications

PN may be required in the short term where the GI tract is temporarily unavailable.

- Examples where PN may be required short-term:
  - post-operative ileus with high gastric aspirates;
  - severe pancreatitis;
  - intensive chemotherapy causing mucositis;
  - multi-organ failure where nutritional requirements cannot be met by the enteral route alone;
  - prolonged nil by mouth following excisional surgery;
  - high output fistula.
- Examples where PN may be required long term:
  - extreme short bowel syndrome;
  - high output fistulae;
  - inflammatory bowel disease in conjunction with fistulae or short bowel syndrome;
  - radiation enteritis;
  - motility disorders (e.g. scleroderma);
  - chronic malabsorption.

Routes for provision of parenteral nutrition

Patients who require PN in the short term, can be fed continuously, and a regimen of <1200 mOsmols may be fed via a peripheral route, e.g. a venflon. The following may minimize the risk of thrombophlebitis.

- Access the largest peripheral vein available.
- Use a small cannula (18 Fr).
- Use a GTN (glyceryl trinitrate) patch distal to the exit site.

Where it is anticipated that patients may require parenteral nutrition for >14 days, are to be fed on a cyclical basis, or require a regimen of >1200 mOsmols, central catheter insertion may be appropriate. The tip of the catheter is surgically placed to lie in the lower portion of the superior vena cava or right atrium in order to minimize the risk of thrombosis. Examples include:

- single dedicated feeding line;
- peripherally inserted central catheter (PICC);
- multi-lumen using one lumen as dedicated feeding line.

Where PN is anticipated to be required on a more permanent basis, access can be provided by:

- Hickman or implanted port (Portacath);
- cuffed (Dacron cuff to secure).

Complications

See Table 25.11.

Table 25.11 Possible complications

Related to	Examples
Line insertion	Pneumothorax, haemothorax, air embolism
Access routes	Thrombophlebitis, central vein thrombosis, catheter-related bloodstream infection
PN solution	Fluid and electrolyte imbalance, metabolic disturbances, impaired liver function (long-term use)

To minimize the risk of infection always feed via a dedicated feeding line.

Parenteral nutrition regimens

PN is not an emergency treatment. Before introducing PN, it is important that a full assessment is completed by a suitably trained healthcare professional. This should include anthropometry, clinical condition, medication, biochemistry, recent oral history and risk of refeeding syndrome. From this, the nutritional requirements can be calculated and the appropriate regimen prescribed in order to minimize metabolic complications.

- To meet a patient's nutritional requirements PN must contain:
  - fluid;
  - nitrogen;
  - source of energy as a combination of carbohydrate and fat;[1]
  - electrolytes;
  - fat- and water-soluble vitamins;
  - trace elements.
- A pharmacy production unit may provide compounded solutions or standardized fixed feeding regimens.
  - *advantages of compounded regimens:* greater flexibility to meet the needs of complex patients whose individual requirements could not be met with a standardized regimen;
  - *advantages of standardized regimens:* wide range of formulations available, still some flexibility for adding electrolytes, less cost of having a specialized pharmacy production unit.
- Vitamins and trace elements should always be added to ensure the feed is complete.

Monitoring parenteral nutrition

See Table 25.12. Patients receiving parenteral feeding must be monitored to:

- detect potential complications associated with the feeding and/or their clinical condition;
- evaluate their nutritional status in relation to the nutrients provided and/or their clinical condition.

[1] Inclusion of fat is not necessary on a daily basis (can be given less often).

Table 25.12 Guidelines for nutritional, anthropometric and clinical monitoring of parenteral feeding*

Variable	Frequency
Nutrient intake	Daily
Volume of feed delivered[c]	Daily
Fluid balance chart	Daily
Weight[c]	Daily if concerns about fluid balance, otherwise weekly reducing to monthly
BMI[c]	Start of feeding and then monthly
Mid-arm circumference[c]	Monthly
Triceps skin-fold	Monthly
Nausea/vomiting[c]	Daily
Diarrhoea[c]	Daily
Constipation[c]	Daily
Abdominal distension	As necessary
Catheter entry site[c]	Daily
Skin over catheter tip[c]	Daily
General clinical condition[c]	Daily
Temperature	Daily
Blood pressure	Daily
Drug therapy[c]	Daily
Are goals being met?[c]	Daily initially, reducing to twice weekly and then progressively to 3–6 months unless clinical condition changes
Are goals still appropriate?[c]	Daily initially, reducing to twice weekly and then progressively to 3–6 months unless clinical condition changes

* Adapted from NICE (2006). *Nutritional support in adults*. Clinical guideline 32.

http://www.nice.org.uk/nicemedia/live/10978/29979/29979.pdf

[c] Variables that should be monitored in patients receiving parenteral nutrition in the community; see NICE guideline for frequency.

Table 25.13 Guidelines for laboratory monitoring of parenteral feeding*

Variable	Frequency
Na^+, K^+, urea, creatinine	Baseline, daily if at risk of refeeding syndrome until stable, then 1–2 times/week
Blood glucose	Baseline, 1–2 daily (or more if needed, e.g. in diabetes) until stable, then weekly
Mg^{2+}, PO_4	Baseline, daily if risk of refeeding, 3 times/week until stable, then weekly
LFTs, INR	Baseline, twice/week until stable, then weekly
Ca^{2+}, albumin	Baseline, then weekly
C reactive protein	Baseline, then 2–3 times/week until stable
Zn^{2+}, Cu^{2+}	Baseline, then every 2–4 weeks depending on results
Se	Baseline if risk of depletion, further tests depending on baseline
Full blood count, MCV	Baseline, 1–2 times/week until stable, then weekly
Fe^{2+}, ferritin	Baseline, then every 3–6 months
Folate, B_{12}	Baseline, then every 2–4 weeks
Mn^{2+}	Every 3–6 months if on long-term PN
25 OH vitamin D	Every 6 months if on long-term PN
Bone density	On starting long-term PN, then every 2 years

* Adapted from NICE (2006). *Nutritional support in adults.* Clinical guideline 32
http://www.nice.org.uk/nicemedia/live/10978/29979/29979.pdf

Monitoring parenteral feeding in stable patients in the community

- Who by? Monitoring should be undertaken on a routine basis by a community-based nurse or dietitian with training and skills in this area with regular follow up by an experienced hospital team. Some clinical observations may be checked by the patient and/or their carers and it is essential that training and support are provided for them, especially in understanding when to report observations.
- Frequency? Initially, at least weekly and, in addition, reviewed at a specialist hospital clinic every 3–6 months. Monitoring should be more frequent during the early stages of home PN or if there is a change in clinical condition.
- Which variables? Those variables marked with[c] in Table 25.12 and all variables in Table 25.13.

Estimating requirements in disease states

Estimating the nutritional requirements of healthy populations with any degree of accuracy is difficult; it is even more challenging to try to predict the needs of individuals in ill health and disease when substantial changes may arise. However, providing appropriate nutrition, i.e. sufficient but not excess, is important to maximize benefits and so every attempt should be made to tailor nutritional intake to the individual.

❗ The following guidelines provide a useful starting point but it must be remembered that:

- they are guidelines and not precise values;
- a carefully calculated estimate of requirements only has the benefit to help the patient if this intake is achieved, i.e. their feed must be given or supplements taken.

Energy

Table 25.14 gives a method of estimating energy requirements that is used in the following examples.

Example 1

Man aged 58 years, weight 81 kg, height 1.72 m, sitting up in bed following cerebral vascular accident. Unable to swallow at present and enteral feed to be started.

BMR = 11.5 x 81 + 873 = 1805 kcal
Clinical stress factor = 5% of 1805 = 90 kcal
Activity factor = 15% of 1805 = 271 kcal
Estimated energy requirement = 1805 + 90 + 271 = 2166 kcal

Example 2

Woman aged 23 years, weight 48 kg, height 1.64, immobile and septic on intensive care unit following complicated intestinal resection 2° to Crohn's disease. Nutrition support instigated.

BMR = 14.8 x 48 + 487 = 1197 kcal
Clinical stress factor = 60% of 1335 = 718 kcal
Activity factor = 10% of 1197 = 120 kcal
Estimated energy requirement = 1197 + 718 + 120 = 2035 kcal

Protein

Requirements should be based on actual body weight except in obesity (if BMI 30–50 kg/m^2 use 75% value; if BMI >50 kg/m^2 use 65% value). Table 25.15 gives an estimation of protein requirements in terms of nitrogen.

Carbohydrate

- Requirements for healthy and chronically sick are similar: 4–5 g glucose/kg/day.
- In critical illness, glucose oxidation rate should be considered. The maximum of 4–7 mg glucose/kg/minute should not be exceeded.

Table 25.14 Estimating energy requirements*

1	Estimate basal metabolic rate using the Schofield equation[a] (📖 Appendix 4)	
2a	*Either:*	
	Add to BMR a stress factor for specific clinical condition (calculated as % of BMR):	
	Condition[b]	**Stress factor (%)**
	Cerebral vascular accident (stroke)	5
	Chronic obstructive pulmonary disease	15–20
	Infection	25–40
	Inflammatory bowel disease	0–10
	Intensive care (ventilated)	0–10
	Intensive care (septic)	20–60
	Lymphoma	0–25
	Pancreatitis (chronic to acute ± abscess)	3–20
	Surgery (uncomplicated to complicated)	5–40
	Tumour (solid)	0–20
	Transplantation	20
2b	*Or:*	
	Add to BMR 400–1000 kcal/day if ↑ in body weight (lean ± fat) is desired or subtract 400–1000 kcal if ↓ in body fat is desired and patient is not metabolically stressed	
3	Add to BMR an activity factor (calculated as % of BMR)	
	Activity level	**Activity factor (%)**
	Patient in bed and immobile	10
	Patient in bed but able to move and sit up	15–20
	Patient mobile on ward	25
	Patient living in the community	Use PAL[c]

*Adapted from pp. 3.1–3.3a of Todorovic V.E., and Micklewright, A. (2007). *A Pocket Guide to Clinical Nutrition*. Permission requested for 1st edn from the British Dietetic Association.

[a] Requirements should be based on actual body weight except in obesity (if BMI 30–50 kg/m^2 use 75% value; if BMI >50 kg/m^2 use 65% value).

[b] For burn injury see Table 25.20.

[c] PAL – physical activity level (see 📖 Appendix 4, p. 772).

Table 25.15 Estimating protein requirements*

	Nitrogen (g/kg/day)†	
	Mean	**Rang**
Normal	0.17	0.14–0.20
Hypermetabolic		
+5 to +25%	0.20	0.17–0.25
+25 to +50%	0.25	0.20–0.30
+>50%	0.30	0.25–0.35
Depleted	0.30	0.20–0.40

*Adapted from pp. 3.9 Todorovic V.E., *et al.* (2007). *A Pocket Guide to Clinical Nutrition*. from the British Dietetic Association.

†1 g nitrogen ≡ 6.25 g protein; 1 g protein ≡ 0.16 g nitrogen.

Lipid

- Requirement in health: 1.0–1.5 g/kg/day.
- In critical illness: 0.8–1.0 g/kg/day.
- Lower levels of fat intake can be tolerated well but ~3.0–4.5% total energy should be provided as lipid to prevent essential fatty acid deficiency.

Fluid

- Basic requirements: 25–35 ml/kg (but evaluate carefully in very underweight or obese patients).
- Pyrexia: add 2.0–2.5 ml/kg for every °C above 37°C.
- Fluid lost via body secretions: replacement should be considered on an individual basis.

Electrolytes

Table 25.16 lists the basic daily requirement for electrolytes.

Micronutrients

There is limited evidence about the requirement of micronutrients (vitamins, minerals, and trace elements) in disease states. In most cases, it is probably appropriate to provide the equivalent to the reference nutrient intake (see 📖 Appendix 6, p. 780). These values are based on providing sufficient for 97% of people in a healthy population, but this may not be adequate for some individual patients if previously depleted. In these cases, it is probably better to provide additional micronutrients in the form of a multi- rather than single-nutrient supplement, unless there is clinical or biochemical evidence of a specific deficiency. This is because of the complex interrelationships between many micronutrients and the potential for competitive absorption and/or biochemical pathways.

Table 25.16 Basic daily requirements for electrolytes

Electrolyte	Basic daily requirement
Sodium	1.0 mmol/kg
	In pyrexia, add 1.5 mmol to each additional 10 ml fluid given (see 📖 p. 542 for fluid requirements)
	In hyponatraemia, additional Na^+ (mmol) required = (140 – actual serum Na^+) x 0.2 x body weight (kg)
	Unless clinically indicated, all enteral and parenteral feeds should provide at least 50 mmol/day
Potassium	1.0 mmol/kg
	In hypokalaemia, additional K^+ (mmol) required = (4.0 – actual serum K^+) x 0.4 x body weight (kg)
Calcium	10.0–17.5 mmol (400–700 mg)
Magnesium	Male: 7.8–12.3 mmol (190–300 mg)
	Female: 6.2–10.9 mmol (150–270 mg)
Phosphate	Equimolar with calcium; do not exceed 50 mmol/day in enteral or parenteral feeds

* Adapted from Todorovic V.E., and Micklewright, A. (2007). *A Pocket Guide to Clinical Nutrition.* British Dietetic Association, London, pp. 3.11–3.11a

Refeeding syndrome

❗ Enthusiasm for nutritional support and a desire to replete very undernourished patients rapidly can be fatal unless care is taken to avoid refeeding syndrome (RfS). See Box 25.7 for a list of patients potentially at risk and Box 25.8 for the clinical features.

Definition

Severe fluid and electrolyte shifts and related metabolic complications in malnourished patients undergoing refeeding.

Pathophysiology

- In starvation, ↓ intake of energy and particularly carbohydrate → ↓ insulin secretion and ↑ catabolism of fat and protein for energy → ↓ intracellular electrolytes and especially ↓ phosphate (↓ intracellular phosphate co-exists with normal serum phosphate levels).
- Initiating feeding → change from predominantly fat and protein metabolism to carbohydrate with ↑ insulin secretion → stimulation of cellular uptake of phosphate, potassium, and water → hypophosphataemia, hypokalaemia, and hypomagnesaemia → RfS.

Key points for refeeding syndrome awareness

- Risk of refeeding may occur in response to oral, enteral, or parenteral nutrition support.
- The early features of RfS are non-specific and may not be recognized.
- Awareness and understanding of RfS by clinical staff is limited and serum phosphate is often not routinely measured and the significance of depleted levels is not always appreciated. Dietitians who work in nutrition support have an important role in increasing awareness about RfS.
- Normal serum values *before* feeding starts do not indicate that the patient is at low risk of RfS. In RfS, serum levels only fall *after* feeding starts so this is when monitoring must take place.
- In most cases, RfS can be anticipated and prevented.

Further reading

Boateng, A.A., Sriram, K., Mequid, M.M., *et al.* (2010). Re-feeding syndrome: treatment considerations based on collective analysis of literature case reports. *Nutrition* **26**, 156–67.

Stanga, Z., Brunner, A., Leuenberger, M., *et al.* (2008). Nutrition in clinical practice—the re-feeding syndrome: illustrative cases and guidelines for prevention and treatment. *Eur. J. Clin. Nutr.* **62**, 687–94.

Box 25.7 Patients potentially at risk from refeeding syndrome

Unintentional weight loss
- >5% body weight in 1 month
- >7.5% body weight in 3 months
- >10% body weight in 6 months

Low nutrient intake
- Patients starved for >7 days
- Prolonged hypocaloric feeding or fasting
- Chronic swallowing problems and other neurological disorders
- Anorexia nervosa
- Depression in the elderly
- Patients with cancer
- Chronic infectious diseases (AIDS, tuberculosis)
- During convalescence from catabolic illness
- Postoperative patients
- Diabetic hyperosmolar states
- Morbid obesity with profound weight loss
- Chronic alcoholism, homelessness, social deprivation
- Idiosyncratic/eccentric diets
- Hunger strikers

↑ *Nutrient losses or* ↓ *nutrient absorption*
- Significant vomiting and/or diarrhoea
- Dysfunction or inflammation of the GI tract
- Chronic pancreatitis
- Chronic antacid users
- Chronic high-dose diuretic users
- After bariatric surgery.

Adapted from Stanga, Z., Brunner, A., Leuenberger, M., *et al.* (2008). Nutrition in clinical practice—the re-feeding syndrome: illustrative cases and guidelines for prevention and treatment. *Eur. J. Clin. Nutr.* **62**, 687–94.

Box 25.8 Clinical features of refeeding syndrome

- Rhabdomyolysis, weakness, paralysis
- Leukocyte dysfunction, haemolytic anaemia
- Respiratory depression and failure
- Hypotension, arrhythmias, cardiac failure
- ↓ Glomerular filtration rate
- Liver dysfunction
- Diarrhoea, constipation, ileus
- Seizures, coma, sudden death.

Management of refeeding syndrome

See Fig. 25.5.

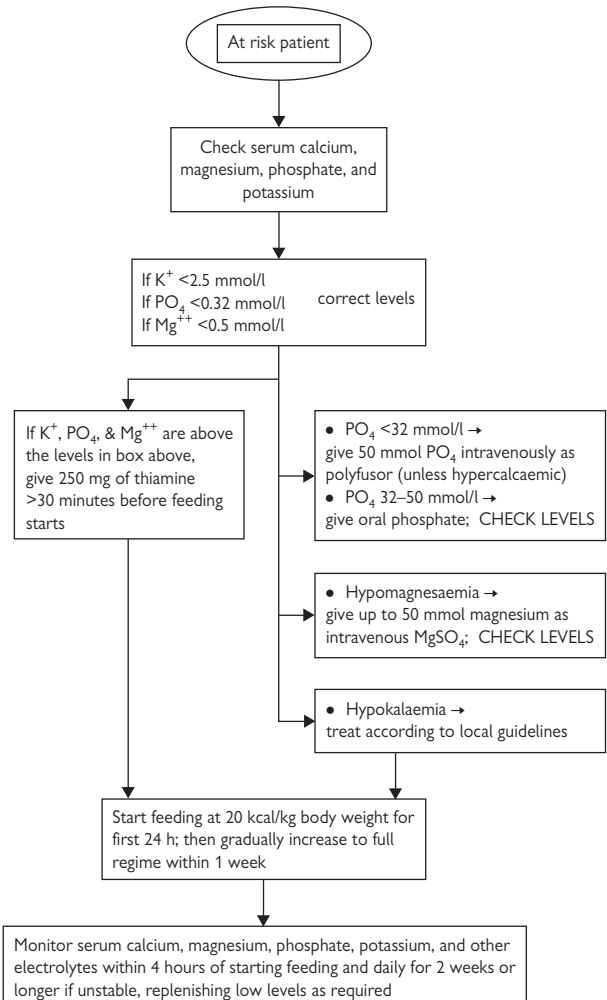

Fig. 25.5 Algorithm for managing refeeding syndrome. (Adapted from p. 13.7 of Todorovic, V.E., and Micklewright, A. (2004). *A pocket guide to clinical nutrition.* Reproduced with permission from British Dietetic Association, Birmingham).

Metabolic response to injury

The term 'metabolic response to injury' describes the biochemical and hormonal consequences of major injury, trauma, surgery ± infection and the resulting nutritional changes that may have very significant clinical effects. Traditionally, the response has been described as having two phases, known as the ebb and flow (Table 25.17). Recent studies have shown that good acute clinical management may reduce or possibly eliminate the ebb phase, and that it may not be detectable at all in less severe injury.

Overall effects

- Loss of appetite → ↓ nutrient intake.
- Perturbation of fat and carbohydrate metabolism with apparent inability to use these as metabolic substrates (hence ↑ circulating levels and deposition of lipid in adipose and vital organs). Controlling hyperglycaemia by giving insulin will help ↓ risk of death.
- Lean tissue broken down may provide amino acids required during inflammatory response, e.g. acute phase proteins, lymphocyte proliferation, glutathione synthesis.
- Protein loss—may be substantial and have clinical consequences (see Table 25.18). In context of 'whole body protein', lean tissue contains ~205 g protein/kg → an average 70 kg man comprises ~10 kg protein.

❗ Most of this protein is 'essential' and cannot be lost without functional implications, i.e. ↓ resistance to infection, ↑ muscle weakness (including respiratory and skeletal muscle) leading to ↓ pulmonary function and ↓ physical activity.

Effect of starvation

Although there are some similarities between the metabolic response to injury and starvation, i.e. both lead to depletion, there are important differences (Table 25.19). Starvation may interfere with the metabolic response.

❗ Providing nutritional support to patients after injury will not reverse the biochemical effects observed, during the metabolic response to injury, e.g. nitrogen loss. However, it will help to ameliorate the effects of depletion and limit the clinical consequences (see 📖 this Chapter, 'Undernutrition', p. 508).

Table 25.17 Simplified model of metabolic response to injury

Metabolic and clinical effects	Ebb phase* (acute)	Flow phase† (hypermetabolic)
Energy expenditure	↓	↑
O_2 consumption	↓	↑
Cardiac output	↓	↑
Body temperature	↓	↑

Table 25.17 *(Cont'd.)*

Metabolic and clinical effects	Ebb phase* (acute)	Flow phase† (hypermetabolic)
Circulating levels of		
Glucose	↑	↑
Lactate	↑	↔
Free fatty acids	↑	↑
Catecholamines	↑	↑
Glucagon	↑	↑
Cortisol	↑	↑
Insulin	↓	Insulin resistance
Urinary nitrogen loss	↑	↑

*Occurring immediately after trauma, the ebb phase is the brief 'shock' phase (lasts ~0–8 h).

†The flow phase follows the ebb phase and is a longer 'catabolic' phase (lasts ~5–10 days).

Table 25.18 Estimated loss of protein (g) over 10-day period following trauma and untreated infection

	Tissue loss	Blood loss	Protein catabolism
Muscle wound	500–750	150–400	650
35% burn	500	150–400	600
# Femur	–	up to 200	580–860
Gastrectomy	up to 60	20–180	525–650
Typhoid fever	–	–	675

Table 25.19 Differences between the metabolic response to injury and starvation

	Injury	Starvation
Energy expenditure	↑	↓
Nitrogen losses	↑	↓
Plasma insulin and glucose	↑	↓
Plasma free fatty acids	↑ Turnover	↓Turnover
Plasma clearance of exogenous triglycerides	↑	↓

Critical care

Defined as patients requiring:
- advanced respiratory support alone *or;*
- basic respiratory support together with the support of at least two organ systems;
- includes patients with multi-organ failure.

The aims of nutrition support in critical care (CC) patients are to minimize nutritional losses and provide basic nutrient requirements to sustain life. Repletion of pre-existing undernutrition during a period of critical care is unlikely to be achieved and should not be a goal because of ↑ risk associated with overfeeding.

Route of feeding

Access to feeding routes may be limited by the patient's condition, but wherever possible, the gut should be used as first choice (see 📖 this Chapter, 'Routes for enteral feeding', p. 518).

Enteral feeding

There is evidence 👁 (Kreymann *et al.* 2006) to support the early initiation of enteral feeding in haemodynamically stable CC patients. This will require a standard initial regimen to be available to all clinical staff so that feeding can commence as soon as possible.
- Gastric aspirate should be checked every 4–6 h. Volumes <200ml with nasogastric feeding and <150 ml with gastrostomy feeding are acceptable and should be returned to the stomach.
- Gastric aspirates above these levels indicate delayed gastric emptying but a single high measurement should not lead to the cessation of feeding unless there is overt regurgitation or signs that the patient is aspirating fluid into the respiratory tract.
- Risk of aspirating feed can be reduced (see 📖 this Chapter, 'Complications of enteral feeding', p. 530).
- If absorption is limited and precludes administration of the total volume of enteral feed prescribed to meet requirements, it can be combined with parenteral nutrition. This will facilitate an adequate total nutrient intake but continue the physiological benefits of feeding via the gut.
- Immune-modulating formulae should be used with caution in patients in critical care (see 📖 'Clinically Functional Nutrients', p. 560). Read the literature related to the use of a particular supplement in a specific condition to determine if use will enhance the individual patient's recovery.

Parenteral nutrition *(see 📖 this Chapter, p. 536)*
- Parenteral nutrition can play an invaluable role where enteral feeding is not possible or not succeeding.
- Avoidance of overfeeding is important (see Box 25.9).
- Maintaining good glycaemic control (blood glucose 4.4–6.1 mmol/l) and avoiding swings in blood sugar level are associated with lower mortality. Additional insulin may be required to achieve this.

Nutritional requirements in critical care

- *Energy:* requirements can be estimated by calculation using standard formulae but ❗ resulting values provide only an approximation and may need adjusting to suit individual patients. Energy requirements are slightly ↓ if patients are ventilated rather than breathing spontaneously but will ↑ as weaning progresses. Consider the energy contribution of fluids or medicines used with the patient, e.g. patients sedated using propofol will receive additional energy from the lipid emulsion it is carried in (both 1% and 2% propofol contain between 1.06 and 1.10 kcal/ml). The maximum dose recommended for a 70kg CC patient could ∴ provide as much as 739kcal/24 h if given as 1% (or 370 kcal/24 h if given as 2%) so care must be given to ensure that adequacy of other nutrients is maintained in the remaining energy provided.
- *Protein/nitrogen:* CC patients have ↑ protein turnover and ↑ nitrogen loss is an unavoidable feature of the metabolic response to injury: nutritional support will not reduce this, but will help minimize the accompanying depletion. Evidence suggests that providing >0.2 g nitrogen/kg body weight/day has no additional benefit in septic or trauma patients.
- *Carbohydrate:* in critical illness, glucose administration should not exceed the maximum glucose oxidation rate of 4 mg/kg/min. Consider glucose contribution of medication, particularly in patients <45 kg.
- *Lipid:* in critical illness give 0.8–1.0 g/kg/day (see top bullet, this section *re* propofol). Approximately 3.0–4.5% total energy should be provided as lipid to prevent essential fatty acid deficiency.
- *Fluid, electrolytes, micronutrients:* no evidence is available about specific requirements in CC patients.

Box 25.9 Potential consequences of overfeeding

- ↓ Tolerance of feeding, e.g. diarrhoea
- ↑ Physiological stress
- Metabolic acidosis
- Uraemia
- ↑ Respiratory quotient, i.e. ↑ CO_2 produced so weaning harder
- Hyperglycaemia → impaired wound healing
- Hypercholesterolaemia & hypertriglyceridaemia
- Excess lipid → ↓ reticuloendothelial system → ± immunosuppression
- Hepatic steatosis
- Refeeding syndrome.

Further reading

Kreymann, K.G., Berger, M.M., Deutz, N.E., *et al.* (2006). ESPEN guidelines on enteral nutrition: Intensive care. *Clinical Nutrition* **25**, 210–23.

Singer, P., Berger, M.M., Van den Berghe, G., *et al.* (2009). ESPEN guidelines on parenteral nutrition: Intensive Care. *Clinical Nutrition.* **28**, 387–400.

NICE (2009). *Clinical guideline 83. Rehabilitation after critical illness.* 🖱 http//:www.nice.org.uk/CG83.

Surgery

Nutritional depletion is associated with ↑ morbidity and mortality following surgery. Surgery itself is associated with ↑ nutritional losses (see 📖 this Chapter, 'Metabolic response to injury', p. 548) and nutritional support is not able to prevent these. However, appropriate nutrition support is capable of minimizing the depletion (i.e. loss of lean body mass) accompanying major surgical intervention, and is associated with:

- repletion of lean body mass;
- improved skeletal muscle force;
- ↓ fatiguability;
- ↑ ventilatory, cardiac, and gut function;
- ↑ immunity;
- ↑ sense of well-being.

There is less evidence that nutrition support has beneficial effects in relatively well-nourished individuals who have undergone minor–moderate surgery indicating that routine nutritional support for all surgical patients is inappropriate, especially considering the potential side-effects of feeding and cost implications.

However, it is widely accepted that patients who are critically ill, severely injured, or nutritionally depleted prior to surgery will benefit from nutrition support. This raises two issues:

- Identifying patients who will benefit (see 📖 'Individual assesment', Chapter 4, p. 38).
- Optimum timing of nutrition support.

Pre-operative nutrition

Feeding undernourished patients 7–10 days prior to surgery is associated with a reduction in non-infectious complications. Shorter periods show no benefit. Obviously, delaying surgery is clinically inappropriate in some patients, but pre-operative feeding should be considered before elective surgery in those who are severely depleted. NICE guidance[1] recommends that malnourished patients who are due to undergo major abdominal surgery and have a functioning GI tract but are unable to take adequate nutrition orally, should be considered for pre-operative enteral tube feeding.

Pre-operative fasting

Overnight fasting prior to surgery is unnecessary,[2] metabolically disadvantageous and ∴ clinically detrimental. Evidence shows that allowing patients to take clear fluids until 2 h before surgery and solid food until 6 h before elective surgery is safe. Oral carbohydrate (50 g) and fluid loading prior to surgery is associated with reduced post-operative insulin resistance, improved well-being, and ↓ length of hospital stay. As a result, pre-operative carbohydrate loading is recommended treatment in the Enhanced

[1] NICE (2006). *Nutritional support in adults.* Clinical guideline 32 http://www.nice.org.uk/nicemedia/live/10978/29979/29979.pdf.

[2] Brady, M.C., Kinn, S., Stuart, P., et al (2010). Preoperative fasting for adults to reduce postoperative complications. *Cochrane Database of Systematic Reviews*. DOI: 10.1002/14651858.CD004423.

Recovery After Surgery protocol.[3] A commercial product is available to facilitate this (Preload® Vitaflo, Liverpool) or 'home made' versions can be mixed from simple ingredients at ward level.

Peri-operative nutrition

Traditionally, recommencing oral or enteral intake after surgery was determined by the audible detection of bowel sounds or passing of flatus. This is no longer considered best practice. NICE guidance[1] recommends:

- Post-caesarean or gynaecological surgical patients who can swallow safely should be provided with some oral intake within 24 h of surgery.
- Post-abdominal surgery patients who can swallow safely, and in whom there are no specific concerns about gut function or integrity, should be provided with some oral intake within 24 h of surgery. Monitor carefully for any signs of nausea or vomiting.
- General surgical patients should not have enteral tube feeding within 48 h post-surgery unless they are malnourished, have a functioning GI tract but are unable to take an adequate intake orally.

[3] Ljungqvist, O. (2009). Modulating postoperative insulin resistance by preoperative carbohydrate loading. *Best Pract. Res. Clin. Anaesthesiol.* **23**, 401–9.

Spinal cord injury

Spinal cord injury (SCI) can result in either temporary or permanent impairment of the normal motor, sensory, or autonomic function.

- *Tetraplegia:* injury to the spinal cord in the cervical region with associated loss of function in all four extremities.
- *Paraplegia:* injury in the thoracic, lumbar, or sacral segments resulting in loss of function in the lower limbs.

Short-term nutritional issues (<3 months after injury)

- Patients should undergo assessment to determine their nutritional status as soon as possible after injury. Determining body weight may be difficult; estimating requirements may be inaccurate due to variation in individual needs, other coexisting injuries, and reduced mobility.
- Nutritional support may be required if the patient is unable to eat sufficient. Adequacy of energy, protein and all micronutrient intakes should be considered as well as fibre and fluid.
- Limited evidence from animal studies suggests potential for omega-3 fatty acids to contribute to improved neurological outcome at 6 weeks post-injury. Whilst it is reasonable to provide these within a balanced intake of other nutrients, there is no evidence they will provide a 'miracle cure' in devastating injury.
- The effects of very low fat ('ketogenic') diets have been investigated but there is insufficient evidence on which to base recommendations.
- Depression, anxiety and loss of appetite as well as frequent clinical investigations and treatment may limit nutrient intake. Awareness of the importance of good nutrition is needed amongst the whole multidisciplinary care team and patients' family and friends. Input from a registered dietitian is needed to optimize nutritional care.

Longer-term nutritional issues (>3 months after injury)

- An optimum nutrient intake will help support an active rehabilitation programme. This will include the provision of adequate energy to participate in physiotherapy sessions and sufficient protein and micronutrient to facilitate any continuing healing process and minimize complications associated with limited mobility, e.g. loss of skin integrity.
- Constipation is very common and can have a serious impact on quality of life and long-term health. The degree of bowel dysfunction depends on the extent and location of the injury on the spinal cord with complete damage above the 12th thoracic vertebra (T12) associated with loss of anal muscle control. However in most patients, a high fibre diet with ↑ intake of fluid (>35 ml/kg/day) will help to regulate bowel movements and reduce risk of constipation. For those previously unfamiliar with a high fibre diet, sources should be introduced slowly over a 6-week period to optimize tolerance (see 📖 Chapter 26, 'Constipation', p. 602).
- Increasing body weight may become a concern in some people whose level of energy expenditure is curtailed by their lack of mobility. There is also evidence of reduced resting energy expenditure (↓ ~20%),

probably 2° to loss of lean body mass. Excessive weight gain may hamper rehabilitation if wasted muscles are overburdened and ↑ the chance of pressure sores (as does underweight). Increasing energy expenditure through limited activity should be encouraged where possible and dietary energy should be tailored to this. If an energy-restricted diet is required to match limited energy expenditure on a long-term basis, care must be taken to ensure that the diet is totally adequate in all other nutrients. A regular review by a dietitian may be appropriate.

- Life expectancy after SCI ranges from 70 to 92% of normal. As a consequence and because many SCI patients are young adults, the influence of nutrition on the promotion of long-term good health is important. Advice should be based on the guidance given in 'The Eatwell Plate' (see 📖 Chapter 2, p. 27) accompanied by consideration of energy balance to maintain an optimum weight.
- For individuals whose activity and socializing may be curtailed by their injury, food can provide an important pleasure and this should not be subverted because of the therapeutic effects of a healthy diet!

Further information

http://www.spinal.co.uk/userfiles/images/uploaded/pdf/279-55691.pdf.

Head injury

Patients sustaining brain damage through external injury to the head or surgery to treat a CVA (see 📖 Chapter 23, 'Stroke/cerebrovascular accident', p. 478) will require nutritional support in the short- and long-term.

Nutrition may not be a priority immediately (<24 h) after injury as resuscitation and emergency surgery may be required to preserve life.

However, in the following days, patients may become hypermetabolic and hypercatabolic as a consequence of the metabolic response to injury (see 📖 this Chapter, p. 548). Requirements should be calculated on an individual basis by a dietitian with experience in caring for the critically ill.

Energy requirements

- Resting energy expenditure may ↑ ~40–200% of normal although this increase may be moderated by pharmaceutical sedation (↓ ~12–32%).
- Energy expended through physical activity is usually minimal.
- Most head injured patients are well-nourished at the time of injury although this should not be assumed.
- Accompanying injuries, e.g. after a road traffic accident, must be considered and may ↑ energy requirements.
- Prediction equations often provide unreliable estimates of energy expenditure.
- Actual expenditure should be measured using indirect calorimetry if possible.

Protein requirements

- Protein is used as a preferred source of energy and so nitrogen losses increase in the 1st week post-injury and may remain raised for some weeks.
- Reported N losses vary from 0.29 to 0.73 g/kg body weight/day (≡ 125–300 g protein for a 70 kg man). Negative nitrogen balance results.
- Monitoring N losses will give an indication of requirements and, although feeding cannot prevent N loss, it will minimize consequences.
- Providing 0.35 g N/kg body weight is associated with better outcome than lower intakes.

Nutritional support

Systematic review[1] has shown that instigation of early nutrition support (<48 h) is associated with ↓ 0.67 relative risk of death (95% CI 0.41–1.07). It also suggests that parenteral nutritional support is associated with a more favorable outcome than enteral, but this may be confounded by the fact that parenteral feeding usually commences earlier (Box 25.10).

[1] Perel, P., Yanagawa, T., Bunn, F., *et al.* (2008). Nutrition support for head-injured patients. *Cochrane Database of System. Rev.* CD001530.pub2.

Box 25.10 Clinical practice guidelines for nutritional support in patients with head injury

- Establish nutrition support protocol for patients with head injury.
- Estimate energy expenditure using indirect calorimetry once or twice weekly or when change in clinical status.
- Assess protein status using urine urea nitrogen twice weekly (not reliable method in renal dysfunction).
- If possible, initiate nutrition support <72 h after injury.
- Preferred feeding route is enteral via jejunum.
- Administer continuous feeding using a pump to control delivery rate.
- Monitor feed tolerance using nutrition support protocol (see 'Monitoring enteral feeding', 📖 this Chapter, p. 526).

Adapted from Vizzini, A., *et al.* (2010). Nutritional support in head injury. *Nutrition*.

Burn injury

Assessment of nutritional requirements

Medical history

- History of accident (to consider whether other injuries have been sustained and/or the presence of smoke inhalation).
- Type of injury (flame, flash, contact, electrical, chemical, friction, radiation or scald).
- Total % body surface area burn (BSA) and its distribution (e.g. arms, face) together with % burn depth (superficial, partial thickness, deep dermal or full thickness).
- Previous medical history, e.g. diabetes, GI problems.

Social factors Nutritional status on admission will be affected by physical health, mental health, income, cooking skills and home circumstances.

Body weight Measured immediately on admission to calculate fluid resuscitation regimen and energy requirements.

Diet history

- Assessment of nutritional intake prior to admission.
- Food preferences/allergies.
- Special dietary requirements.
- Vegetarian ± religious beliefs.

Energy requirements

Thermal injury results in ↑ production of catecholamines producing a hypermetabolic response. This results in accelerated protein and fat breakdown and altered carbohydrate metabolism. Energy requirements are assessed using the specific formulae (see Table 25.20).

Protein requirements

- Adults—give 20% of energy as protein (1.5–2 g protein/kg).
- Children—<1 year use reference nutrient intake (RNI) for protein (see 📖 Appendix 6, p. 780); 1–3 years use 2–3 g protein/kg; ≥3 years use 1.5–2.5 g protein/kg.

Electrolytes

Large losses of electrolytes can occur via exudates and urine and as a result of diarrhoea, vomiting and pyrexia. Low levels of phosphate and magnesium are common in patients with large burn injuries. Replacement should take place orally or intravenously according to biochemical variables. Hypernatraemia can result from dehydration or high Na^+ loads administered from IV fluids with insufficient free water for excretion.

Vitamins, minerals, and trace elements

With the possible exception of vitamin C, additional vitamin and mineral supplementation is unnecessary for patients receiving full enteral feed or nutritional supplements. For children receiving mainly milk, a vitamin supplement should be prescribed. Patients with total body surface burns >20% are at risk of copper, zinc and selenium depletion due to losses through burn exudates.

Table 25.20 Estimating energy requirements in burn injury

Adults
Schofield equations used to calculate basal metabolic rate (see 📖 Appendix 4, p. 772)
Add 10–90% as stress factor per % burn and 10–25% for activity to a maximum of 2 x basal metabolic rate
Children
Galveston formulae:
0–1 years
2100 kcal x body surface area (m^2) + 1000 kcal x body surface area burned (m^2)
1–12 years
1800 kcal x body surface area (m^2) + 1300 kcal x body surface area burned (m^2)
>12 years
1500 kcal x body surface area (m^2)+ 1500 kcal x body surface area burned (m^2)

Feeding routes

Oral feeding with a high protein, high energy diet should be encouraged in well-nourished patients with minor burns (<15% BSA in adults and <10% BSA in children). Oral supplements can be used. Regular assessment of intake is needed as patients with minor burns frequently need enteral feeding.

Nasogastric or nasojejunal feeding is necessary in patients with major burns (>15% BSA in adults and >10% BSA in children). The target is to commence within 4 h of admission. If gastric stasis develops, NJ feeding is usually successful. Total parenteral feeding is rarely used due to risk of infection.

All feeding requires monitoring (Box 25.11).

Box 25.11 Monitoring

Daily
- Food and fluid intake
- Bowel activity
- Description of wound healing, skin graft take and % left to heal
- Serum urea and electrolytes
- Blood glucose (at regular intervals)
- Maximum body temperature

3 x per week
- 24-h urine collection
- C-reactive protein, liver function tests, calcium, phosphate, magnesium
- Haemoglobin, white cell count

Weekly
- Body weight
- Trace elements

Clinically functional nutrients

Functional foods have been defined as 'foods that by virtue of physiologically active food components provide health benefits beyond basic nutrition' by the International Life Sciences Institute of North America (ILSI 2008) (see 🕮 Chapter 8, 'Functional foods and nutraceuticals', p. 174). This section considers nutrients that may have some clinical benefits if consumed in larger than usual intakes in specific medical conditions.

Glutamine

Glutamine (Gln) is a conditionally indispensable amino acid:

- an important source of fuel in rapidly dividing cells, e.g. enterocytes and immune cells;
- precursor for antioxidant glutathione;
- becomes indispensable in stress situations, e.g. catabolic patients, as body pool ↓ rapidly to fuel stimulated lymphocytes, etc.;
- standard parenteral solutions do not include Gln.

Reports of benefit associated with Gln supplementation include ↓ mucosal atrophy after prolonged parenteral nutrition, ↓ bacterial translocation, and ↑ systemic immune function. Systematic reviews have reported ↓ mortality, ↓ infection and ↓ organ failure associated with enteral and parenteral Gln supplementation.[1] However, it is likely that there is publication bias towards study results which support benefit which has not been universally observed and reflect variation in Gln dose (optimum not yet defined), route of administration (oral or IV), and patient population.

Arginine

Arginine (Arg) is a conditionally indispensable amino acid:

- plays role in transport, storage, and excretion of nitrogen;
- precursor for nitric oxide;
- becomes indispensable in stress situations, e.g. trauma and sepsis, when Arg levels ↓ as it is used for nitric oxide pathways.

Arg supplementation may benefit the microcirculation and protein anabolism and has been associated with ↑ muscle and protein metabolism and intestinal motility. However, there are concerns that ↑ Arg intake in septic patients may ↑ nitric oxide production resulting in hypotension, poor perfusion, and ↑ risk of multi-organ failure. This is supported by a review of good quality studies showing ↑ mortality associated with Arg-enriched feeding (RR 1.19, 95% CI 0.99–1.43) even though fewer infectious complications were observed (RR 0.53, 95% CI 0.42–0.68).[2] A meta-analysis[3] of studies undertaken in patients with acute myocardial infarction found a non-significant 7% ↓ in mortality associated with Arg supplementation and concluded it had no clinical benefit in the population.

[1] Avenell, A. (2006). Glutamine in critical care. Current evidence from systematic reviews. *Proc. Nutr. Soc.* **65**, 236–41.
[2] Duggan, C., Gannon, J., Walker, W.A., (2002). Protective nutrients and functional foods for the gastrointestinal tract. *Am. J. Clin. Nutr.* **75**, 789–808.
[3] Sun, T., Zhou, W.B., Luo, X.P., *et al.* (2009). Oral L-arginine supplementation in acute myocardial infarction therapy: a meta-analysis of randomized controlled trials. *Clin. Cardiol.* **32**, 649–52.

Chapter 26

Nutrition in gastrointestinal diseases

Mouth disorders

Injury or disease in the mouth (including lips, oral cavity, tongue, and nasopharynx) can rapidly compromise nutritional status by inhibiting eating and drinking. To counter this, nutrient intake can be optimized through the modification of food texture or by instigating nutritional support via tube feeding.

Cancer of the mouth and pharynx

Mouth and pharyngeal cancers account for ~4% cancers worldwide but are more common in developing countries; oral cancer represents only about 2% of total malignancies in the UK. Associated risk factors include smoking and chewing tobacco, chewing betel nut, alcohol intake (risk trebles with >3 drinks/day), low vegetable and fruit intake, consumption of salted fish and exposure to sun and human papillomavirus. Treatment includes surgery and radiotherapy which should be undertaken at a specialist centre. Patients should be assessed by an experienced dietitian before treatment and offered individual dietary advice to help maximize nutrient intake and tube feeding initiated early if required (See 🕮 'Chapter 24, 'Nutritional management in cancer', p. 494).

Further reading

Mouth Cancer Foundation. Available at: www.rdoc.org.uk

NICE (2004). *Improving outcomes in head and neck cancers.* Available at: http://www.nice.org.uk/nicemedia/live/10897/28851/28851.pdf

Salivary gland disorders

Disorders include saliva deficiency, inflammation secondary to infection, and calculi. Inflammation can hinder chewing and reduce the flow of saliva, further impeding food intake. Treatment of the underlying condition is required and nutrient intake should be supported by providing moist food that requires little chewing (see 🕮 Chapter 23, 'Texture modification', p. 480). Xerostomia (dry mouth) relating to lack of saliva is also associated with Sjögren syndrome, diabetes mellitus, and taking anticholinergic, antihistamine, and decongestant medications, as well as some anticancer treatment. Artificial saliva substitutes are available on prescription as gel, spray, and tablets, and may help both food intake and promote oral hygiene. Dentures incorporating a refillable reservoir have been devised to facilitate delivery of saliva substitutes.

Jaw wiring

Fixation of the maxilla/mandible may be undertaken following a fractured jaw, oral surgery, or (very rarely) in the treatment of obesity. The procedure may accompany complex maxillofacial surgery in the presence of severe trauma or may be relatively straightforward in elective jaw wiring for obesity.

- A liquid or semi-liquid diet is required (Table 26.1). This can be based on a combination of supplemented drinks, both homemade and commercial, that can be sucked through the gaps between the jaws.
- Consideration must be given to the total nutrient intake to ensure adequacy for the duration of the fixation (usually 3–8 weeks following fracture); a higher protein intake may be required by patients who

have suffered a traumatic fracture while energy should be limited in the treatment of obesity.

- Including some soluble fibre, e.g. pureed porridge or lump-free lentil soup, may help alleviate constipation that is common due to the preclusion of most fruit, vegetable, and wholegrain items. Alternatively, the bulk-forming laxative, ispaghula husk, may be given, but care must be taken to ensure an adequate fluid intake.
- Mouth hygiene should be maintained by gently brushing the exterior tooth surfaces and fixtures and using saline or antiseptic mouthwash on waking, before retiring to bed, and after every meal and snack.

Table 26.1 Example of liquid diet

	Volume (ml)	Energy (kcal)	Protein (g)
Orange juice	250	110	2
Porridge, pureed with milk and sugar	300	200	7
Milky coffee with sugar	250	180	8
Drinking yogurt	200	190	8
Tomato juice	250	50	2
Proprietary nutrition supplement*	200	200	8
Mug of tea	300	20	—
Ice cream	100	180	4
Creamy lentil soup (lump free)	200	130	5
Proprietary nutrition supplement*	200	200	8
Pureed fruit with very thin custard	250	220	6
Banana smoothie	250	180	2
Hot chocolate with milk	250	240	10
Approximate total	3000	2100	70

❗ Vitamin and mineral supplementation may be required, depending on the duration of liquid diet.

*Volume and composition vary with brand. See Table 25.5 for examples of proprietary oral nutrition supplements.

Dental health

Healthy teeth and gums contribute to overall health and well-being.

- Efficient, pain-free mastication facilitates the intake of a varied and well balanced diet.
- Good oral hygiene is associated with a ↓ risk of cardiovascular disease and ↓ occurrence and progression of respiratory tract infections.
- Complete and decay-free teeth contribute to psychosocial well-being by enhancing facial appearance and speech.

Dental caries

Definition

Caries (cavities, tooth decay) are holes in the structure of the tooth.

Prevalence

In the UK has ↓ greatly since the 1970s when fluoride toothpaste was introduced. In 2005 (most recent data available), a 12-year-old child has on average <1 decay, missing, or filled tooth and the percentage of adults with no teeth of their own has fallen from 37% to 12% in the last 40 years. However, there are considerable inequalities between socio-economic groups with children from deprived backgrounds experiencing most poor dental health.

Pathogenesis

Bacteria living in the dental plaque ferment dietary carbohydrate into acid, which dematerializes the tooth enamel, initiating the cariogenic process (see Box 26.1). The pH of the mouth determines the extent of decay as this only takes place if <5.7.

Prevention

In addition to reducing plaque bacteria by regular brushing and flossing, and regular dental visits, dietary prevention includes:

- minimizing effects of fermentable carbohydrate;
- maximizing oral pH.

Dental erosion

Definition

Erosion is the acidic destruction of the tooth surface. It does not include the abrasion and attrition associated with normal wear and tear on the biting surface of the teeth. The phrase 'non-carious cervical tooth surface loss' (NCCTSL) is used to describe erosion, abrasion and attrition.

Prevalence

In the UK it is increasing and associated with the rise in consumption of acidic and/or carbonated soft drinks and herbal teas. Erosion exposing the palatal and occlusal dentine has been observed in 3 and 18%, respectively, of children aged 13–14 years in the UK. It was more common in boys than girls and associated with consuming fizzy drinks more than once per day (OR 1.6, 95% CI = 1.1–2.3).

Pathogenesis

The acid eroding tooth enamel is not derived from the bacterial fermentation of dietary carbohydrate (Box 26.1), but from acidic fluid in the mouth, either from dietary intake or regurgitation of stomach contents (reflux or vomiting).

Prevention

This is based on reducing acid contact. Reflux or vomiting (spontaneous or self-induced, e.g. in bulimia nervosa) require investigation. Dietary prevention includes:

- limiting acidic food and drink, including diet drinks, to mealtimes;
- finishing meal with alkaline food, e.g. cheese or milk;
- avoiding acid food or drink last thing at night;
- drinking acidic drinks through a straw and minimizing sipping, swishing, and frothing in the mouth, including fizzy water;
- beware acidic medication, e.g. chewable vitamin C tablets;
- avoiding brushing teeth immediately after acidic food, ideally wait 1 hour after meals;
- ❗ Using a baby feeder filled with fruit juice or other acidic drink as a comforter leads to prolonged contact with teeth and potentially very severe erosion.

Box 26.1 Fermentable carbohydrate

- *Type of carbohydrate* determines cariogenicity: sucrose > fructose, glucose, maltose > lactose, galactose > maltodextrins, polysaccharide > sorbitol, xylitol.
- *Frequency of exposure:* regular ingestion of small quantities of carbohydrate is more damaging to teeth than one larger intake because repeated exposure prevents oral pH from increasing above the 5.7 threshold, thus perpetuating tooth demineralization.
- *Texture of foods:* sticky/chewy food leaves residue on teeth that prolongs exposure to carbohydrate and leads to a lower oral pH. Toffees and dried fruit have a greater potential to contribute to dental caries than the same quantity of carbohydrate taken as fruit juice which rapidly leaves the mouth.

Maximizing oral pH

- Milk and dairy products are alkaline and contain protein, calcium, and phosphate, which play a role in re-mineralizing dental enamel following acid exposure.
- Saliva can be stimulated by chewing sugar-free gum for 10 min after meals. Freshly secreted saliva has a pH of >6.3, i.e. above critical threshold and ∴ protective.
- Using a drinking straw with acidic drinks (fruit juices and carbonated beverages) can reduce the fall in pH compared with drinking from a cup.

Oesophageal disorders

After chewing and swallowing, food is transported via the oesophagus (or gullet) to the stomach. Although food passes rapidly through the oesophagus compared to other parts of the gastrointestinal (GI) tract, disorders that restrict food intake can have a major detrimental influence on nutritional status (see 📖 Chapter 1, 'Digestion', p. 14).

Achalasia

Achalasia leads to food being retained in the oesophagus due to reduced peristalsis and incomplete opening of the lower oesophageal sphincter. Difficulty with swallowing food leads to weight loss in up to 60% of patients.

Treatment includes balloon dilatation, surgery, stent insertion, or injection of botulinum toxin to relax the sphincter.

Nutritional management may also help and includes small frequent meals, avoiding foods that exacerbate dyspepsia and very hot or cold foods, and not eating late at night or before lying down. Eating in an upright position (rather than reclining or slumped) may facilitate the passage of food into the stomach. Nutritional assessment should be undertaken to ensure that weight loss through an inadequate intake is prevented.

Dysphagia

Dysphagia (discomfort, difficulty, or pain when swallowing) is common in oesophageal disorders. Inflammation or occlusion of the oesophageal lumen impairs the final stage of swallow as food passes from the pharynx to the stomach by the combined effects of peristalsis and gravity. Patients with oesophageal dysphagia are less at risk from aspirating food or liquid into the respiratory tract than those with dysphagia 2° to stroke where the oral and pharyngeal stages of swallow may also be impaired.

Nutritional management should include assessment of the swallowing problem and an evaluation of the optimum texture of foods and liquids (see in 📖 Chapter 23, 'Texture modification', p. 480). In general, patients with more severe oesophageal disorders will require more liquids and thinner textures than those with milder dysphagia. The complete nutritional adequacy of the intake should be determined and progress monitored in the context of the underlying condition.

Oesophageal cancer

Approximately 7800 cases are diagnosed in the UK annually, making this the ninth most common malignancy. It is more common in men than women and in people aged >60 years. Smoking, heavy alcohol intake, being overweight, low intake of fruit and vegetable, consuming caustic substances, and a pre-cancerous condition, Barrett's oesophagus, are risk factors.

Treatment may include surgery, chemotherapy, radiotherapy (see 📖 Chapter 24, 'Chemotherapy', p. 490 and 'Radiotherapy', p. 491), laser treatment and/or the insertion of a stent (see 📖 p. 568).

Nutritional management should include assessment of nutritional status and aim to provide an adequate energy and nutrient intake in a format that can be swallowed and is acceptable to the patient (see 📖 Chapter 23, 'Texture modification', p. 480). Patients with oesophageal cancer are often undernourished on diagnosis as a result of an inadequate intake due to symptoms; depletion may be exacerbated by treatment. Appropriate nutritional support is ∴ essential and associated with better tolerance of chemo- and radiotherapy. Oral supplements may help, but tube feeding may be required, particularly if surgery is undertaken and a gastrostomy tube, possibly inserted at theatre, may provide valuable access.

Oesophageal stricture

Strictures may arise from benign causes (e.g. gastro-oesophageal reflux, damage secondary to intubation) or secondary to malignancy and results in increasing difficulty swallowing.

Treatment is aimed at the underlying cause and attempting to limit the occlusive effects of the stricture including balloon dilatation and stenting.

Nutritional management should ensure an adequate intake. Small, frequent meals comprising moist, semi-solid food and nourishing liquids may be tolerated but, if not, tube feeding should be instigated before severe depletion or dehydration occurs.

Oesophageal varices

See 📖 Chapter 28, p. 619.

Oesophagitis

Inflammation of the oesophagus is associated with acid reflux from the stomach and hiatus hernia. Prolonged oesophagitis can lead to thickening and hardening of the mucosal cells, known as Barrett's oesophagus, a pre-cancerous risk for developing oesophageal cancer.

Nutritional management See 📖 this Chapter 'Indigestion, heartburn, gastro-oesophageal reflux disease (GORD), and hiatus hernia', p. 570).

Stents

The endoscopic insertion of a self-expanding metal stent (SEMS) into a strictured oesophagus may prevent total occlusion and help the patient to maintain their oral intake. Stents are mainly used as palliation in oesophageal cancer, but can also play a role in the management of benign strictures and oesophageal fistulae. Complications include haemorrhage, migration, tumour overgrowth, and food-related blockages. SEMS are recommended in preference to plastic as they are associated with fewer blockages.

Nutritional management should include dietary advice about maintaining an adequate energy and nutrient intake and how to minimize the risk of tube blockage (see Box 26.2). Even though this treatment is seen primarily as palliative, 75–90% of patients resume a near-normal diet after stent insertion and improvements in nutritional status and survival have been reported.

Box 26.2 Dietary advice after oesophageal stent placement*

Fluids

- Prescribe a fluid-only diet for first 24 h after insertion
- Once food is introduced, advise frequent consumption of any type of liquid after eating food in order to wash away any debris
- There is no evidence to support the use of fizzy drinks. These may cause problems with acid reflux if stent is placed distally
- If the stent becomes blocked following eating, drink warm water to flush through

Food

- Advice given should be modified to take account of the patient's tumour (in oesophageal cancer), their ability to chew, continuing dysphagia, and posture/position
- Texture modification should reflect individual patient needs (see Chapter 23, 'Texture modification', p. 480) and appropriate written advice should be given
- If no texture modification is required, patients should be advised to:
  - take small mouthfuls and chew all food well
  - sit upright when eating
  - eat slowly and without rushing
  - drink plenty of fluid
- Patients are often advised to restrict foods considered potentially stent-blocking. However, experimental evidence suggests that few items need to be totally avoided.
  - *Foods causing occlusion:* dry meat, fruit with pith, skins of capsicum peppers and tomatoes, >7 sultanas, dried apricots
  - *Foods able to pass through stent if taken in small mouthfuls and chewed for twice the usual time:* sandwiches, dry toast, apple, tinned pineapple, fresh orange segments with pith removed, ≤6 sultanas, chopped dried apricots, boiled egg, muesli, meat, and poultry
  - Controversial items like nuts and vegetables including lettuce caused no occlusions.

Nutritional support

Most patients continue to need nutritional support and their nutritional status should be regularly assessed.

* Adapted from British Dietetic Association (2003). *Dietetic advice post oesophageal stent placement.* BDA, Birmingham; Holdoway, A. Stacey, B. and Davis, M. (2003). Palliative management of cancer of the oesophagus—opportunities for dietetic intervention. *J. Hum. Nutr. Dietet.* **16**, 369.

Stomach disorders

Nausea and vomiting

Nausea and vomiting can have a significant effect on nutritional status by greatly reducing intake or preventing the digestion and absorption of food consumed. These symptoms may relate to a GI disorder, food poisoning, or other systemic condition, e.g. uraemia, or treatment, e.g. chemotherapy (see 🕮 Chapter 24, 'Chemotherapy', p. 490). Treatment of the underlying cause or self-limitation may bring resolution but, in many cases, managing the situation may help maintain an adequate nutritional intake. For vomiting in children <5 years, see NICE Clinical guideline 84.[1]

Nutritional management in adults

Try:

- chilled foods as these may be more acceptable than hot items;
- plain foods in small quantities may be better tolerated;
- sip drinks throughout day, but wait for 15 min after eating before taking more fluid;
- ginger flavours, mints, and plain biscuits.

Avoid:

- off-putting smells (food or others);
- foods that don't appeal: may include spicy or greasy items;
- lying down after eating: a gentle walk may help;
- extreme hunger by eating small amounts regularly.

In severe cases, dehydration may be a concern and oral rehydration solution or intravenous fluids may be required.

Indigestion, heartburn, gastro-oesophageal reflux disease (GORD), and hiatus hernia

This spectrum of gastric disorders is common with an estimated 20–25% of adults in the UK and USA experiencing symptoms of GORD although most do not require investigation.[2] Although diet has been implicated in the aetiology (erratic eating habits, obesity, alcohol, and other specific food items), there is little firm evidence to confirm this.

Symptoms range from post-prandial discomfort to sharp burning pain below the sternum or between the shoulder blades, regurgitation of acidic stomach contents into oesophagus and possibly mouth and, in severe cases, mucosal damage.

Nutritional management (in addition to proton pump inhibitors and H_2 blockers) should include review of diet and lifestyle with the aim of reducing excess body weight and introducing a regular eating pattern based on the 'Eatwell Plate' (see 🕮 Chapter 2, p. 27). Specific dietary

[1] 🖱 http://www.nice.org.uk/nicemedia/live/11846/47350/47350.pdf

[2] Banks, M. (2009). The modern investigation and management of gastro-oesophageal reflux syndrome (GORD). *Clin. Med.* **9,** 600–4.

advice is mostly anecdotal and not supported by systematic review[3] of limited studies. Practical suggestions that may help include:

- elevating the head-end of the bed to facilitate a semi-upright position while sleeping;
- eating small, regular meals in place of less frequent, but larger meals;
- eating earlier in the evening and avoid late night meals;
- sitting upright, rather than slumped while eating and avoid bending, lifting, or lying down immediately after meals;
- avoiding foods that are known to cause discomfort to individual.

Gastritis and peptic ulcers

Gastritis is the inflammation of mucosal surface of the stomach. It can range from a mild, asymptomatic form to severe ulceration, which if untreated may lead to perforation. Peptic ulcers include lesions in the stomach and duodenum. 80% of gastritis and peptic ulcers are associated with *Helicobacter pylori* infection (see Box 26.3), but a high intake of alcohol and non-steroidal anti-inflammatory drugs is also implicated.

Symptoms include nausea, vomiting (possibly blood-stained), and pain.

Nutritional management In severe cases, patients have no desire to eat and 'resting' the stomach from food for 1–2 days may help alleviate pain; adequate fluid including sugar and electrolytes will minimize risk of dehydration, but clearly are not nutritionally adequate. Nutrient intake should be gradually ↑ over 1–3 days by providing other nourishing fluids and then bland, non-irritating foods that the patient feels able to manage. There is little clinical evidence about specific foods to avoid, but most individuals are aware of items that exacerbate symptoms (often spicy, highly flavoured foods with a high fat content) and thus should decide whether or not to risk eating them (see Box 26.4). Fruit and juice with perceptible acidity have traditionally been avoided on the grounds that these exacerbate gastric pH; there is no evidence for this and the antioxidants provided by these food items play a valuable role in promoting healing so they should not be avoided. Some people may find plain, bland food is tolerated best and that milk and milky foods are most agreeable; again, there is no evidence to support this and patients should be encouraged to eat a wide range of foods. Ultimately, a varied and well-balanced diet that sustains a healthy weight should be the goal.

[3] Kaltenbach, T., Crockett, S., and Gerson, L.B. (2006). Are lifestyle measures effective in patients with gastroesophageal reflux disease ? An evidence-based approach. *Arch. Intern. Med.* **166,** 965–71.

Box 26.3 *Helicobacter pylori*—are there any nutritional implications?

H. pylori is a bacterium commonly found in the stomach. Infection may be asymptomatic, but is associated with gastritis, ulceration and ↑ risk of stomach cancer. Prevalence of infection ↑ with age and is highest in developing countries and people of lower socio-economic status. In the UK, ~30% of people born in 1930s are infected compared with <5% born in 1970s.

- Acquisition of infection is by person-to-person transmission (oral–oral and faecal–oral) and possibly also via food. Overcrowding and poor hygiene practices are implicated. Good personal and food hygiene may ↓ risk of transmission
- Oral probiotics may act as a beneficial adjunct to antibiotic eradication therapy. Further studies are needed to clarify optimum dose and population
- A high salt diet and *H. pylori* infection may act synergistically to promote atrophic gastritis.[1] This effect may be partly attenuated by dietary antioxidants, i.e. from fruit and vegetables, and these cellular mechanisms are supported by epidemiological findings
- Although there is no evidence from dietary trials, it would be logical to recommend ↑ fruit and vegetable intake and avoid excessive dietary salt, i.e. a healthy diet, for people with *H. pylori* infection.

[1] Izzotti, A., Durando, P., Ansaldi, F., *et al.* (2009). Interaction between *Helicobacter pylori*, diet, and genetic polymorphisms as related to non-cancer diseases. *Mutat. Res.* **667,** 142–57.

Box 26.4 Is it necessary for people with stomach disorders to avoid spicy food?

- Studies investigating the clinical effects of eating spicy food have provided results that vary with the type and quantity of spice eaten and the habitual intake and GI health of consumers:
  - Curcumin, extract of turmeric, appears to both inhibit and promote DNA mutation in mucosal cells *in vitro*
  - Capsaicin, extract of chilli, can aggravate symptoms in some people with dyspepsia and irritable bowel syndrome, but regular ingestion may ↓ symptoms in GORD and is associated with ↓ incidence of gastric ulcers in epidemiological studies
  - Spice, garam masala, is associated with more rapid gastric emptying in humans
  - Spice extracts (including clove, ginger, and nutmeg) inhibit *Helicobacter pylori* growth *in vitro*, possibly explaining low rates of gastric disease in countries like Thailand
- *Conclusion:* inadequate evidence for advice—more research required.

Stomach cancer

Approximately 8000 cases are diagnosed in the UK annually, making this the 8th most common malignancy in men and 13th most common in women. It is more common in individuals with *Helicobacter pylori* infection and those eating a high intake of smoked, cured, and salted food. A diet high in fruit and vegetables is protective.

Symptoms include heartburn, anorexia and bloating progressing to vomiting (sometimes blood stained), pain on eating and severe weight loss.

Treatment is surgical resection (see 🕮 this Chapter, 'Gastrectomy and stomach surgery', p. 574) if the tumour is operable. Chemotherapy and radiotherapy may be used in conjunction or as alternatives (see 🕮 Chapter 24, 'Chemotherapy', p. 490 and 'Radiotherapy', p. 491).

Nutritional management depends on treatment, but should aim to maintain an optimum nutritional intake whether by mouth or through artificial nutrition support. Each patient should be individually assessed and their requirements evaluated and nutrition support planned on the basis of these and the access available for feeding. Specific advice is required after surgical resection.

Gastrectomy and stomach surgery

The type of surgical resection of the stomach, e.g. for cancer, perforation following severe ulceration or traumatic injury, varies depending on the degree and position of the lesion to be removed, but can be briefly summarized as follows (see Fig. 26.1):

- *Total gastrectomy:* resection of complete stomach with anastomosis of oesophagus to the small bowel and reconnection of the duodenum to the small bowel (Roux-en-Y reconstruction). Cardiac and pyloric sphincters removed.
- *Partial gastrectomy:* resection of distal (pyloric) end of stomach by anastomosis of remaining part of upper stomach to duodenum or, more commonly, small bowel (Biliroth 2 reconnection). Pyloric sphincter removed.
- *Oesophago-gastrectomy:* resection of proximal (cardiac) end of stomach and lower oesophagus by anastomosis of lower stomach to upper oesophagus. Cardiac sphincter removed.
- *Vagotomy:* cutting the vagus nerve, to reduce acid secretion also causes a decrease in peristalsis and alters the emptying patterns of the stomach. It is often undertaken with gastrectomy or a pyloroplasty, a procedure to widen the outlet from the stomach to the small intestine.

The type of surgery, anastomosis, and removal of sphincter muscles have nutritional relevance because they influence eating-related symptoms after surgery. Meta-analysis[1] of studies of total gastrectomy indicates that surgical formation of a pouch is nutritionally and symptomatically better than reconstruction without a pouch and is associated with similar morbidity and mortality.

Nutritional management

Recommencing oral intake, usually in the form of clear fluids, should be undertaken as soon as possible after surgery. Gradually increase from liquids to solid food so that in most cases some solid food is being taken 1 week post-operatively (although this will vary with surgical procedure and reconstruction). Food-related complications include the following:

- Feeling full after very small quantities of food is common, particularly following total gastrectomy, so very small meals eaten frequently (~ hourly, initially) will help to maximize nutrient intake and thus contribute to healing. Bulky foods and fizzy drinks may be best avoided at first as these may exacerbate feelings of fullness. Drinking separately from eating may also help.
- Dumping syndrome is caused by the rapid movement of dietary sugar/refined carbohydrate into the intestine. Early post-prandial symptoms include dizziness, faintness, sweating, and a sudden drop in blood pressure. Later symptoms can occur ~2 h after eating including weakness, cold, and faintness associated with hypoglycaemia resulting from excessive release of insulin in response to rapidly absorbed dietary carbohydrate. Both early and late symptoms can be controlled

[1] Gertler, R., Rosenberg, R., Feith, M., *et al.* (2009). Pouch *vs* no pouch following total gastrectomy: meta-analysis and systematic review. *Am. J. Gastroenterol.* **104,** 2838–51.

by eating small meals regularly, limiting refined carbohydrate, including small quantities of high fibre foods if tolerated, and drinking liquids separately from meals. The intensity of symptoms may resolve within 3 months of surgery.

- Diarrhoea is relatively common in the first 1–2 months after gastric surgery. Dietary modification is not required, but anti-motility medication, e.g. codeine phosphate or loperamide hydrochloride, may help.
- Vomiting of bile and other digestive juices may occur after partial gastrectomy, particularly in the morning. No dietary modification is required; antacids or motility stimulants, e.g. domperidone or metoclopramide, may help but some patients require reconstructive surgery to alleviate the problem.
- Indigestion may be relieved by peppermint oil. Foods that exacerbate should be avoided.
- Post-operative weight loss occurs in most patients and an average loss of 16 kg has been reported after total gastrectomy for malignancy; loss of >10% of body weight is associated with ↑ risk of complications and death.[2] Nutritional support is ∴ important.

Supplementation of nutrients

Not routinely required by all patients, but should be determined on an individual basis depending on the patient's underlying disorder, extent of surgery, and oral intake.

- *Energy and macronutrients:* weight loss may indicate an inadequate intake or a recurrence of malignant disease; intake should be assessed and, if necessary, supplemented.
- *Vitamin B_{12}:* prophylactic vitamin B_{12} supplementation by intramuscular injection is mandatory following total gastrectomy due to loss of stomach-derived intrinsic factor required for absorption. In patients with partial gastrectomy, vitamin B_{12} absorption test should be checked to identify requirement.
- *Iron and folate:* regular blood tests are required to identify anaemia and iron and\or folate supplemented as necessary.
- *Calcium and vitamin D:* bone disease is common after gastrectomy and supplementation may help prevent this.

[2] Ryan, A.M., Healy, L.A., Power, D.G., *et al.* (2007). Short-term nutritional implications of total gastrectomy for malignancy and the impact of parenteral nutrition support. *Clin. Nutr.* **26,** 718–27.

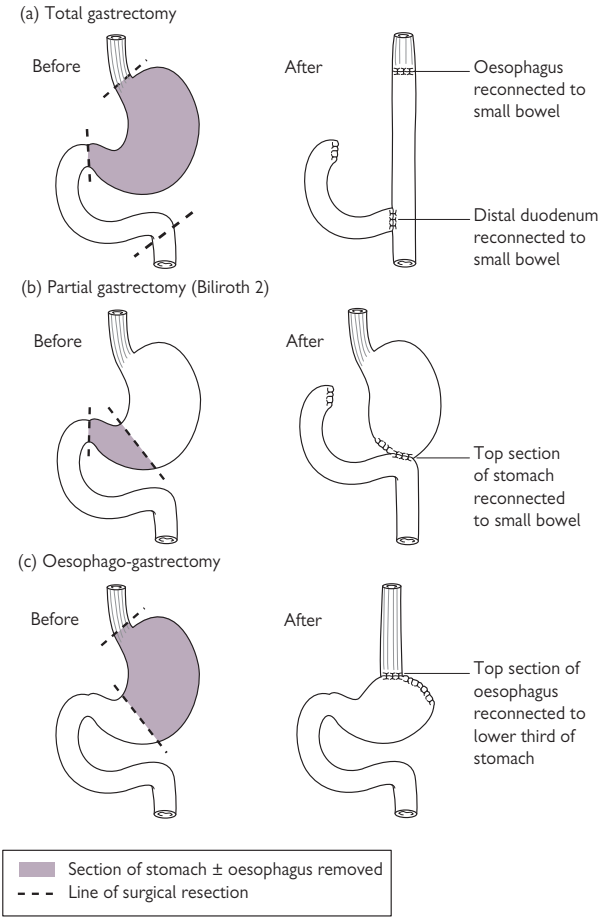

Fig. 26.1 Gastrectomy and stomach surgery.

Small intestine disorders: introduction

The small intestine comprises ~7 m of the GI tract running from the pyloric sphincter of the stomach to the ileo-caecal valve and comprises:

- duodenum;
- jejunum;
- ileum.

Its main function is to digest and absorb energy, nutrients, and water from the partially digested food passing through its lumen. As a consequence, any disorders of the small intestine that result in impaired function will potentially have a significant influence on absorption and ∴ on nutritional status.

Disorders with nutritional implications include:

- malabsorption (including steatorrhoea and lactose intolerance);
- inflammatory bowel disease (Crohn's disease and ulcerative colitis);
- coeliac disease;
- intestinal failure and short bowel syndrome;
- fistulae.

Malabsorption: introduction

Symptoms

Diarrhoea, abdominal distension, flatulence due to intestinal gas production, weight loss.

Aetiology

Multifactorial, (see Box 26.5).

Treatment

The underlying cause of malabsorption should be treated wherever possible, e.g. treating infections, prescribing pancreatic lipase in insufficiency, avoiding gluten in coeliac disease. Dietary manipulation may also be required.

Nutritional management

- Individual patients should be assessed and advised by a dietitian with expertise in treating patients with malabsorption.
- Consideration must be given to the cause of malabsorption in order to identify the specific section of the small intestine that is affected and thus which nutrients are likely to be inadequately absorbed, e.g. dissaccharides are absorbed in the proximal jejunum, vitamin B_{12} in the ileum (see 📖 Chapter 1, 'Digestion', p. 14).
- The inadequate absorption of some specific nutrients, e.g. fat and lactose, will result in generalized malabsorption of most other nutrients because of the effects of interaction with other unabsorbed components and\or bacterial action.
- In addition to dietary manipulation which may resolve symptoms, consideration must be given to overall nutritional adequacy and, in patients who have become nutritionally depleted by malabsorption, restoration of nutritional status.

Box 26.5 Main causes of malabsorption

Anatomical
- Surgical resection
- Fistulae

Luminal factors
- Altered pH, e.g. Zollinger–Ellison syndrome
- Bile salt insufficiency

Enzyme insufficiency
- Pancreatic insufficiency, e.g. lipase
- Lactase deficiency, 1° or 2°

Mucosal insufficiency
- Villous atrophy
- Coeliac disease
- Crohn's disease
- Radiation enteritis
- Impaired transport
- Lymphangiectasia

Infection
- Bacterial overgrowth, e.g. in blind loops
- Parasitic infections

Systematic conditions
- Scleroderma
- Lymphoma

Drugs
- Antibiotics
- Excessive laxative use.

Steatorrhoea

Untreated fat malabsorption is potentially very serious because undigested fat forms complexes within the GI lumen with calcium and other minerals preventing them and a wide range of other nutrients from being absorbed.

If steatorrhoea arises from pancreatic lipase insufficiency, this should be treated with pancreatic enzymes, rather than dietary fat restriction (see Chapter 27, 'Pancreatic enzyme replacement therapy', p. 616). However, steatorrhoea due to bile insufficiency cannot be so readily treated and a low fat diet may be required.

Low fat diet

The amount of fat tolerated varies between individuals and it is recommended that a very low fat diet of ~20 g/day (Table 26.2) is instigated temporarily (days only) until symptoms resolve and then small amounts of additional dietary fat are added to the diet to tolerance. Most patients with malabsorption can tolerate a diet providing ± 40 g fat and there is no benefit from advising a lower level of restriction than one that ameliorates symptoms. Attention must be given to the following:

- Total energy content of the diet. 40 g fat provides <20% of the energy requirements of most adults (in a healthy diet, fat provides 30–35% of energy) so the deficit must be made up by ↑ carbohydrate ± protein intake or by supplementing with medium chain triglycerides (see Box 26.6).
- Fat-soluble vitamins, A, D, E, and K. If absorption is in doubt, vitamin status should be assessed. Supplements can be given orally or by intramuscular injection depending on the degree of malabsorption. Dose of A, D, E and K supplementation in patients with cystic fibrosis is given in Table 30.1. Studies are required to identify optimum dose in other patients with steatorrhoea.
- Calcium. Supplements, e.g. 1600 mg/40 mmol daily, should be given if steatorrhoea is prolonged or there is evidence of bone thinning.
- Essential fatty acids. The limited dietary fat consumed should include some linolenic and linoleic fatty acids.

Table 26.2 Sample menu for temporary very low fat diet (20 g). Fat intake should be ↑ to tolerance according to individual needs—most patients will tolerate ~40 g/day*

	Weight (g)	Energy (kcal)	Fat (g)
Orange juice	250	110	—
Cereal with sugar	55	210	0.5
Skimmed milk for whole day	600	210	0.6
Toast, 2 slices	75	180	1.7
Marmalade	20	50	—
Coffee with skimmed milk + sugar	250	40	—
Low fat yoghurt	150	170	1.7
Dates	40	120	0.2
Sandwich, 4 slices bread	140	330	2.2
½ tsp polyunsaturated margarine†	3	20	2.4
Lean ham	80	80	2.2
Sliced tomatoes	60	10	—
Banana	110	90	0.4
Tea with skimmed milk + sugar	250	40	—
Toasted tea cake with honey	45	120	0.9
Roast chicken, no skin	120	120	3.7
Boiled potatoes, large serving	200	170	0.2
Carrots and peas	200	60	0.4
Thin gravy	80	—	—
Tinned fruit in syrup	100	70	—
Low fat custard	75	70	1.1
Low-fat chocolate drink	250	40	1.4
Marshmallow or jelly sweets	30	90	—
Approximate total	—	2400	19.6

*Supplementary fat-soluble vitamins ± calcium supplements should be prescribed.

† Source of essential fatty acids.

Box 26.6 Medium chain triglycerides

- Medium chain triglycerides (MCT) comprise fatty acids with 6–12 carbon atoms (long chain triglycerides (LCT) have fatty acids with >12 carbons)
- Partially water-mixable so more easily emulsified than LCT—useful in bile insufficiency
- More easily hydrolysed than LCT—useful in lipase insufficiency
- Absorbed directly into portal circulation—chylomicron transport via lymph not required
- Provide 855 kcal/100 ml (approximately 8.4 kcal/g compared with 9.0 kcal/g with LCT)—a useful source of energy if ordinary fat cannot be taken
- Available as:
  - oil (MCT oil module, SHS)
  - emulsion (Liquigen, SHS)
  - powder, with carbohydrate (MCT Duocal, SHS)
  - complete or partially complete feeds (Nutrison MCT, Nutricia Clinical; Peptimen HN, Nestlé; Perative, Abbott)
- Indications include steatorrhoea, lymphangiectasia, and ketogenic diets for epilepsy
- MCT should be introduced into the diet gradually (<15 ml oil per dose) to avoid diarrhoea
- In cooking, the oil has a low flash point so very high temperatures can give food a burnt taste
- Emulsion can be added to low fat milkshakes to increase energy.

Lactose intolerance

Insufficiency of lactase is the most common cause of carbohydrate-related malabsorption (sucrase and maltase deficiency is very rare except in Greenland). The term 'lactose intolerance' is used to describe the symptoms associated with this form of malabsorption that depend on (1) the quantity of lactose ingested and (2) the degree of lactase deficiency. This can be categorized as:

- 1° due to autosomal recessive disorder where lactase production is ± normal in children <4 years, but declines in older children and adults leading to lactose intolerance. Prevalence varies with ethnicity: African-Caribbean >80%, Indian >50%, White Europeans <10%. 1° lactose intolerance may also occur at birth (but is rare) due to a hereditary total lactase deficiency.
- 2° due to ↓ lactase production as a result of intestinal villi damage typically after GI infection. This is usually temporary (~weeks) and lactase production may slowly resume spontaneously as damage resolves.

Nutritional management

This is based on a low lactose diet (Table 26.3), which is relatively straightforward in adults and older children providing that the rest of the diet includes sufficient variety to meet all nutrient requirements including calcium. However, more expertise is required to plan a regime for infants and younger children, because of the important nutritional role milk usually plays, to ensure that their intake is sufficiently low in lactose and yet remains otherwise nutritionally adequate.

Table 26.3 Foods that are usually lactose free and those that contain lactose

Foods usually free from lactose	Foods containing lactose*
Soya milks	Milk: skimmed, semi-skimmed, and whole (cow's, goat's, sheep's)
Rice milk drinks	Cheese spread, cream, and cottage cheese
Non-dairy creamers (check label)	Cream and sour cream
Most hard cheese contains very little lactose so is well tolerated, e.g. Cheddar, Brie, Edam	Evaporated and condensed milk
	Yogurt
Breads made without milk	Breakfast cereal with milk
Breakfast cereals made without milk	Instant mashed potato mixes
Pasta, noodles, macaroni	Prepared breads, muffins, biscuits, or rolls made with milk
Potatoes, rice, other cooked grains	Pancakes or batter made with milk
Rice cakes	

(*Continued*)

Table 26.3 *(Cont'd.)*

Foods usually free from lactose	Foods containing lactose*
Margarine without whey (check label)	Butter
Non-dairy creamers (check label)	Margarines with butter or milk
Oils	
Some salad dressings (check label)	
All fresh fruits and vegetables	Creamed vegetables, e.g. mashed potato
Cooked fruit or vegetables made without milk products	Fruit smoothies made with yogurt
Fruit & vegetable juices	Fruits or vegetables cooked with milk
	Vegetables coated in batter
All fresh cooked, plain meat, and fish	Breaded or battered meat or fish
Cooked dried peas and beans	Main dishes with white sauce such as macaroni cheese, fish in parsley sauce
Eggs cooked without milk	Meats in cream sauces
Peanut butter, nuts, and seeds	Omelette or soufflés with milk
Soya cheese & tofu products	
Broth, bouillon, consommé	Cream soups
Vegetable or meat soups without milk	Soup mixes with milk products
Gravies made with water	White sauces and gravies
Plain herbs and spices	
Fruit ices and sorbets	Custard or sauce made from milk
Honey, sugar, syrups, molasses, and powdered sweeteners	Cream or cheese filled cake or pastries
Jellies, jams, preserves	Fudge, coated candies, and chocolates
Pies and other baked foods without milk	Ice cream unless lactose-free
	Toffee, butterscotch, or caramels

* Many people with lactose intolerance can eat some of these foods and a very strict, lactose-free diet is required by very few individuals. It is recommended that very intolerant individuals and carers of small children check labels of food products and proceed with caution if the following ingredients are listed: milk powder, milk protein, milk solids, non-fat milk solids, whey, whey solids or protein.

Diet for lactose intolerance

Individuals vary in the amount of lactose they can tolerate without experiencing symptoms of malabsorption (diarrhoea, bloating, and discomfort). There is no benefit in avoiding more lactose than is necessary to control symptoms. Systematic review[1] has indicated that 12 g of lactose as a single dose of milk (i.e. ~240 ml) is tolerated by most people with lactose intolerance (15 g if consumed with other nutrients) whilst doses of 24 g are associated with substantial symptoms.

There is insufficient evidence[1] to support adaptation of the colon to small doses of lactose or of benefit from lactose-reduced milk, lactase supplements taken with milk, probiotics, or treatment with rifaximin.

Dietary calcium intake may be compromised in individuals avoiding milk and other dairy products. This is particularly a concern for children, teenagers, pregnant women, and those with a family history of osteoporosis. Good sources of non-milk calcium include: oily fish, e.g. sardines, white or brown bread, calcium-fortified soya drinks (see 📖 Chapter 6, 'Calcium', p. 122 and discussion of calcium in 📖 Chapter 16, 'Vegetarians', p. 315).

Infants and children <5 years with confirmed lactose intolerance should be given an appropriate lactose-free milk substitute (e.g. Enfamil O-Lac, Mead Johnson; Galactomin 17, SHS; SMA LF, SMA Nutrition) under the advice of a registered dietitian.

❗ A diet for lactose intolerance is not the same as a milk-free diet which is required for cow's milk allergy (see 📖 Chapter 37, 'Food hypersensitivity', p. 730).

[1] Shaukat, A., Levitt, M.D., Taylor, B.C., *et al.* (2010) Systematic review: Effective management strategies for lactose intolerance. *Ann. Intern. Med.* **152,** 797–803.

Inflammatory bowel disease

Inflammatory bowel disease (IBD) includes two major disorders, Crohn's disease (CD) and ulcerative colitis (UC), which both involve chronic inflammation of the GI tract, sometimes with acute episodes. IBD should not be confused with irritable bowel syndrome (IBS).

Crohn's disease

Prevalence

Approximately 1 in 1000 people in the UK have CD. It is less common in Africa, Asian, and Central and South America. All ages can be affected but diagnosis is most frequent in children and young adults.

Aetiology

The condition is related to an immune response to GI tract bacteria. Smoking may increase risk, but dietary origins have not been substantiated.

Symptoms

Inflammation can affect any part of the GI tract, but is most common in the ilio-caecal region of the small intestine and colon. Abdominal pain and diarrhoea (with mucus and blood) feature. Recurrent episodes of inflammation lead to deep ulceration, strictures, and fistulae; surgical resection may be required, sometimes repeatedly. Patients often lose weight and feel very unwell.

Nutritional management

Assessment

- A registered dietitian with IBD expertise should undertake a nutritional assessment and provide individual advice.
- Weight (and height in children and adolescents) should be monitored and documented at all outpatient appointments and weekly during admissions.
- Vitamin B_{12} status should be measured annually in patients with ileal CD.

Preventing/treating undernutrition

- Patients with CD are often undernourished (up to 85%) as a result of inadequate intake and ↑ losses through malabsorption. Undernutrition includes energy and protein depletion and, in some patients, specific micronutrient deficiencies. Loss of >5% body weight at diagnosis is independently associated with development of severe disease.
- Providing nutritional support is a high priority and should be an integral part of management. The form/degree of support will depend on current severity, i.e. in exacerbation or remission, and part of GI tract affected.
- Those most likely to require nutritional support include children and adolescents (who are still growing), patients with existing undernutrition, and those with an exacerbation of symptoms, partial obstruction and requiring or recovering from surgery.
- Although patients may avoid specific foods, wherever possible restricted items should be minimized and a 'normal' diet encouraged including energy- and protein-dense foods, i.e. there is no proven specific diet for CD.

- Patients with strictures or partial obstruction should avoid fibrous foods that may cause pain and/or lead to an obstruction, e.g. stringy beans, citrus fruit pith, meat gristle, nuts.
- In proven fat malabsorption, limiting dietary fat (~40 g/day or to tolerance) may help. However, this should *not* be routinely advised because of the concentrated energy provided by dietary fat and its potential therapeutic effects mediated via rate of apoptosis in inflammatory cells.
- The anti-inflammatory effects of *n*-3 fatty acids (i.e. from oily fish, see Table 23.3) on remission in CD have been investigated. Studies to date have provided inconsistent results and there is insufficient evidence to support recommending omega-3 supplementation.
- Oral nutritional supplements should be offered if insufficient food is consumed (see 📖 Chapter 25, 'Treatment of undernutrition', p. 512). Most commercial products (except powders that are mixed with milk) are low in lactose so acceptable even if 2° lactose intolerance is suspected (see 📖 this Chapter, 'Lactose intolerance', p. 584).
- Enteral feeding is the preferred route if oral intake is inadequate and should be considered early before nutritional depletion is allowed to progress.
- Total parenteral nutrition (see 📖 Chapter 25, 'Parenteral nutrition', p. 536 is an appropriate adjunctive therapy in patients with fistulae if an adequate nutritional intake cannot be achieved enterally.
- Standard micronutrient supplementation should be given if oral intake is inadequate or absorption compromised.
- Calcium and vitamin D supplementation should be considered in all patients receiving corticosteroids.
- There is insufficient advice to recommend probiotics as beneficial in maintaining remission in CD.

Nutritional versus other treatment

- In adults, nutritional intervention is adjunctive rather than primary treatment, i.e. to provide nutrition support and prevent or ameliorate malnutrition. In children and adolescents, specific consideration must be given to increased nutritional requirements associated with growth.
- Elemental (amino acid based) or polymeric (containing whole protein) enteral feeds are equally effective as each other, but less effective than corticosteroids in the treatment of active CD in adults. They may be used in patients with active CD who prefer not to use this medication. Elemental enteral feeds are less palatable and more expensive than polymeric and consideration should be given to compliance if patients are advised to consume these over a long period.

Further reading

Dignass, Van Assche, G., Lindsay, J.O., *et al.* (2010). The second European evidence-based consensus on the diagnosis and management of Crohn's disease: Current management. *J. Crohn's Colitis* **4,** 28–62.

Ulcerative colitis

Prevalence

Approximately 1 in 600 people in the UK have UC. It is more common in people of white European descent and less common in Asians. Men and

women are equally affected and diagnosis is most frequent between the ages of 15 and 30 years although it can appear at any age.

Aetiology

The cause has not yet been definitively identified, but dietary causes are unlikely. A genetic component and altered response to colonic microflora have been implicated, but it is likely that triggers vary between individuals.

Symptoms

Inflammation affects the rectum and may extend continuously towards the caecum. The small intestine is not involved. Abdominal pain, diarrhoea (with blood and mucus), and anaemia feature in a chronic, relapsing pattern. Acute episodes may occur and complications include strictures, perforation and toxic megacolon. Surgical resection may be required leading to the formation of colostomy or ileo-rectal pouch.

Nutritional management

Severe nutritional depletion is usually less of a problem in adults with UC compared with those with CD, probably as a result of inflammation affecting areas of the gut distal to the site of absorption of most nutrients. However, UC patients frequently have a poor appetite or consume a restricted diet in an effort to relieve their symptoms and poor nutritional status is common. Nutritional management should ∴ focus on the adequacy of patients' intake and their nutritional status.

- No dietary restrictions should be routinely advised (there is no evidence to support the use of low residue diets).
- Patients should be encouraged to eat as normally as possible.
- If they are unable to consume an adequate intake orally, enteral tube feeding should be considered. This is preferable to parenteral as it is associated with significantly fewer complications in UC. Using parenteral nutrition to 'rest the bowel' does not alter clinical outcome.
- There is no evidence that elemental or other dietary interventions have a specific therapeutic impact on the condition although maintaining nutritional status is important, particularly if surgery is anticipated.
- Patients undergoing colectomy may benefit from specific advice about their food intake after surgery.
- Many patients with UC have iron deficiency anaemia and may require supplementation.
- Calcium intake, vitamin D status and bone density should be monitored in those receiving corticosteroids for more severe disease and supplements may be required.
- Patients with UC have ↑ risk of colon cancer and, in order to minimize this, those in remission should be advised to eat a healthy diet along the lines of the 'Eatwell Plate' (see 📖 Chapter 2, p. 27).

Further reading

Travis, S.P.L., Stange, E.F., Lémann, M., *et al.* (2008). European evidence-based consensus on the management of ulcerative colitis: Current management. *J. Crohn's Colitis* **2**, 24–62.

Coeliac disease

Coeliac disease (CoD) is an autoimmune condition resulting in mucosal damage of the duodenum. It is caused by the ingestion of 'gluten', a generic term for specific proteins (prolamines) found in the endosperm of certain cereals including wheat (gliadins), rye (secalins) and barley (horedins). Up to 1% of the UK population is affected by CoD, although delayed and late diagnosis is common.

Diagnosis

This is a two-stage process: first, a serological test measuring total IgA level as well as either IgA tissue transglutaminase (tTGA) or IgA endomysial antibodies (EMA) (depending on what is available locally). Both antibody tests are highly sensitive and specific to coeliac disease but may fail to diagnose up to 10% of cases. The second step is a duodenal biopsy, which is considered to be the 'gold standard' diagnostic test. It is essential that anyone being tested for CoD continues to eat a normal gluten-containing diet, since avoiding gluten beforehand can cause false negative results. In patients who are antibody-negative, but where there is still clinical suspicion of CoD (e.g. based on symptoms or positive family history), a duodenal biopsy should still be undertaken, having ensured that the patient is not on a self-imposed gluten-free (GF) diet. Coeliac disease can present at any age and has a wide range of symptoms. Some affect the gut and include bloating, constipation or diarrhoea, excessive wind, nausea and vomiting (with or without unintentional weight loss). Other symptoms include tiredness, recurrent mouth ulcers, anaemia (deficiency of any one or combination of iron, folic acid, or vitamin B_{12}), osteoporosis, joint or bone pain, dermatitis herpetiformis (DHp), depression, alopecia, infertility, and repeated miscarriage. There is also greater appreciation of neurological symptoms of CoD, such as peripheral neuropathy and ataxia.

The only treatment for CoD is the life-long exclusion of gluten from the diet. This is a major task and should always involve expert dietetic counselling; an annual dietetic review for all patients with CoD is also recommended.

Coeliac disease is associated with increased risk of reduced bone mineral density (osteopenia), osteoporosis and a modestly increased risk of malignancy. Following a strict GF diet for 5 years or more protects against malignancies.

The gluten-free (GF) diet

Foods can be categorized into three groups:

- Foods and ingredients that are naturally GF including rice, corn (maize), potato, polenta, quinoa, milk, cheese, eggs, fresh fruit and vegetables, fresh meats including poultry, fresh fish and seafood, pulses, butter, margarine, cooking oils.
- Foods that contain obvious sources of gluten (e.g. made with gluten-containing cereals), such as bread, pasta, breakfast cereals, cakes, and biscuits.
- Foods that may include a gluten cereal as an ingredient, e.g. used as a thickener, filler, or carrier for flavours. These include some manufactured soups, sauces, sausages, and ready meals.

Prescribable products

There are a wide range of GF products available on prescription for those medically diagnosed with CoD. The range includes staple parts of the diet such as breads, pasta and flours. Luxury items such as biscuits are not prescribable but can be bought in supermarkets, on the internet or in health food shops. A full list of prescribable products can be found in the Monthly Index of Medical Specialties (MIMS), the British National Formulary (BNF), the Drug Tariff and Coeliac UK's Food and Drink Directory. Guidelines are available to assist healthcare professionals on adequate amounts to prescribe, which were developed by the British Dietetic Association (BDA), the Primary Care Society for Gastroenterology (PCSG), and Coeliac UK (℔ www.coeliac.org.uk).

Food labelling

It is essential that people with CoD understand food labels, so they know how to check that a food is suitable. All pre-packaged foods bought in the UK must comply with EU allergen labelling legislation. If an ingredient containing gluten is deliberately used in any pre-packed food (regardless of the amount), it must be stated on the ingredients list. Manufacturers must name the gluten containing grain such as, 'wheat', 'rye', 'barley', 'oats'. Some will also include the word 'gluten' as well, e.g. 'wheat gluten'.

From January 2012, legislation based on the revised Codex Standard for gluten (2008) will apply. This means that:

- Foods that contain less than 20 ppm of gluten can be labelled 'gluten-free'. This includes naturally gluten-free foods, specialist substitute products and pure, uncontaminated oats and oat products.
- Products that contain between 21 and 100 ppm gluten can be labelled 'very low gluten'. This will include specialist substitute products (mainly obtained on prescription).

Further information on this legislation can be found from Coeliac UK or Food Standards Agency

Crossed Grain symbol

Some GF manufactured products can be identified using the *Crossed Grain* symbol, which is used under licence from the charity, Coeliac UK. Some supermarkets have their own version of the Crossed Grain symbol, which also show that the food is suitable to eat.

Contamination

People with CoD should be advised to avoid contamination with gluten. It is important when at home to use a separate toaster (or use toaster bags) to prepare gluten-free toast, as well as separate bread boards and spread containers to prevent contamination with bread crumbs. Sometimes even naturally gluten-free grains (such as buckwheat) can be contaminated during milling or processing. Manufacturers may issue voluntary guidance on food labels that state the product 'may contain' a gluten cereal, which means they have identified a risk that the product may be contaminated.

Other sources of gluten

- The Medicines and Healthcare products Regulatory Agency (MHRA) advise that listed prescribed medications in MIMS and BNF are gluten-free; people with CoD should discuss with their doctor about any unexpected side effects.
- Wine, cider, liqueurs, and spirits (including malt whisky) are gluten-free, but beer, lager, stout, and ales may not be so should be avoided. Specially manufactured gluten-free ales and beers are available, details are available from Coeliac UK.
- Most traditional communion wafers are made from wheat flour. Specially manufactured GF wafers are available, details can be obtained from Coeliac UK.

Oats

There is limited good quality evidence that adults with CoD can tolerate the gluten-like protein found in oats (avenins). However, most oats and oat products are often contaminated with other sources of gluten during milling which makes them unsuitable for someone with CoD.

Anyone with CoD wanting to eat oats must make sure that they are pure and uncontaminated, as well as receiving follow-up from their healthcare team. Details of products which are free from gluten contamination are available from Coeliac UK.

Compliance

Everyone with CD is advised to follow a strict gluten-free diet. Evidence suggests that compliance can vary between 45 and 94%. The reasons for non-compliance are numerous and varied, but include:

- Restrictions to everyday lifestyle.
- Difficulties maintaining a strict diet when eating out, at school, whilst travelling or when on holiday.
- Longer food preparation and cooking.
- Some patients can feel well and lack symptoms so perceive little benefit from following GF diet.

Other dietary factors to consider

Calcium

Adults with CoD are recommended to aim for ↑ calcium intake to help optimize their bone health. The requirement for men aged ≥55 years and post-menopausal women is 1200 mg/day and for other adults with CoD is 1000 mg/day. In some cases, supplements may be required, which should be determined on individual basis. There is no increased calcium requirement for children.

Healthy eating

At diagnosis of CD, most adults have a normal body mass index or are overweight. In addition, weight gain on a GF diet is common. Once established on a GF diet, people with CD should be advised to combine this with healthy eating (see 📖 Chapter 2, 'Eatwell Plate', p. 27) and healthy weight recommendations for the general population (see 'Adult BMI ready reckoner' inside front cover).

Gluten sensitivity

Typical CoD has been described as the 'tip of the iceberg' of a spectrum of gluten sensitivity that includes asymptomatic latent and potential CoD.[1] A recent model has been proposed, linking CoD with gluten sensitivity (both GI and extra intestinal) and irritable bowel syndrome.[2]

Further information

- British Society of Gastroenterology (2010). *The management of adults with coeliac disease*. www.bsg.org.uk
- Coeliac UK www.coeliac.org.uk Helpline 0845 305 2060.
- NICE (2009) *Recognition and assessment of coeliac disease*. www.nice.org.uk/CG86.

[1] Hopper, A.D., Hadjivassiliou, M., Butt, S., *et al.* (2007). Adult coeliac disease. *Br. Med. J.* **335**: 558–62.

[2] Ball, A.J., Hadjivassiliou, M., Sanders, D.S. (2010). Is gluten sensitivity a 'no man's Land' or a 'fertile crescent' for research? *Am. J. Gastroenterol.* **105**: 222–3.

Intestinal failure and short bowel syndrome

Patients with intestinal failure (IF) require specialized nutritional support which is integral to their medical management. IF may result from either a loss of function or a reduction in GI tract length (short bowel syndrome, SBS). Common causes include post-operative complications, resection (often multiple) 2° to Crohn's disease, mesenteric infarction, radiation enteritis and traumatic injury.

Nutritional implications

- IF depends on the degree and site of intestinal disease or resection and whether there is continuity with the rest of the gut or a high output stoma.
- Parenteral support will usually be required in individuals with SBS with:
  - <100 cm of intestine + jejunostomy (fluid ± electrolytes only);
  - <75 cm of intestine + jejunostomy (parenteral nutrition);
  - <50 cm of intestine + colon intact (parenteral nutrition).
- The remnant gut may be able to adapt and increase absorptive capacity (depending on underlying condition) over a period of up to 2 years. Dietary modification and hormonal stimulation can help optimize this.
- Each patient must be individually assessed and advised by a registered dietitian with experience in IF/SBS.[1] Most patients are treated at specialist centres where relevant expertise is available. Regular monitoring of nutritional status is an essential component of ongoing medical care which will facilitate optimum oral intake and thus ↓ dependency on parenteral nutrition.

Diet in SBS—Jejunum in continuity with colon

A high carbohydrate (60% energy), low fat (20% energy) diet is associated with reduced faecal weight and ↑ water absorption. Increasing CHO intake is important as colonic bacteria will convert it to short chain fatty acids, which will be readily absorbed in the colon ∴ contributing significantly to energy balance. Reducing fat intake is essential to help decrease steatorrhoea and thus maximize absorption of other nutrients, although restrictions should be minimized to those needed to control symptoms because fat is a good source of energy. Medium chain triglycerides may be used to provide additional energy (see Box 26.6). Fat soluble vitamins should be supplemented or status monitored. Oxalate intake should be minimized due to ↑ risk of calcium oxalate renal stones (ideally avoid rhubarb, spinach, beetroot, peanuts, and excessive tea).

Diet in SBS—Jejunostomy

By contrast, reducing fat intake is not necessary in patients with a jejunostomy as the proportion of fat absorbed remains constant as fat intake increases. Medium chain triglycerides do not increase overall energy absorption so confer no benefit. Hypotonic drinks should be avoided because fluids providing <90 mmol Na^+/l will result in sodium secretion

[1] Culkin, A., Gabe, S.M., and Madden, A.M. (2009). Improving clinical outcome in patients with intestinal failure using individualised nutritional advice. *J Hum Nutr Dietet* **22,** 290–8.

into the lumen leading to ↑ fluid loss. A high salt intake is recommended due to jejunal losses of sodium, and patients will benefit from an oral rehydration solution containing 90 mmol Na^+/L.

Further reading

'Understanding intestinal failure' for patients. Available at: www.stmarkshospital.org.uk/uploads/content/docs/patientinformationleaflets/Understanding%20IF.pdf

Fistulae

Gastrointestinal fistulae are abnormal holes in the gut allowing the contents (partially digested food, secretions, water, and electrolytes) to escape either into another part of the thoracic or abdominal cavities or to the outside. Most (~80%) arise as a complication of surgery, while some occur spontaneously in inflammatory bowel disease, diverticular disease, and radiation enteritis or as a result of trauma. The mortality, morbidity, and nutritional consequences are significant.

Nutritional implications

- Patients with fistulae are frequently undernourished because of their underlying disease and the consequences of nutrient loss via the fistula. Impaired nutritional status will adversely affect closure, whether surgical or spontaneous.
- There is no evidence that parenteral nutrition is superior to feeding via the enteral route. Current practice in specialist units is to allow all patients to eat, if they are able to, regardless of the fistula output unless there is intra-abdominal sepsis which is exacerbated by using the enteral route. In these patients parenteral nutrition may be required (see 🕮 Chapter 25, 'Parenteral nutrition', p. 536). This should take account of the individual's losses via the fistula and, depending on the site, additional fluid and electrolytes may be required to replace losses.
- Fistuloclysis, enteral feeding via the fistula, may be possible and desirable in some patients. If achieved, this avoids the complications which are associated with parenteral nutrition and *may* promote faster fistula healing.[1] Nutrition support can be provided by accessing the GI tract distally to the fistula if high, e.g. oesophageal, gastric, jejunal. A minimum length of 75 cm of small bowel distal to the fistula is required to achieve independence from parenteral nutrition. Evaluation of fistula losses is required to facilitate adequate replacement of nutrients, fluid, and electrolytes and, in some patients, enteral feeding proximal to a fistula may increase output. Opinion is divided about whether high output fistulae (≥200 ml/day pancreatic, 500–1500 ml/day intestinal) are less likely to spontaneously close. However, enteral feeding should not be automatically discounted in fistula patients due to its advantages associated with lower rates of infection and maintaining GI integrity.

[1] Ham, M., Horton, K., and Kaunitz J. (2007). Fistuloclysis: Case Report and Literature Review. *Nutr. Clin. Pract.* **22,** 553–7.

Gastrointestinal stoma

Stoma differ from GI fistulae in that access to the GI tract is deliberate and aims to facilitate either:

- *Input* = nutrient intake into the stomach (feeding gastrostomy) or jejunum (feeding jejunostomy), (see 🕮 Chapter 25, 'Routes for enteral feeding', p. 518).
- *Output* = effluent from the jejunum (jejunostomy), ileum (ileostomy), or colon (colostomy).

Jejunostomy

See 🕮 this Chapter, 'Intestinal failure and short bowel syndrome', p. 596.

Ileostomy

Usually formed after resection in inflammatory bowel disease. Although most patients eat a normal diet, depending on the length of remaining ileum, attention must be given to the fluid and electrolytes lost from the ileostomy effluent. Losses >1500 ml/day will require additional fluid and salt. Vitamin B_{12} is absorbed at the distal end of the ileum so patients with ileo-caecal resection will require supplementation, e.g. intramuscular injection 1 mg hydroxocobalamin every 3 months.

Some patients prefer to avoid specific foods if they experience unacceptable symptoms (see Box 26.7). The identification of recognizable food remains, e.g. pips, skins, grain husks, in the effluent may concern some patients. Providing this is not associated with a high output or other symptoms, reassurance should be given and patients advised to chew their food well.

Box 26.7 Reported effects

- ↑ *Output:* high fibre foods, beetroot, mushrooms, spicy foods, alcohol, fruit juice, milk
- *Flatus-producing:* onions, peas, beans, carbonated drinks, spicy foods, beer, milk
- *Offensive odour:* fish, onions, leeks, garlic, eggs.

Colostomy

Like ileostomy, often formed after resection in inflammatory bowel disease, bowel cancer, or diverticular disease, although more common. As effluent leaves the GI tract distally to the ileum, there is less concern with fluid and electrolyte losses. Patients should ∴ eat as normally as possible but avoid specific foods that cause unacceptable symptoms (see Box 26.7). Patients with a colostomy may experience constipation and should be encouraged to increase their intake of fruit and vegetables and wholegrain cereals (see 🕮 this Chapter, 'Constipation', p. 602). Diarrhoea should be investigated if prolonged and exceeding 1000 g/day.

Intestinal transplantation

Transplantation of the intestine (small bowel) may be undertaken singly or in conjunction with other abdominal organs, including the liver and pancreas. Approximately 90 transplants have been undertaken in the UK (1987–2009), predominantly in children, with ~180 being undertaken annually in the USA.

Indications include intestinal failure where total parenteral nutrition is associated with cholestatic liver failure.

Long-term follow-up studies (up to 10 years) show that surviving patients can be sustained independently of parenteral nutrition and that growth velocity in children can be maintained, although catch-up growth is rare.

Nutritional management

Goals include:

- Reducing the risk of graft rejection.
- Improving gut trophicity, i.e. maximizing function, which may be acquiescent.
- Optimizing nutritional status.

Route

There are no studies to indicate optimum post-transplant nutritional management, but parenteral nutrition, which is usually essential *before* surgery, is required in early post-operative period. Enteral nutrition is theoretically well-placed to improve gut trophicity, optimize absorptive function and prevent bacterial translocation. Although tolerance of enteral feeds may be limited after surgery by ischaemia-reperfusion injury, denervation and absence of lymphatic drainage of the graft, feeding into the gut should commence as soon as possible and ideally within 5–7 days.[1]

Access

Some centres feed into the stomach and others into the jejunum via either a jejunostomy or gastro-jejunostomy because of delayed gastric emptying.

Feed

Practice varies between centres with elemental feeds commonly used and some evidence that peptide-based formulae are tolerated. Diluted feeds are used in some centres whilst others use full strength from initiation. Clearly, studies are needed to identify best practice.

Restrictions

Avoiding dairy products (due to lactose and potential allergens) and limiting fat intake is also reported in the post-operative period or if chylous ascites is present.

Long-term

Most patients are able to transfer to an oral intake and maintain an adequate nutritional status after transplantation although continued nutritional support may be required for some years in a few. Absorption of

[1] Columb, V., and Goulet, O. (2009). Nutrition support after intestinal transplantation: how important is enteral feeding ? *Curr. Opin. Clin. Nutr. Metab. Care* **12,** 186–9.

lipids and fat soluble vitamins may remain below normal in some patients, and is probably associated with an inadequate post-surgical lymphatic circulation. Monitoring of fat soluble vitamin status is, therefore, required.

Disorders of the colon

Constipation

Constipation is a significant health problem in the UK and most high income countries and is associated with a high number of medical consultations and expenditure on laxatives, especially in older people. It is also prevalent in children with 5–30% affected.

Intractable and severe constipation requires investigation and treatment of any specific underlying pathology. However, in many cases, a regular and more frequent bowel habit can be achieved by simple dietary measures, although dietary interventions alone should not be used as first-line treatment in idiopathic constipation in children.[1]

Nutritional management is compatible with 📖 Chapter 2, 'Eatwell Plate', p. 27 and is based on increasing dietary fibre (see 📖 Chapter 5 'Carbohydrate', p. 72) and taking an adequate fluid intake:

- Change to wholegrain bread and gradually increase daily intake to 200 g (6–7 average slices).
- Take a large bowlful (50 g) of wholegrain cereal daily.
- Increase fruit and vegetable intake to 400 g/day. This is equivalent to five portions per day.
- Try wholegrain rice and pasta as alternative to white varieties.
- Include nuts, beans, and pulses.

Other measures include the following:

- In order to be effective in increasing faecal bulk and promoting bowel activity, cereal fibre must be able to absorb fluid. In order to do this, there must be sufficient fluid in the colon. In adults, fluid intake should be a minimum of 35 mL/kg/day (i.e. ~2½ L for a 70-kg man) and should lead to passing of pale, straw-coloured urine. For children, see Table 13.1.
- People with a very low fibre diet should be advised to increase their intake of fibre gradually to help the GI tract adapt and to avoid possible side-effects of abdominal distension and ↑ flatus. If these symptoms occur, they are often self-limiting and resolve spontaneously. If they do not, selective manipulation of fibre-containing foods may help identify ones that cause less difficulty, but are still effective at promoting bowel evacuation.
- A high intake of cereal bran is not recommended as a first-line treatment in constipation because of the potential for sequestering micronutrients and thus reducing absorption. Its effects are variable, rapid in some people but less effective in others. In addition, bran has been associated with bloating and flatulence and, in elderly people with a low fluid intake, bowel impaction. If bran is used, it should be introduced slowly into the diet and limited quantities (<10 g/day) taken in conjunction with plenty of fluid and a well-balanced diet.
- Prunes and prune juice are frequently used to treat constipation and are probably effective through their content of sorbitol and phenolic compounds.

[1] NICE (2010). *Constipation in children and young people*. NICE, London. Available at: 🖱 www.nice.org.uk/CG99

Diverticular disease

Diverticula are blind pouches found in the intestinal wall, particularly in older people. Traditionally, this has been attributed to ↑ intraluminal pressure as a result of constipation and a low fibre diet but age, diet, inflammation and genetics may all contribute to diverticular formation.[2] In themselves, diverticula are not considered pathological, but with a continued low fibre diet, may become inflamed and infected resulting in diverticulitis. This is manifest as colicky pain with diarrhoea and/or constipation; bleeding, abscesses, and perforation may complicate. An acute episode requires treatment with antibiotics and surgery in severe cases. On recovery, recurrence can be reduced by following a high fibre diet and an adequate fluid intake (see 📖 'Constipation', p. 602).

Haemorrhoids

Haemorrhoids (or piles) are swollen and inflamed blood vessels in the rectum and anus. They arise after prolonged constipation when pressure in the distended colon is combined with straining to evacuate the bowel, and are most common in older adults and during/after pregnancy. Patients presenting with internal (non-prolapsing) haemorrhoids should be advised to increase their fibre and fluid intake. Others should be referred to a colorectal surgeon and will benefit from advice to ↑ fibre and fluid intake after treatment.

Increasing fibre intake is useful for softening faeces, relieving constipation, and thus reducing straining. Fibre supplementation reduces episodes of bleeding and discomfort in patients with internal haemorrhoids although this may take up to 6 weeks; it does not improve external (prolapsed) haemorrhoids. Cereal fibre is most effective in increasing stool weight (see 📖 this Chapter, 'Constipation', p. 602) and bulk-forming laxative, ispaghula husk, may help.

Cancer of the colon and rectum

The incidence of colorectal cancer in the UK is ~46 cases per 100,000 population making this one of the three most common malignancies. Worldwide, it is estimated that 1.25 million new cases were diagnosed in 2008. It is more common in men, people aged >60 years and socio-economically deprived groups. Nutritional factors contribute to causation (Table 26.4).

Table 26.4 Nutritional factors and risk of colorectal cancer

Factors associated with ↑ risk	Factors associated with ↓ risk
Red and processed meat	Dietary fibre
Obesity	Fruit and vegetables
Abdominal fatness	Physical activity

[2] Commane, D.M., Arasaradnam, R.P., Mills, S., *et al.* (2009). Diet, ageing and genetic factors in the pathogenesis of diverticular disease. *World J. Gastroenterol.* **15,** 2479–88.

Treatment

Usually includes surgery (colectomy with or without formation of a temporary or permanent colostomy; see 🕮 'Gastrointestinal stoma', this chapter, p. 599), and sometimes chemotherapy and/or radiotherapy (see 🕮 Chapter 24, 'Chemotherapy', p. 490 and 'Radiotherapy', p. 491).

Nutritional management

Evidence that dietary or lifestyle interventions can influence remission or survival is limited but considering the prevalence of colorectal cancer and the efficacy of medical and surgical treatment, many people living with this condition could benefit from further study in this area. At present, no specific therapeutic dietary regime can be recommended, but maintaining an adequate and well-balanced intake can help to optimize the patient's nutritional status and his/her ability to withstand surgery or other treatment. Patients may seek dietary advice relating to a colostomy (see 🕮 this Chapter, 'Gastrointestinal stoma', p. 599) or may benefit from guidance if the tumour or treatment is compromising nutritional intake. Depending on the stage of the tumour at diagnosis, long-term dietary advice compatible with the Eatwell Plate (see 🕮 Chapter 2, p, 27) may be appropriate whilst for others, nutrition support to counter weight loss (see 🕮 Chapter 25, 'Treatment of undernutrition', p. 512) or a more palliative approach to eating (see 🕮 Chapter 35, p. 715) should be considered.

Irritable bowel syndrome

Irritable bowel syndrome (IBS) is a chronic GI disorder with no identified structural pathology (Box 26.9). It affects all ages and races and ~twice as many women as men. Estimated prevalence in the general population in the UK is 10–20%, but this may be an underestimate as many sufferers are thought to self-care. Symptoms may overlap with other GI disorders, e.g. dyspepsia or coeliac disease, include:

A – abdominal pain or discomfort.
B – bloating or abdominal distension.
C – change in bowel habit (constipation or diarrhoea).

Box 26.9 Diagnosis of IBS*

IBS should be diagnosed if a person has abdominal pain or discomfort that is:

- either relieved by defaecation or associated with altered bowel frequency or stool form *and*
- has at least two of the following symptoms:
  - altered stool passage (straining, urgency, incomplete evacuation
  - abdominal bloating (more common in women than men), distension, tension or hardness
  - symptoms made worse by eating
  - passage of mucus.

Other supporting characteristics include lethargy, nausea, backache and bladder symptoms.

* NICE (2008). *Irritable bowel syndrome in adults*. NICE, London. Available at: www.nice.org.uk/CG61

Aetiology

Definitive causes have not been determined but probably include disturbed colonic motility, post-infective dysfunction ± altered anti-pain response. Stress is reported as a triggering factor in ~50% of sufferers. Dietary factors probably play a role in some individuals and intolerance to wheat, dairy products, and coffee is frequently reported, but true food allergy is rare. Lactose intolerance is found in 10% of IBS patients, but low lactose diets rarely resolve the condition. Excessive caffeine intake may explain some symptoms.

Dietary and lifestyle advice

People with IBS should be offered information explaining about the importance of self-help in managing their condition, and including advice about diet, physical activity, and relaxation.

- Encourage regular meals and taking time to eat.
- Avoid missing meals or leaving long gaps between eating.
- Drink ≥8 cups/day, especially water or other non-caffeinated drinks.
- Restrict tea and coffee to ≤3 cups/day.
- Reduce intake of alcohol and fizzy drinks.
- Consider limiting intake of high-fibre food, e.g. wholemeal or high-fibre flour and breads, cereals high in bran, whole grains such as brown rice.

- Discourage use of added bran.
- Reduce intake of 'resistant starch' (see Table 5.10).
- Limit fresh fruit to 3 portions daily (a portion ≈ 80 g).
- Avoid sorbitol, an artificial sweetener found in sugar-free sweets, chewing gum and drinks.
- Oats and linseeds (up to 1 tablespoon/day) may help bloating.
- If probiotics are tried, it is advisable to take for at least 4 weeks at dose recommended by manufacturer while monitoring the effect.
- Discourage use of aloe vera.
- There is insufficient evidence to support use of herbal remedies in IBS.

If, after following the above advice, food intake is still considered to be adversely associated with IBS symptoms a referral to a registered dietitian for further advice should be offered. This may include a single food or full exclusion diet or restricting intake of rapidly fermentable carbohydrate.

Exclusion diets

Although food allergies are rare in IBS and effects of dietary exclusion are mixed (see next section), evaluating 'offending' food items through a proper exclusion diet may provide reassurance. This should be managed with the assistance of a dietitian with experience in this area to ensure that the nutrient intake remains adequate.

Strict elimination diets (avoiding all food except one meat and one fruit before gradual reintroduction of single items) may benefit up to 70% of those completing the regime; less stringent schemes yield benefits in ± 50% of patients. When this treatment option is considered, the potential benefits should be weighed against the effort and restriction required to comply and a fixed time, rather than open-ended approach taken.

FODMAP diet

A new approach for managing IBS is based on restricting dietary rapidly fermentable carbohydrates, abbreviated to FODMAP (**f**ermentable **o**ligosaccharides **d**isaccharides **m**onosaccharides **a**nd **p**olyols). This involves manipulation of intake to reduce dietary fructose, lactose, fructans, and galactans, and is associated with a reduction in symptoms that can be provoked by reintroducing these dietary carbohydrates. The proposed mechanism involves the delivery of water to the distal small intestine which leads to symptoms. Early evidence suggests that this approach may have benefits in other GI conditions including inflammatory bowel disease.

Further information

Gibson, P.R., and Shepherd, S.J (2010). Evidence-based dietary management of functional gastrointestinal symptoms: the FODMAP approach. *J. Gastroenterol. Hepatol.* **25**, 252–8.

NICE (2008). Irritable bowel syndrome in adults: diagnosis and management of irritable bowel syndrome in primary care. NICE, London. Available at: http://www.nice.org.uk/nicemedia/live/11927/39746/39746.pdf

Spiller, R., Aziz, Q., Creed, F., *et al.* (2007). Guidelines on the irritable bowel syndrome: mechanisms and practical management. *Gut* **56**, 1770–98.

Gall bladder disorders

Gallstones

Gallstones are relatively common in populations consuming a 'Western' diet and it is estimated that ~10% of adults in the UK have gallstones, two-thirds of whom are asymptomatic. The prevalence increases with age and is more common in women. Most gallstones (~80%) are composed of predominantly cholesterol, while others have a higher proportion of bilirubin, calcium, and pigments. Cholesterol precipitates into stones when bile becomes (1) supersaturated and (2) gallbladder emptying is reduced. See Box 26.10 for the risk factors for cholesterol gallstones.

Box 26.10 Risk factors for cholesterol gallstones

- Age >40 years
- Female
- Genetic variation
- ↑ Dietary fat
- ↑ Dietary refined carbohydrate
- ↓ Dietary fibre
- Obesity
- Yo-yo dieting (repeated cycles of losing and re-gaining weight)
- Hyperlipidaemia
- Pregnancy
- Diabetes mellitus and insulin resistance
- Liver disease
- Cystic fibrosis
- Gall bladder dysmotility
- Bile salt loss (ileal disease ± resection)
- Nil enterally (fasting or parenteral nutrition)
- ↓ Physical activity.

Preventing gallstones

- A diet compatible with the 'Eatwell Plate' is optimal (see 📖 Chapter 2, p. 27).
- Reduction of excess body weight using moderate energy restriction ~1.5 kg/week (very low calorie diet may exacerbate bile saturation).
- Eat regularly to minimize bile stasis, which accompanies fasting.
- Eat breakfast on rising (cholesterol concentrations are highest in bile produced overnight).
- Take regular exercise.

Cholecystitis

Approximately 1–4% of people with gallstones develop symptoms each year including pain (epigastric, upper or lower abdominal), nausea, and vomiting; generally, these are non-specific. Acute cholecystitis features prolonged severe pain (right subcostal) and fever.

Dietary management of cholecystitis

Historically, a low fat diet was advocated on the basis that restricting fat intake reduced gall bladder contractions and reduced pain. However, the gallbladder is known to contract in response to the oral intake of most nutrients and also in anticipation of intake. Therefore, the restriction of dietary fat is unnecessary unless a stone obstructs the hepatic or common bile duct, precluding the delivery of bile to the GI tract and thus provoking steatorrhoea (see 📖 this Chapter, 'Steatorrhoea', p. 580), jaundice, and nausea. This is relatively uncommon and requires medical intervention. If a patient is well enough to eat, s/he should be advised to follow the five points listed under 'Preventing gallstones' (p. 608) and only to avoid specific foods if they are definitely associated with symptoms.

Cholecystosteatosis

Similar to, but less common than non-alcoholic steatohepatitis (NASH) that affects the liver (see 📖 Chapter 28, 'Liver disease', p. 619) cholecystosteatosis is fatty infiltration of the gall bladder accompanied by chronic infiltration and antioxidant tissue damage. This results in ↓ contractility and thus bile stasis, which ↑ risk of gall stone formation. Although associated with dietary carbohydrate, the pathogenesis, prognosis and role of nutrition in management are unknown.

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Chapter 27

Pancreatic disease

Pancreatic disorders

See Table 27.1.

Table 27.1 Synthetic functions of the pancreas and consequences of impairment

Exocrine functions	Endocrine functions
Pancreatic lipase	Insulin
Pancreatic proteases	Glucagon
Pancreatic amylase	Somatostatin
↓	↓
Inability to digest macronutrients leading to malabsorption and undernutrition	Inability to control blood sugar level can occur in severe damage, usually after exocrine insufficiency

The major pancreatic disorders include pancreatitis and pancreatic cancer.

Pancreatitis

Pancreatitis results from the auto-digestion of the pancreas by activated pancreatic enzymes. It can be categorized as:

- Chronic pancreatitis (CP).
- Acute pancreatitis:
  - mild acute;
  - severe acute pancreatitis (SAP).

Chronic pancreatitis

Annual incidence in the UK is 40–70 per 100,000 with men approximately four times more likely to be affected than women. Rates are ↑ with ↑ alcohol intake.

Aetiology includes excessive alcohol intake (60–80% of cases in populations with 'Western lifestyle'), idiopathic (20% of cases). Other causes include biliary tract disease, duct obstruction and trauma. CP can develop following repeated acute episodes.

Symptoms/complications include severe abdominal pain, malabsorption, diabetes, malnutrition. Malnutrition is frequently reported (but no figures available) and arises due to poor intake 2° to pain, malabsorption, hypermetabolism, hyperglycaemia, and failure to provide nutrition support (i.e. inappropriate nil-by-mouth/enteral restrictions to 'rest' the pancreas).

Nutritional management of chronic pancreatitis, (Box 27.1)

- *Poor intake 2° to pain:*
  - pancreatic enzyme replacement therapy (PERT) may help, but more evidence needed;[1,2]

[1] Winstead, N.S., and Wilcox, C.M. (2009). Clinical trials of pancreatic enzyme replacement for painful chronic pancreatitis—a review. *Pancreatology* **9,** 334–50.
[2] Shafiq, N., Rana, S., Bhasin, D., *et al.* (2009). Pancreatic enzymes for chronic pancreatitis (Review). *Cochrane Database,* CD006302.

  - mixed antioxidant supplements may help;[3]
  - give analgesia before meals.
- *Malabsorption:* impairment of fat digestion is >protein >carbohydrate. Resulting steatorrhoea will lead to ↓ transit time and ↑ faecal loss of energy, protein, vitamins A, D, E, K, thiamin, folic acid, Ca, Mg, and Zn. Prescribe PERT and advise about dosage in relation to food. Maintain dietary fat intake ~30–40% of energy, i.e. not restricted. Most PERT studies in pancreatitis have focused on pain relief rather than digestive function.
- *Hypermetabolism:* limited data suggests 30–50% of CP patients have raised resting energy expenditure (~110% of predicted values) even when expressed per kg fat free mass, i.e. taking account of malnutrition. In absence of individual measurements, base energy requirements on predicted value +10%.
- *Hyperglycaemia:* even though endocrine function is usually preserved after exocrine function declines, 40–90% of patients with severe CP have ↑ blood glucose which, if uncontrolled, will exacerbate weight loss. There is no evidence to support the restriction of dietary CHO! Intake should be modified in line with low glycaemic index (see 📖 Chapter 22, 'Diabetes', p. 441) if patient is well enough to eat this. If necessary insulin should be prescribed and a diabetologist opinion sought.

Box 27.1 Key points for nutrition support in CP

- Patients with mild CP require no dietary restrictions and, if well enough to eat sufficient, will not benefit from enteral nutrition
- Patients with more advanced CP are often underweight and frail ∴ providing adequate nutritional support is paramount but may be confounded by the symptoms and complications described above. Input from a registered dietitian with expertise in this area is required
- Systematic review found inconclusive evidence of benefits of enteral versus parenteral feeding in acute pancreatitis. It is reasonable, ∴ to recommend that feeding is attempted first via the enteral route because of its associated preservation of mucosal function, which is impaired in the inflammatory response, and its lower cost
- Nasogastric feeding is feasible in ~80% of patients. Successful long-term enteral nutrition support provided by elemental feeding via an endoscopically-placed naso-jejunal tube associated with relief of pain has been reported in three cases[4]
- Parenteral nutrition is preferable to no nutrient intake where enteral feeding cannot be undertaken, e.g. ileus >5 days
- There is limited evidence, at present, to support the use of specific formulae, e.g. standard, semi-elemental, elemental, or 'immune enhanced'.

(continued)

[3] Kirk, G.R., White, J.S., McKie, L., *et al.* (2006). Combined antioxidant therapy reduces pain and improves quality of life in chronic pancreatitis. *J Gastrointest Surg* **10,** 499–503.
[4] Lordan, J.T., Phillips, M., Chun, J.Y., *et al.* (2009). A safe, effective and cheap method of achieving pancreatic rest in patients with chronic pancreatitis with refractory symptoms and malnutrition. *Pancreas* **38,** 689–92.

Box 27.1 (*cont*)

Key points for dietary advice

- Patients with a poor appetite may benefit from practical advice about increasing oral intake (see 🕮 Chapter 25 'Treatment of undernutrition', p. 512)
- If malabsorption is present, advice should be given about pancreatic enzyme replacement (see 🕮 this chapter, 'Pancreatic enzyme replacement therapy', p. 616)
- Low fat diets have no role to play in the treatment of pancreatitis as they will exacerbate energy depletion and steatorrhoea should be controlled by pancreatic enzyme replacement
- A compromise should be reached between dietary advice to optimize blood sugar if hyperglycaemia develops (see 🕮 Chapter 22, 'Goals and principles of dietary management', p. 448) and to enhance intake to maintain body weight or reverse weight loss
- Abstaining from alcohol is highly advisable as continuing alcoholism is associated with ↑ morbidity and mortality.

Acute pancreatitis (Box 27.2)

Annual incidence internationally is 5–80 in 100,000.

Aetiology includes gallstones (~40% of cases in populations with 'Western lifestyle'), excessive alcohol intake (~35% of cases), idiopathic (15% of cases). Repeated acute episodes can lead to CP.

Symptoms/complications include severe pain, nausea, vomiting, pseudocysts, fistulae, shock, and renal failure.

Box 27.2 Key points for nutrition support in acute pancreatitis

- Meta-analysis[1] shows better clinical outcomes are associated with:
  - enteral feeding rather than starvation (1 study showing 78% ↓ mortality risk, $p = 0.01$)
  - enteral, rather than parenteral nutrition (10 studies, relative risk of infective complications, 0.41 (95%CI, 0.30, 0.57), $p = 0.00001$; relative risk of mortality, 0.60 (0.32, 1.14), p = 0.12)
- Meta-analysis in SAP[2] shows enteral rather than parenteral nutrition is associated with more diarrhoea (5 studies, 29% vs. 7% with PN), but less hyperglycaemia (11% vs. 29% with PN).

[1] Petrov, M.S., Pylypchuk, R.D. and, Emelyanov, N.V. (2008). Systematic review: nutritional support in acute pancreatitis. *Alimentary Pharmacol Therapeut* **28,** 704–12.
[2] Petrov, M.S, and Whelan, K. (2010). Comparison of complications attributable to enteral and parenteral nutrition in predicted severe acute pancreatitis: a systematic review and meta-analysis. *Br. J. Nutr.* **103,** 1287–95.

Pancreatic cancer

Cancer of the pancreas is the 11th most common malignancy diagnosed in the UK (~8000 cases per year). It is equally common in men and women but more likely to occur in people aged >50 years. Smoking and a history of pancreatitis are risk factors.

Treatment may include surgery, chemotherapy, and radiotherapy (see 📖 Chapter 24, 'Chemotherapy', p. 490 and 'Radiotherapy', p. 491) depending on the stage of the tumour at diagnosis.

Nutritional management should include assessment of nutritional status and aim to provide an adequate energy and nutrient intake in a format that can be tolerated by the patient. Patients with pancreatic cancer are often undernourished on diagnosis as a result of poor intake and severe pain; depletion may be exacerbated by treatment. Appropriate nutritional support is ∴ essential, but depends on the treatment given and overall prognosis.

- No dietary restrictions are needed.
- Nutritional support, preferably via the enteral route, may improve well-being.
- After pancreatico-duodenectomy, combined enteral and parenteral nutrition in the post-operative period.
- Pancreatic enzymes should be used to reduce steatorrhoea and to help control pain (even if steatorrhoea is absent).
- Supplementation with *n*-3 fatty acids (see Table 23.3) is associated with weight gain (including lean tissue) and improved quality of life.

Further reading

Gianotti, L., Meier, R., Lobo, D.N., *et al.* (2009). ESPEN Guidelines on parenteral nutrition: Pancreas. *Clin Nutr* **28**, 428–35.

Meier, R., Ockenga, J., Pertkiewicz, M., *et al.* (2006). ESPEN guidelines on enteral nutrition pancreas. *Clin Nutr* **25**, 275–84.

Pancreatic enzyme replacement therapy

Pancreatic enzymes include lipase, amylase, and proteases, e.g. trypsin, chymotrypsin, which contribute to the digestion of fat, carbohydrate, and protein, respectively. Their insufficiency can result in malabsorption but this can be treated effectively with pancreatic enzyme replacement therapy (PERT) where combined enzymes are provided as capsules, granules, or tablets (Table 27.2). Patients require advice about the use of PERT in order to optimize the effects of therapy.

- The dose prescribed may need to be adjusted to resolve malabsorption and should be varied depending on what is eaten. Specific advice should be given to individual patients by healthcare professionals with expertise in using PERT.
- Enzymes should be taken with all foods containing fat, protein, or starchy carbohydrate, e.g. meals and snacks. Sugary foods containing no fat and protein do not require PERT.
- Enzymes should be taken just before and/or with the meal or snack.
- Enzymes are deactivated by heat so should not be mixed with hot food or drinks.
- Enzymes are also deactivated by acidity (pH<4). Capsules containing enterically-coated microtablets should be swallowed whole or, if opened, swallowed without chewing the contents as this will expose the enzymes to the denaturing effects of gastric acid.
- H_2 antagonists may be prescribed to reduce gastric secretions and maximize the effects of PERT.
- Skin contact with enzymes taken as granules may cause irritation and should be avoided.
- High strength preparations are available. Reports have associated these with the rare development of large bowel strictures when taken by children with cystic fibrosis (CF). As a result, daily dose in CF is recommended as not exceeding 10,000–250,000 units lipase/kg body weight/day[1] and adequate hydration should be ensured. New or changing abdominal symptoms should be investigated.

[1] Munck, A. (2010). Nutritional considerations in patients with cystic fibrosis. *Expert Rev Resp Med* **41,** 47–56.

Table 27.2 Examples of pancreatic enzyme replacements available in the UK

Brand name (manufacturer)	Enzyme content (BP units/capsule*)		
	Lipase	Protease	Amylase
Creon Micro (Solvay)	5 000	200	3 600
Pancrex (Paines & Byrne)	5 000	300	4 000
Nutrizym 10 (Merck Serono)	10 000	500	9 000
Creon 10 000 (Solvay)	10 000	600	8 000
Higher strength preparations			
Nutrizym 22 (Merck Serono)	22 000	1100	19 800
Pancrease HL (Janssen-Cilag)	25 000	1250	22 500
Creon 25 000 (Solvay)	25 000	1000	18 000
Creon 40 000 (Solvay)	40 000	1600	25 000

* Except for Creon Micro (gastro-resistant granules, enzyme content in BP units/100 mg) and Pancrex (granules, enzyme content in BP units/g).

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Chapter 28

Liver disease

Introduction and nutritional assessment

Nutrition-related functions of the liver include:
- emulsification of dietary fat by bile prior to digestion;
- carbohydrate metabolism (maintain blood glucose, store glycogen);
- protein metabolism (synthesis and regulation of amino acids);
- lipid metabolism (production of triglycerides and lipoprotein);
- micronutrients (storage of vitamins A, B_2, B_3, B_6, B_{12}, K, folate).

Impairment of these functions, combined with poor nutrient intake, leads to many patients with liver disease being undernourished and requiring nutrition support.

Nutritional assessment

Assessment of nutritional status is difficult in patients with liver disorders because standard methods are confounded by the disease process, e.g. fluid retention distorts body weight (Table 28.1), ↓ liver function precludes use of most biochemical markers. A global assessment (see Box 28.1) provides a reliable evaluation, which has predictive and construct validity.

Box 28.1 Key points for assessing global nutritional status in patients with liver disease*

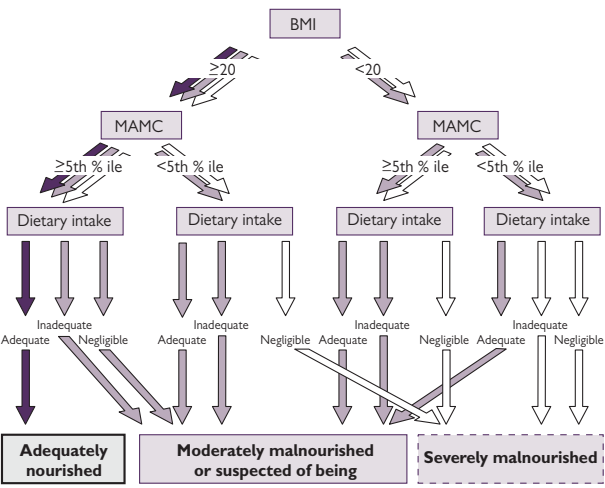
- Determine body mass index (derived from estimated dry weight, see Table 28.1) relative to 20 kg/m^2
- Determine mid-arm muscle circumference (MAMC) relative to the 5th percentile of gender- and age-matched reference values (Bishop's standards; see Appendix 2, p. 751)
- Estimate adequacy of recent energy intake relative to estimated requirements (see 📖 Chapter 25, 'Estimating requirements in disease states', p. 540)
- Follow algorithm (Fig. 28.1), using additional factors likely to impair nutritional status (e.g. ascites, malabsorption) if present to subjectively override final category of nutrition.

* Morgan, M.Y., Madden, A.M., Soulsby, C.T., *et al.* (2006). Derivation and validation of a new global method for assessing nutritional status in patients with cirrhosis. *Hepatology* **44**: 823–35.

Table 28.1 Guidelines for estimating fluid weight (kg) in patients with ascites and peripheral oedema*

	Ascites	Oedema
Minimal	2.2	1.0
Moderate	6.0	5.0
Severe	14.0	10.0

*Mendenhall, C.L. (1992). Protein–calorie malnutrition in alcoholic liver disease. In: Watson, R.R. and Watzl, B., eds. *Nutrition and alcohol*. Boca Raton, FL: CRC Press, pp. 363–84.



Subjectively override using additional factors likely to impair nutritional status.

Fig. 28.1 Algorithm for assessing nutritional status in patients with liver disease.

Hepatitis and cirrhosis

Hepatitis—acute and chronic

Hepatitis is an inflammation of the liver and can arise from a viral infection, e.g. hepatitis A, B, C, D, E, and G, an auto-immune response, or from other damage, e.g. alcoholic hepatitis. The nutritional implications vary depending on the severity and duration of the condition. See Box 28.2 for implications for breastfeeding.

Acute hepatitis In general, patients with acute hepatitis are very ill, have a poor appetite, and eat very little. No dietary restrictions should be imposed and patients should be encouraged to eat what they can. Small frequent snacks and nourishing drinks may be better tolerated than large meals. Fat restrictions used in the past are not evidence-based and dietary fat will not exacerbate the condition if the patient is able to eat it. Severe undernutrition may accompany acute alcoholic hepatitis and instigating early nutrition support, usually through enteral feeding, is recommended.

Chronic hepatitis Patients with chronic hepatitis may vary from those who are very undernourished following prolonged illness and poor intake to those who are obese, either incidentally or secondary to long-term treatment with steroids. Individual assessment is required and nutritional advice tailored accordingly.

Nutritional management

Appropriate nutritional management is important and influences prognosis.

- Undernourished patients who go on to liver transplantation have a greater risk of complications than those who are better nourished.
- Obesity is a risk factor for development and progression of chronic liver disease and associated with poor response to antiviral therapy.

Box 28.2 Is it safe for women with viral hepatitis to breastfeed?

- *Hepatitis B:* Yes. Small amounts of hepatitis B surface antigen have been detected in some samples of breast milk. However, the risk of transmission via breastmilk is considered to be negligible compared with the risk of transmission at birth and is outweighed by the benefits associated with breastfeeding. Infants should receive hepatitis B immuno globulin and the first dose of hepatitis B vaccine within 12 h of birth followed by completion of the vaccination series. Breastfeeding should not be delayed until the infant is fully immunized. It is advisable to abstain from breastfeeding if nipples become cracked and bleed
- *Hepatitis C:* Limited data, but most guidance consider it safe to breastfeed. It is advisable to abstain if nipples become cracked and bleed

http://www.cdc.gov/breastfeeding/disease/hepatitis.htm

Cirrhosis

Cirrhosis is irreversible damage of the hepatocytes. Most patients with cirrhosis have ↑ energy and protein requirements (Table 28.2), and are frequently undernourished (prevalence between 10 and 100% depending on population), ∴ requiring nutritional support. Dietary restrictions should not be routinely imposed. Complications arising from cirrhosis, including ascites, oedema, encephalopathy, and steatorrhoea, may benefit from dietary modification providing it is undertaken within a context of nutritional adequacy, i.e. feed first, restrict second.

Table 28.2 Energy and protein requirements recommended by the ESPEN* Consensus Group

	Non-protein energy (kcal/kg/day)	Protein (g/kg/day)
Cirrhosis	35–40	1.2–1.5

*European Society for Parenteral and Enteral Nutrition. Plauth, M., Cabré, E., Riggio, O., *et al.* (2006). ESPEN guidelines on enteral nutrition: liver disease. *Clin Nutr* **25**, 284–94.

Ascites and oedema

Fluid retention is common in cirrhosis and end-stage liver disease, and has relevance to nutrition.

- Abdominal distension can impair food intake leading to an inadequate nutritional status.
- Energy expenditure increases due to exertion of carrying additional weight.
- Negative nitrogen balance can be induced by repeated large volume paracentesis, even if intravenous albumin infused.
- Restricting dietary sodium can help reduce or control the degree of fluid retention, and thus improve symptoms.

Nutritional management of ascites and oedema

- Patients should be encouraged to eat as much as they are able and be tempted with tasty food.
- Small, frequent meals with snacks every 1–2 h may optimize intake.
- The energy density of foods may be ↑ by the addition of concentrated sources, e.g. sugar, honey, double cream, oil, and salt-free butter.
- Fat should not be restricted unless the patient finds it unpalatable or has clinically diagnosed steatorrhoea.
- Nutritional status should be monitored regularly. Body weight cannot be used; MAMC and triceps skin-fold thickness are most appropriate.
- Decisions about whether to restrict sodium intake must balance the potential benefits against the risk of reducing nutrient intake. Patients should be advised individually by a registered dietitian with experience in this area.

Low sodium diets for ascites and oedema

Patients are usually prescribed diuretics that ↑ urinary Na^+ output and ∴ induce negative Na^+ balance and fluid loss. Limiting dietary Na^+ intake will facilitate this and → a more rapid resolution, ↓ doses of diuretics being prescribed, and fewer associated complications. However, these advantages must be balanced against the negative effect that a low Na^+ diet may have on total nutrient intake. Two levels of restriction are generally advised.

No added salt diet (usually 80–100 mmol Na^+/day)

- Avoid salt at the table.
- Keep salt in cooking to minimum.
- Avoid high salt foods including most preserved or tinned items, such as bacon, ham, sausages, tinned and packet soups, stock cubes, tinned vegetables, meats and fish, crisps and similar savoury snacks.
- Cheese (cheddar-type) should be limited to 100 g/week.
- Fast foods and ready-meals should be avoided unless they are known to provide <30 mmol Na^+ per portion.

Low sodium diet (40 mmol Na^+/day)

- Avoid all salt at the table and in cooking (prepare food separately).
- Avoid food prepared outside home unless specific arrangements have been made.

- Avoid high salt foods listed above under 'no added salt', including all cheese, fast food, and ready meals.
- Restrict bread intake to two slices per day.
- Use salt-free butter or margarine.
- Breakfast cereals should be salt-free, e.g. Puffed Wheat, Shredded Wheat.
- Restrict milk to half a pint (300 ml) per day.
- Use pepper, vinegar, spices, herbs, and lemon for flavouring.
- Fried food may be more appealing than boiled/grilled.

Patients are rarely advised to restrict their intake to <40 mmol Na^+ although lower restrictions (~20 mmol/day) have been used in the past, and these have required substituting ordinary bread and milk with unpalatable, low-sodium alternatives. This should only be attempted under close supervision by a registered dietitian, and discontinued if energy and protein intakes fall below estimated requirements.

Other sources of sodium

- Antacids.
- Some antibiotics—check with pharmacist for suitable alternatives.
- Salt substitutes—most contain K^+ salts, but some also contain Na^+.
- Sodium bicarbonate.

Portal systemic encephalopathy

Traditional treatment of portal systemic encephalopathy (PSE) included the restriction of dietary protein on the basis that nitrogenous compounds of dietary origin were implicated in the pathogenesis. Re-evaluation of the evidence has shown that dietary protein restriction is not an effective treatment[1] and may even exacerbate encephalopathy.[2] In addition, it can lead to rapid deterioration in nutritional status because protein requirements are increased in chronic liver disease.

Nutritional management of portal systemic encephalopathy

- Sufficient protein to meet the estimated requirements should be provided (ESPEN recommendation in cirrhosis is 1.2–1.5 g/kg/day, see Table 28.2).
- Protein is best tolerated if spread out across the day. Eating several smaller meals and a bedtime snack is preferable to fewer large meals.
- Vegetable and dairy protein may be better tolerated than protein from meat and fish.
- A high fibre diet, if acceptable to the patient, can contribute to a short gastrointestinal (GI) transit time, minimizing the opportunity for absorbing nitrogenous compounds of potential concern.

[1] Soulsby, C.T., and Morgan, M.Y. (1999). Dietary management of hepatic encephalopathy in cirrhotic patients. *Br.. Med.. J.* **318**, 1391.
[2] Merli, M., and Riggio, O. (2009). Dietary and nutritional indications in hepatic encephalopathy. *Metab. Brain Dis.* **24,** 211–21.

Steatorrhoea and oesophageal varices

Steatorrhoea

Although ≥50% of patients with chronic liver disease have ↑ faecal fat excretion, relatively few have steatorrhoea. Those who do predominantly have biliary liver disease (e.g. primary biliary cirrhosis or primary sclerosing cholangitis) rather than parenchymal disease (e.g. 2° to alcoholic or viral damage) and will benefit from restricting dietary fat to tolerance (see 📖 'Low fat diet' Chapter 26, p. 580).

Dietary fat contributes to the palatability of food and also provides:

- energy;
- fat-soluble vitamins;
- essential fatty acids.

A low fat diet, i.e. <30% of dietary energy, is not recommended unless the symptoms of steatorrhoea are jeopardizing nutritional status or quality of life. Advice must be given to ensure that the diet remains adequate in all other nutrients, especially energy, fat-soluble vitamins, and essential fatty acids.

Steatorrhoea due to bile insufficiency cannot be treated with pancreatic enzyme replacement therapy, although this may have a role if there is concomitant pancreatic disease (see 📖 Chapter 27, p. 611).

Oesophageal varices

Bleeding oesophageal or gastric varices are a life-threatening complication of chronic liver disease. Patients remain 'nil by mouth' during active bleeding and until their condition stabilizes. Nutritional implications arise if the patient's intake remains inadequate for prolonged periods, either because s/he is 'nil by mouth' or because of fear of eating.

There is no evidence that eating rough food, e.g. toast or crisps, increases the incidence of re-bleeding although some patients may prefer to take softer foods temporarily, particularly if they have undergone repeated endoscopic treatment. Feeding via a naso-enteric tube is recommended if oral intake is inadequate even in the presence of oesophageal varices and is not associated with ↑ incidence of re-bleeding or mortality or ↑ length of hospital stay.[1,2]

[1] Plauth, M., Cabré, E., Riggio, O., *et al.* (2006). ESPEN guidelines on enteral nutrition: liver disease. *Clin. Nutr.* **25,** 284–94.
[2] Cabré, E., Plauth, M., Riggio, O., *et al.* (2007). Reply to Dr Andus' letter. *Clin. Nutr.* **26,** 273–4.

Non-alcoholic fatty liver and non-alcoholic steatohepatitis

Fatty liver and steatohepatitis are part of a progressive spectrum of conditions although progression is not inevitable:

- Non-alcoholic fatty liver (NAFLD)
  ↓
- Non-alcoholic steatohepatitis (NASH)
  ↓
- Fibrosis
  ↓
- Cirrhosis
  ↓
- Hepatocellular carcinoma.

Pathogenesis is poorly understood, but relates to obesity → ↑ insulin resistance → peripheral lipolysis →↑ free fatty acids → hepatic *de novo* synthesis of triglycerides → deposits of lipid in hepatocytes. A putative *HFE* gene mutation may mediate effects of obesity as not all patients with NAFLD are obese and only a proportion of obese people develop NAFLD.

Prevalence depends on diagnostic method and population, but is currently rising with increasing rates of obesity and potentially exceeds prevalence of viral hepatitis:

- *NAFLD*: 7–35% of adult population.
- *NASH*: 2.5–6% of adult population.

Prognosis for patients with NASH, survival is 95%, 90%, and 84% at 1, 3, and 10 years, respectively. Deteriorating health associated with NASH represents a considerable health burden due to the ↑ prevalence.

Treatment options have been described until recently as being 'limited'. Most trials have focused on pharmacological interventions (including vitamin E and folate derivatives) although reducing excessive weight is considered important. In the absence of any dietary interventions, but on the basis of other published evidence, nutritional priorities[1] in NAFLD include ↓ excessive body weight, ↓ glycaemic response, ↓ hyperlipidaemia and ↓ cardiovascular risk (see Box 28.3).

[1] Zivkovic, A.M., German, J.B., and Sanyal, A.J. (2007). Comparative review of diets for the metabolic syndrome: Implications for non-alcoholic fatty liver disease. *Am. J. Clin. Nutr.* **86**, 285–300.

Box 28.3 Dietary advice in NAFLD[1]

- Moderate energy restriction ~100–500 kcal/day by ↓ portion size
- Dietary energy from 47% CHO, 38% fat, 15% protein
- ↓ Total fat intake to reduce postprandial triglyceride response
- ↑ Monounsaturated fatty acid
- Include sources of n-3 fatty acid
- Low glycaemic index and high fibre
- ↓ Refined CHO
- Regular moderate exercise (2 miles x 3 per week at 60% cardiac reserve).

This advice is more explicit than, but compatible with, most current dietary guidelines including the Eatwell Plate (see 🕮 Chapter 2, p. 27).

Liver transplantation

Approximately 700 liver transplants are undertaken annually in the UK.

Pre-operatively Patients being worked up for transplantation should undergo a detailed nutritional assessment (Box 28.1) to establish their nutritional status and identify scope for nutritional support. Undernourished patients have a significantly greater risk of increased morbidity and mortality after surgery so implementing early pre-surgery nutritional support is vital.

Immediately post-operatively Post-operative nutrition support varies from centre to centre. Early feeding via a jejunally-placed tube has been shown to be a safe and effective method of feeding post-transplant patients associated with reduced infections. Parenteral nutrition is only indicated if the GI tract cannot be used for >5 days and, wherever possible, attempts should be made to feed via the gut.

Short-term recovery When oral intake resumes, patients should be encouraged to eat an unrestricted diet; transient hyperglycaemia due to medication does not require dietary intervention, but insulin may be needed. Attention to food hygiene[1] is important because of immunosuppression, although practice varies between centres with some advising the avoidance of high risk foods, e.g. unpasteurized dairy products and shellfish, whilst others advise no restrictions. The effects of immunosuppressive drugs, e.g. ciclosporin and tacrolimus, can be potentiated by consumption of large quantities of grapefruit (e.g. 1.5 kg marmalade/week[2]) with serious adverse consequences. However, these effects vary with format of grapefruit, i.e. fresh, juice, concentrate etc., and as a result, total avoidance is advised by some centres (see 📖 Chapter 38 'Drug-nutrient interactions', p. 738).

Medium to long-term Following liver transplantation, most patients gain weight, especially in the first 6 months. This is appropriate if correcting pre-transplant undernutrition, but >30% patients become obese at 3 years after surgery and up to 40% have hyperlipidaemia. Long-term cardiovascular risk is a major cause of post-transplant death and ∴ a concern. In addition, excessive weight gain after transplantation is associated with development of *de novo* non-alcoholic fatty liver disease in the graft with associated morbidity (see earlier pages in this chapter). General dietary advice, e.g. the 'Eatwell Plate' (see 📖 Chapter 2, p. 27), should be routinely made available to patients within the first 6 months and regular monitoring of body mass index and serum lipids used to identify those who need more specific guidance. Appropriate advice about increasing energy expenditure should also be included and statins may be routinely prescribed to help manage hyperlipidaemia.

[1] 🖱 For further information see Food Standards Agency, www.food.gov.uk.
[2] Peynaud, D., Charpiat, B., Vial, T., *et al.* (2007). Tacrolimus severe overdosage after intake of masked grapefruit in orange marmalade. *Eur. J. Clin. Pharmacol.* **63**, 721–2.

Chapter 29

Renal disease

Introduction

Classification

- *Acute kidney injury (AKI):* sudden, rapid deterioration of kidney function caused by injury or illness; often reversible.
- *Chronic kidney disease (CKD):* abnormality of the structure or function of kidneys, lasting >3 months; often progressive. Has been classified into stages 1–5 (Table 29.1)
- *Established renal failure (ERF);* a description of those who have reached stage 5 CKD and require renal replacement therapy (dialysis).

CKD is categorized into stages according to glomerular filtration rate (GFR).

Prevalence of chronic kidney disease

The prevalence of CKD is rising throughout the world in association with the obesity and diabetes epidemics. Figures from the USA estimate that the prevalence of CKD stages 3–5 has ↑ from 5.7% of the population in 1988–1994 to 8.1% of the population in 2003–2006. Increasing prevalence and incidence of CKD is also a particular problem in the developing world and for individuals of ethnic minorities (e.g. South East Asian and Chinese populations) residing in developed countries.

See Box 29.1.

Box 29.1 Contributing causes

Acute kidney injury

- Hypovolaemia (shock)
- Cardiac failure
- Acute glomerulonephritis
- Toxic reaction, e.g. drugs, poison
- Obstruction of urinary output, e.g. tumour, renal stone disease

Chronic kidney disease

- Diabetic nephropathy
- Hypertension
- Infection, e.g. chronic pyelonephritis or sepsis 2° to severe urinary tract infection
- Polycystic kidney disease
- Tumour, e.g. multiple myeloma, amyloidosis
- Familial, e.g. Alport's syndrome
- Unknown reasons

Table 29.1 Stages of chronic kidney disease

Stage	Description	GFR (ml/min/1.73 m^2)
1	Normal kidney function, but urinalysis, structural abnormalities or genetic traits indicate kidney disease	≥90
2	Mildly ↓ kidney function	60–89
3a	Moderately ↓ kidney function	45–59
3b		30–44
4	Severely ↓ kidney function	15–29
5	Very severe, or end stage kidney failure (also known as ERF)	<15 or on dialysis

Nutritional assessment

Nutritional assessment of each patient should be undertaken to form a global picture of the nutritional status of the individual.

This assessment should include;

- Biochemical and haematological data: interpretation of these results are shown in Table 29.2.
- Medical history including duration of diagnosis of CKD and all other co-morbidities.
- Diet history (See 🕮 Chapter 4, 'Individual assessment', p. 38).
- Psychosocial factors: in addition to the usual psychosocial influences on diet (housing, family, support networks, financial situation, and ability to cook) dialysis patients often suffer from loss of independence, depression and anxiety associated with chronic illness.
- Anthropometrics: in addition to height, weight and BMI, it is important to collect anthropometric data that will allow assessment of alterations in lean body mass that can be masked by fluid shifts and changing fat mass over time. Hand grip strength, mid-arm muscle circumference, skin fold thickness and even bioelectrical impedance analysis (see 🕮 Chapter 4, 'Body composition', p. 44) have all been employed to more accurately monitor CKD patients, particularly those on dialysis. Waist circumference can be useful for assessing cardiovascular risk (see Table 21.2).
- *Activity level.*
- *Physical examination* (See 🕮 Chapter 4, 'Individual assessment', p. 38).
- *Fluid status.*
- Subjective global assessment (SGA) has been found to be a useful adjunct to evaluate the nutritional status in CKD patients. The results have been found to be reproducible and a number of formats are available online. Contributing factors include weight changes, dietary intake and gastrointestinal (GI) side effects, physical examination and fluid status on a 7-point scale.

Table 29.2 Interpretation of biochemical and haematological data

Blood result (normal range)	Importance
Potassium (K^+) 3.5–5.0 mmol/l	Hyperkalaemia can result in cardiac arrhythmia or cardiac arrest, related to diet or other reasons (Box 29.3). ↓ values may result in general muscle weakness. Can indicate poor nutritional intake as K^+ is found in many foods.
Sodium (Na) 136–146 mmol/l	Critical for blood pressure control, muscle and neurological function. Not a direct indicator of dietary intake. Most often associated with fluid status.
Urea (Ur) 2.7–7.5 mmol/l	Derived from protein degradation, influenced by diet, hydration and urine production. ↑ blood urea indicates ↓ GFR, also influenced by type of protein consumed, degree of anabolism and dialysis adequacy. Data from urea kinetic modeling can be used indirectly to estimate the protein intake.

Table 29.2 (*Contd.*)

Blood result (normal range)	Importance
Creatinine (Cr) 55–110 mmol/l	Normal waste product of muscle breakdown, not usually affected by dietary protein. Normally ↑ in CKD. Used to calculate the GFR.
Albumin (Alb) 35–50 g/l	A good predictor of mortality in CKD, however requires careful interpretation: can be influenced by fluid imbalance or an acute phase response as indicated by ↑ CRP levels (trauma, surgery, or infection). The large body pool and long half-life (14–20d) renders albumin relatively insensitive to immediate changes in nutritional status.
Pre-albumin 18–38 mg/l	Shorter half-life and smaller body pool than albumin, is influenced by the acute phase response and anaemia. Levels are ↑ in CKD due to ↓ catabolism and excretion, volume changes. Not a useful nutritional marker.
Bicarbonate (HCO_3) 22–29 mmol/l	Reflects the degree of acidosis or alkalosis in the body. In CKD, kidneys are unable to maintain normal acid-base balance and HCO_3 may ↓. May also ↓ with diabetic keto-acidosis, diarrhea, hyperventilation, and lack of O_2, fever or starvation. CKD patients often treated with sodium bicarbonate.
C-reactive protein (CRP) <8 g/l	A non-specific positive acute phase protein that can be used to diagnose infectious and inflammatory diseases, associated with mortality in CKD. ↑ CRP is an indicator of inflammation and can result in ↓ albumin.
Protein catabolic rate (PCR) – calculated	In steady state, PCR correlates well with dietary protein intake. However, in catabolic patients, urea generated by muscle breakdown far exceeds that derived from diet. Conversely, anabolism may produce false ↓ PCR. As PCR is usually normalized for actual body wt, extremes in weight will affect nPCR.
Calcium (Ca) 2.2–2.6 mmol/l	Often corrected for albumin as albumin carries half the calcium in the blood. ↑ levels cause fatigue, difficulty thinking, ↓ appetite, pain, ↑ urination, thirst, constipation, nausea and vomiting. Can be a sign of cancer. ↓ levels are most commonly seen after a surgical parathyroidectomy.
Phosphate (PO_4) 0.78–1.40 mmol/l	↓ levels may indicate poor food intake or may result as part of the metabolic imbalances associated with re-feeding syndrome. ↑ levels are problematic in CKD (see 📖 this chapter, 'Mineral Bone Disease', p. 638).
Haemoglobin (Hb)	↓ levels cause anaemia, ↓ reduce quality of life due to its effect on mental function, exercise tolerance, fatigue, appetite, and sleep patterns. See 📖 Chapter 6, 'Iron', p. 128.
Lipid profile	↓ total plasma cholesterol levels are associated with ↑ mortality, possibly as indicator of ↓ nutritional intakes. See 📖 Chapter 23, 'Cardiovascular disease', p. 465.

Normal levels can differ between both laboratories and hospitals 🕈 Target ranges for many blood results for CKD patients can be controversial, you may want to check recent guidelines and local practice patterns for target ranges.

Malnutrition in renal disease

- 20–60% of all patients with CKD (stages 3–5) are malnourished.
- Poor nutritional status prior to initiation of dialysis is associated with poorer outcomes on dialysis.
- Despite its limitations as an indicator of nutritional status, low serum albumin levels are independently associated with ↑ risk of death in haemodialysis (HD), peritoneal dialysis (PD), and transplant patients.
- Malnutrition in CKD is multi-factorial. Insufficient oral intake due to poor appetite is often problematic.
- Other contributory factors include chronic inflammation, co-morbid conditions, metabolic acidosis and accumulation of uraemic toxins resulting in increased muscle catabolism and appetite suppression.
- Delayed start to dialysis and overzealous attempts to control protein intake in the pre-dialysis period can compromise nutritional status.
- Malnutrition is a particular problem in HD patients who have ↑ requirements, but often miss meals if dialysis coincides with meal times.

Nutrition support

Renal patients who are malnourished or unable to achieve an adequate intake from food should be advised to supplement their oral intake with high energy and/or high protein foods (food fortification) or proprietary products (see 📖 Chapter 25, 'Treatment of undernutrition', p. 512) or provided with enteral or parenteral nutrition support (see 📖, Chapter 25, 'Enteral feeding', p. 516 and 'Parenteral nutrition', p. 536).

- Care must be taken to ensure that supplements are compatible with other dietary restrictions.
- Fluids from supplements contribute to the daily fluid allowance. Where necessary, nutrient-dense supplements should be selected.
- If enteral feeding is required, attention must be given to the total fluid intake. Patients may need daily HD or more hypertonic PD exchanges.
- Gastrostomy feeding is increasingly being used in both HD and PD patients at home. In PD patients, a short period of peritoneal rest while the gastrostomy tube placement site heals is often instigated.
- Specifically formulated renal enteral feeds are available to provide fluid, electrolyte-restricted, nutrient-dense feeds (Table 29.3).
- Parenteral nutrition may be preferred in patients with impaired GI function:
  - energy requirements are best provided using a combination of both glucose and fat so as not to exceed the glucose oxidation rate 4–5g glucose/kg/day (including glucose derived from dialysate during continuous renal replacement therapy (CRRT);
  - excessive glucose is associated with ↑ risk of metabolic disturbances leading to ↑ CO_2 production and lipogenesis;
  - provision of fat should not exceed 1 g/kg/day in AKI;
  - routine use of low electrolyte parenteral nutrition regimens in patients with AKI receiving dialysis (especially CRRT) may cause electrolyte depletion, and necessitate administration of additional potassium, phosphate, or magnesium, or the use of standard parenteral formulae;
  - close monitoring of serum biochemistry is paramount, especially at the onset of feeding.

Table 29.3 Nutritional products widely used in renal disease (also see 📖 Chapter 25, 'Treatment of undernutrition', p. 512)

Type	Examples of prescribable brands
Energy supplements	
Glucose polymers	Caloreen (Nestlé), Polycal (Nutricia Clinical), Vitajoule (Vitaflo)
Fat	Calogen (Nutricia Clinical), Liquigen (SHS)
Combined	Duocal (SHS), Duobar (SHS), Pro-Cal (Vitaflo)
Protein powders	Casilan 90 (Heinz), Renapro (KoRa)
Oral Supplements	Ensure Plus (Abbott), Fortijuce (Nutricia Clinical), Fresubin 2 kcal Drink (Fresenius Kabi), Resource Dessert Energy (Nestlé) etc
Enteral/sip feeds	
Nutrient dense	Ensure Plus (Abbott), Fresubin HP Energy (Fresenius Kabi), Nutrison Energy (Nutricia Clinical)
Low fluid/electrolyte	Nepro (Abbott), Ensure Twocal (Abbott)

Intradialytic nutrition

- *Intradialytic parenteral nutrition (IDPN)* can be used to supplement energy and protein intakes during each HD session. Although it is expensive, it has the advantage of providing nutrients with minimal additional fluid and ensuring patient compliance. Short-term evaluation of IDPN suggests it is of benefit in some patients, but few studies have evaluated the long-term benefits. Metabolic studies suggest that IDPN can switch a patient from a catabolic to an anabolic state, and can ↑ serum albumin, dry weight, muscle strength. Only one controlled trial has been conducted and this showed no mortality benefits over enteral supplementation with sip feeds. In principle it can provide up to 1200 kcal and 75 g protein per session. Metabolic side-effects of IDPN, e.g. hyperglycaemia and GI symptoms require close monitoring.
- *Intra-peritoneal amino acids* have been used in continuous ambulatory peritoneal dialysis patients. Studies using 1.1% amino acid dialysate solutions to replace one of the usual daily glucose exchanges may show some improvement in nutritional status, especially in those patients with moderate to severe pre-existing malnutrition. However, it is expensive and only leads to a small improvement in nutrition. The exchange must be done at the same time as a meal to enhance amino acid uptake.

Nutritional considerations in chronic kidney disease

Mineral bone disease

As the GFR falls <60 ml/min, the kidney's ability to adequately regulate calcium and phosphate homeostasis is also reduced. This can result in secondary hyperparathyroidism, metabolic bone disease and ↑ calcification of the vasculature, known collectively as mineral bone disease (MBD). A chain of interrelated biochemical abnormalities occur in MBD (Table 29.4).

Management of mineral bone disease

Management intensifies with ↑ stages of CKD.

- ***Phosphate*** intake can be restricted to as little as 800–1000 mg/day. Emphasis should be placed on reducing phosphate from foods with low nutrient density e.g. fizzy drinks, processed cheese and pre-mixed dried baking products (e.g. cake mix). Many high phosphate foods are also good protein sources, so care should be taken not to compromise the nutritional status of the patient with excessive restriction.
- ***Phosphate binders*** can be used when it is not possible to control the phosphate with diet alone. These are available as various compounds, but all bind with phosphate in the GI tract to prevent systemic absorption with variable efficacy and GI side effects.
- ***Vitamin D*** deficiency is highly prevalent in the general population as well as in CKD. Controversy exists over the best type of vitamin D to prescribe. Either 25 vitamin D, 1,25 vitamin D or a precurser to 1,25 vitamin D can be used in CKD and will differ between centres.

Table 29.4 Abnormalities of mineral metabolism in CKD

Blood result	Abnormality in CKD
Phosphate	Inability to remove sufficient phosphate begins early in CKD, although serum phosphate levels are maintained in the 'normal' range until GFR<15 ml/min
Calcium	Calcium ↓ with decreased absorption due to a fall in vitamin D
Parathyroid hormone (PTH)	PTH ↑ gradually when GFR <60 ml/min, initially as a response to ↓ calcium and ↑ phosphate
Vitamin D	Reduced conversion of 25 vitamin D into its active form 1,25 vitamin D, begins when GFR <60 ml/min

Cardiovascular risk

Cardiovascular disease (CVD) accounts for as many as 50% of deaths in CKD. An individual with kidney disease is more likely to die of CVD than reach the stage of requiring dialysis. Traditional CVD risk factors account for only some this ↑ risk. CVD risk can be reduced with a combination of changes in diet, lifestyle, and physical activity.

Traditional modifiable CVD risk factors in CKD

- ***Hypertension:*** in addition to medical management, a no-added-salt diet (80–100mmol/day) is beneficial (see 🕮 Chapter 28, 'Ascites and oedema', p. 624).
- ***Hyperlipidaemia:*** renal patients present with ↑ cholesterol and ↑ triglycerides. Therapeutic diet and lifestyle changes could have benefits over and above pharmaceutical control of lipids (see 🕮 Chapter 23, 'Cardioprotective diet', p. 470).
- ***Weight management:*** see 🕮 'Weight management', p. 640.
- ***Lifestyle choices:*** particularly smoking and low physical activity levels.
- ***Diabetes:*** addressed in the section below.

CKD specific modifiable CVD risk factors

- ***Deranged mineral metabolism*** (as described above) is associated with calcification of the heart and blood vessels.
- ***Anaemia*** can contribute to left ventricular hypertrophy, see 🕮 Anaemia, this chapter, p. 639.
- ***Other*** Volume overload; inflammation and malnutrition.

Anaemia

Common in CKD, due to iron deficiency and/or a relative lack of erythropoietin, which develops when GFR falls <60 ml/min, and should be investigated if Hb <13 g/dl (♂ and post-menopausal ♀) and <12 g/dl (pre-menopausal ♀). Intravenous or oral iron can be given as first line treatment (Table 29.9) ferritin levels should not exceed 800 ng/ml. Epoetin, synthetic erythropoietin which stimulates erythropoiesis, can be used to maintain Hb levels between 10.5–12.5 g/dl.

Diabetes

Diabetes is the cause of renal failure in approximately 40% of CKD patients. The nutritional priorities in the diabetic with kidney disease are:

- Optimal glycaemic control (HbA1c <7.0% or <53 mmol/mol).
- Optimal management of blood pressure and proteinuria.
- Emphasis on management of modifiable CVD risks.
- Weight management.

Weight management

Where possible, obese patients (BMI>30 kg/m^2) should be encouraged to lower their body weight through diet and exercise. Weight loss goals should be realistic and tailored to the individual patient.

Reasons for weight management in CKD

- Increased risk of CVD, diabetes, hypertension and some cancers.
- Extra weight may interfere with a patient's ability to mobilize and lead an independent life.
- Longer HD sessions may be required to dialyse a heavier person.
- Being obese may ↓ the chance of receiving a kidney transplant.

Exercise

Patients at all stages of CKD should be encouraged to take regular exercise. It can be a challenge to recommend safe and appropriate ways to exercise if the patient is elderly or infirm although there are multiple resources available and physiotherapy consultation can support.

Table 29.5 Amino acid (protein) losses during dialysis

	Estimated amino acid (protein) loss
HD	1–1.5 g (6–9 g)/session
CRRT	1.5–2.0 g (9–12.5 g)/24 h

Nutrition in acute kidney injury

Nutritional status in AKI is usually influenced by the underlying aetiology of disease, pre-existing malnutrition, degree of catabolism, and prolonged hospitalization. Severely catabolic AKI patients usually require prompt nutritional support.

Continuous renal replacement therapy

Severe AKI can be treated with haemodialysis (HD) or CRRT. CRRT is often used in intensive care to treat critically ill, unstable patients with AKI who might be adversely affected by blood pressure changes and fluid restrictions associated with traditional HD. This slow, continuous dialysis therapy removes fluid and metabolites via diffusion and/or convection over a 24 h period.

Nutritional requirements in acute kidney injury

- *Energy:* similar to healthy individuals in non-septic patients. In sepsis, the BMR may be ↑ by up to 30%.
- *Protein:* Patients with AKI but no additional catabolic stress are usually able to maintain neutral or positive nitrogen balance. Those with sepsis, trauma, inflammation or multiple organ failure may have increased protein requirements. Catabolic patients may have a marked rise in blood urea nitrogen as nitrogen balance becomes negative. Urea nitrogen appearance can be measured to estimate total nitrogen balance. Protein requirements can be estimated using standard formulae, corrected for patient activity and underlying clinical condition (see 🕮 Chapter 25, 'Estimating requirements in disease states', p. 540). Dialysis-dependent patients require additional protein to replace losses during dialysis (Table 29.5).
- *Electrolytes and minerals:* conservatively managed patients usually require dietary restriction of potassium, sodium, and phosphate. Patients undergoing CRRT need a less restrictive diet, and many need full nutrition support. During CRRT many AKI patients develop hypokalaemia, hypophosphataemia, and hypomagnesaemia and require supplementation. These are important metabolic derangements, especially in ventilated patients, as electrolyte depletion can lead to increasing muscle weakness, alterations in acid–base balance, and to further nephrotoxicity (especially tubular damage). Daily monitoring is necessary. At different stages of AKI, supplementation or restriction can be necessary, often determined by the catabolic state of the patient and the modality of dialysis (e.g. intermittent or continuous).
- *Fluid:* not normally restricted on CRRT. Restrictions may be necessary if the patient is receiving HD.

Nutrition in chronic kidney disease stages 3 and 4

- An individual can stay at these stages of kidney disease for any time from a couple of months to decades.
- Ideally, they will be reviewed as an out-patient by their nephrologist or general practitioner thus providing ample opportunity for nutritional assessment and intervention along the continuum of their chronic disease management.
- A new patient requires full dietary assessment and subsequent nutritional advice should be tailored to the specific needs of the individual with a view to minimizing the risk of potential disease progression and related risk factors (e.g. CVD).

Nutritional requirements

- ***Energy*** requirements are normal, i.e. 30–40 kcal/kg ideal body weight (IBW)/day.
- ***Protein*** maximum intake 1 g/kg IBW/day (a mild degree of restriction), especially in patients consuming excessive protein or for the short-term management of uraemic patients awaiting the start of dialysis. Reducing protein intake below 0.75 g/kg IBW/day is not recommended, because although this is thought to ↓ uraemic symptoms and ↓ rate of decline in renal function, benefits are marginal when compared with ↑ risk of malnutrition. Protein intake may ↓ spontaneously as the GFR ↓ below <25 mL/min. It is ∴ more likely that patients nearing ERF will require nutrition support to achieve an adequate intake, rather than restrictions. Timely initiation of dialysis will ↓ extent of malnutrition.
- ***Phosphate:*** early restriction may help prevent renal bone disease, secondary hyperparathyroidism, and may slow progression to ERF. Phosphate containing foods are shown in Table 29.6.
- ***Potassium:*** restriction is required if consecutive K^+ levels run >5.5 mmol/L as is often the case with the use of ACE (angiotensin converting enzyme) inhibitors or ARBs (angiotensin receptor blockers) which reduce renal potassium excretion. Other non-dietary sources of hyperkalaemia are shown in Table 29.7. Strategies for reducing dietary K^+ are described in Box 29.4.
- ***Fluid:*** restrictions are usually only required in CKD if there is a marked ↓ urine output (<1000 ml/day), severe oedema, or a history of coronary heart failure. Fluid restriction may occasionally be necessary in poorly controlled diabetes.
- ***Sodium intake:*** a mild restriction of 80–100 mmol (no-added-salt diet: see Chapter 28, 'Ascites and oedema', p. 624) is recommended.

Chronic kidney disease in the elderly

Nephron mass and ∴ GFR deteriorates with normal aging. Many elderly individuals in whom CKD is diagnosed may never require dialysis. This should be taken into account before advising on overzealous dietary restrictions, particularly in the very elderly.

Table 29.6 Phosphate containing foods

Drinks	Some fizzy drinks including most cola, beer, hot chocolate, milk shakes, milk and other milky drinks.
Dairy products	Processed cheese, red cheddar, custard, and yogurt.
Meat	All meat and fish contain some phosphate but are necessary to meet protein requirements. Avoid high fat, high sodium processed and canned meats. Avoid liver and other organ meats.
Fish	Oily fish and shellfish are significantly higher in phosphate than white fish.
Other	Dried beans and peas. Bran products particularly all-bran and bran buds. Pumpkin seeds and sunflower seeds. Nuts and peanut butter. Whole grain products.
Phosphate additives	Phosphate containing additives in various formats have 100% bioavailability. These are added to a variety of commonly used foods including processed meat and cheese, freeze dried foods, bakery products and beverages. Phosphate is not reported in the nutritional information, but should be listed in the ingredients list. E101, E106, E339-343, E450-452, E540-545, E1410, E1412, E1414, and E1442 all contain phosphates.

Table 29.7 Non-dietary causes of hyperkalaemia

Metabolic factors	Hyperparathyroidism, acidosis, insulin insufficiency.
Drugs	K^+-containing drugs, e.g. penicillin, senna.
	Drugs affecting K^+ excretion, e.g. ACE inhibitors, ARBs, β-blockers, non-steroidal anti-inflammatory drugs (NSAIDs).
	K^+-sparing diuretics, e.g. amiloride, spironolactone.
Cellular trauma	Haemolysed blood sample, post-blood transfusion, infection, gastrointestinal haemorrhage, crush injury, gangrene.
Constipation	Reduced gut excretion.
Dialysis	Inadequate dialysis.

Nephrotic syndrome

Characterized by proteinuria >3 g/day, hypoalbuminaemia and generalized oedema. Hyperlipidaemia, clotting problems and hypertension are also often observed. There are a number of causes (Box 29.2).

Box 29.2 Causes

- Focal segmental glomerulosclerosis (FSGS), 30%
- Membranous nephropathy (MN), 25%
- Diabetes, 20%
- Minimal change nephropathy (MCN), 10%
- Amyloidosis 4–10%
- Others include systemic lupus erythematosus, IgA nephropathy, toxic glomerulopathy, e.g. caused by gold or penicillamine

Treatment

Aims to control oedema, reduce proteinuria, and treat complications that arise including infections, hyperlipidaemia, and clotting problems. Diet therapy has a role to play in several of these areas (Box 29.3).

Oedema A no-added-salt diet (80–100 mmol/d) can potentiate the antihypertensive and antiproteinuric effects of ACE inhibitors. Fluid restriction may be necessary, depending on the response to diuretics and other treatments such as albumin infusions. ❗ Oedema can mask signs of wasting and malnutrition.

Proteinuria/hypoalbuminaemia 0.8–1 g protein/kg ideal body weight/day is recommended. Proteinuria is targeted with ACE inhibitors, strict blood pressure control and good diabetes control. A high protein diet is not advised as this causes ↑ permeability and hyperfiltration in the glomerular basement membrane which could exacerbate proteinuria.

Hyperlipidaemia Incidence of myocardial infarction is 5–6 x greater in nephrotic syndrome (NS). Lipid lowering agents and bile acid sequesters are used to control cholesterol and triglycerides. ↓ protein vegetarian diets based on soy protein can lower cholesterol along with standard lipid lowering advice. Fish oils can ↓ triglycerides.

Thromboembolism 10–30% of adults with NS develop emboli, a ↓ protein diet has been shown to improve fibrinogen levels.

Infections Iron, copper, zinc, and vitamin D are lost in proteinuria. ↓ Vitamin D can result deranged calcium metabolism and supplementation may be necessary. A balanced diet will help maintain micronutrients.

Box 29.3 Summary of diet in nephrotic syndrome

- *Energy:* 'normal requirements', i.e. 30–35 kcal/kg IBW/day
- *Protein:* 0.8–1 g/kg ideal body weight per day
- *Fat:* <30% total kcal, low saturated fat, encourage MUFA, PUFA
- *Micronutrients:* ensure adequate intake, monitor for signs vitamin D deficiency
- *Sodium:* no-added-salt, i.e. 80–100 mmol/d
- *Potassium:* monitor as serum levels may ↑ with ACE inhibitors.

Nutritional requirements in dialysis

Dialysis is usually initiated once GFR drops <10 ml/min (or <15 ml/min in people with diabetes) and the patient has symptoms of uraemia. If a patient chooses conservative care rather than dialysis, their nutritional objectives should be re-assessed to take this into account. Appetite and food intake often improve gradually once dialysis has begun and uraemic symptoms are alleviated. However, some patients may have ↑ malnutrition due to adverse effects of dialysis. Nutritional requirements for dialysis are shown in Tables 29.8 and 29.9. Where possible, general healthy eating guidelines are recommended, e.g. 50% total energy from complex carbohydrates, high fibre (non-starch polysaccharides (NSP)) and 30–35% energy from fat (predominantly poly- and mono-unsaturated fatty acid sources with low saturated fats). If intake is poor, healthy eating guidelines should be relaxed to help achieve nutritional adequacy.

Nutritional requirements in peritoneal dialysis

Energy Overweight patients may require energy restriction to compensate for the additional energy absorbed from dialysate (if the dialysate contains glucose). Initial advice should concentrate on reducing excessive intake of fat and sugar. High use of hypertonic dialysate to control fluid balance will increase energy intake. Fluid management and sodium restriction should then be emphasized.

Protein requirements are increased in PD due to dialysate losses. High protein requirements are often difficult to achieve by diet alone, particularly in those with poor appetite, or during and after peritonitis. Specific targets for protein-rich foods and snacks, and use of prescribed protein supplements may be required.

Phosphate restrictions and phosphate binders are often required based on blood results.

Potassium Dietary restriction is rarely needed in PD because of continuous clearance. Hypokalaemia is often a problem requiring ↑ K^+ diet.

Fluid PD patients usually lose water (up to 2 l/day) by ultrafiltration (UF) depending on the glucose concentration of dialysate. This is particularly important in anuric patients. The volume of UF may be added to the daily fluid intake allowance. Long-term use of hypertonic solutions may cause damage to the peritoneal membrane, producing hyperpermeability, loss of peritoneal integrity and weight gain. Use of higher glucose concentration dialysate should be minimized especially in obese patients, those with diabetes or hypertriglyceridaemia. These patients need more stringent Na^+ and fluid restrictions. Icodextrin dialysate may be beneficial.

Fibre A high intake of soluble NSP is of particular importance in PD (see 📖 Chapter 5, 'Carbohydrate', p. 72). The avoidance of constipation is extremely important for optimal functioning of this type of dialysis.

Table 29.8 Daily nutritional guidelines for patients on HD and PD

	HD	PD	Main food sources
Energy	30–40 kcal/kg IBW†	30–40 kcal/kg IBW†	Cereals, bread, rice, pasta, potato, sugar, fats
Protein	>1.2 g/kg IBW†	>1.2 g/kg IBW†	Meat, fish, eggs, pulses, dairy
Potassium	0.8–1 mmol/kg	Restrict only if hyperkalaemic: 1 mmol/kg	Fruit, vegetables, fruit juice, nuts, coffee, chocolate, crisps
Phosphate	<1000 mg	<1200 mg	Processed meat and cheese, shellfish, fish with bones, fizzy drinks and additives
Sodium	80–100 mmol (no added salt)	80–100 mmol (no added salt)	Table salt, smoked/cured foods, tinned and packet foods, ready meals
Fluid	500 ml + previous day urine output	500 ml + previous day urine output + ultrafiltration	Drinks, gravies, sauces, soups, jelly, yogurt

IBW, Ideal body weight.

†Nutrition in CKD clinical practice guidelines, UK renal association 5th edition 2009–2010.

Table 29.9 Recommendations for vitamin requirements in dialysis

Thiamin (B_1)	1.5 mg	Pantothenic acid	10 mg
Riboflavin (B_2)	1.7 mg	Biotin	300 µg
Pyridoxine (B_6)	10 mg	Folic acid	1000 µg
Cobalamin (B_{12})	6 µg	Ascorbic acid (C)	60 mg
Nicotinamide	20 mg	Iron sulphate	150 mg

Nutritional requirements in haemodialysis

Energy Some patients have difficulty achieving recommended intakes ∴ will need advice aimed at increasing energy intake using energy-rich foods and/or prescribed supplements.

Protein Patients generally have sufficient intake of protein once energy requirements are attained. Combined energy and protein supplements may help if intake from food is inadequate.

Fluid HD patients are usually anuric and often require severe fluid restriction, limiting intake to the daily volume of urine plus 500ml (i.e. to meet insensible losses). Many strategies exist to aid patients with these difficult restrictions (Table 29.10). Interdialytic fluid gain should be monitored by measuring body weight and aiming for a maximum gain of 2kg or 3% dry body weight between HD sessions.

Sodium intake should be restricted to a no-added-salt diet. Patients should be educated about salt being the major drive to thirst, as restricting Na^+ intake is likely to lead to better fluid (and blood pressure) control than attempting to restrict fluid intake alone.

Phosphate Clearance of phosphate is not particularly effective with conventional HD. Daily dialysis and nocturnal dialysis achieve significantly better phosphate clearance, enabling some patients to relax their dietary restrictions and reduce their intake of phosphate binders.

Potassium Dietary K^+ restriction is normally required; the level of restriction is partly dependent on residual renal function. Non-dietary causes of hyperkalaemia (Table 29.7) should be excluded. Restriction of dietary K^+ intake is generally staged following ongoing review of blood levels. K^+-containing foods are shown in Box 29.4.

Vitamin deficiencies can occur in HD due to dialysate losses, abnormal metabolism and dietary restrictions. Overt deficiency is rare. Most renal units prescribe a routine supplementary dose of the water-soluble vitamin B group, vitamin C and folate (Table 29.9), although good evidence for supplementation is lacking. Fat-soluble vitamins A and E are not routinely prescribed due to the risk of hypervitaminosis.

Minerals and trace elements Of the 14 essential minerals and trace elements (see 🕮 Chapter 6, 'Minerals and trace elements', p. 120), deficiencies in Zn, Cu, Mn, and Cr have been reported in CKD, mostly due to dietary restriction and drug interactions. Deficiency should be confirmed before starting supplementation.

Table 29.10 Strategies for achieving fluid restriction

Reduced fluid intake	Use small volume cup for drinks.
	Suck ice cubes and ice lollies.
	Take tablets with food (unless otherwise directed).
↑ Awareness	Education about fluid content of certain foods, e.g. jelly, custard, soup, ice-cream, yogurt, dhal.
	Measuring jug tally, e.g. fluid intake is monitored by taking required liquid throughout the day from a jug/bottle initially containing the desired daily volume.
Thirst prevention	Reduce salt intake.
Techniques	Take sugar-free sweets or chew gum.
	Use of fruit (within K^+ restriction if applicable).
Regular mouth care	Use of mouth wash, lip salves, etc.

Box 29.4 Strategies for achieving K^+ restrictions

Limit K^+ rich food sources

- *Fruits:* apricot, banana, rhubarb, avocado, melon, kiwi, mango, nectarine, large orange, papaya, pomegranate, all dried fruit
- *Vegetables:* spinach, mushroom, beetroot, broccoli, brussel sprouts, carrot, spinach, tomato, jacket or instant potato, chips (if not parboiled), crisps, baked beans, kidney beans, lentils, other beans and pulses
- *Drinks:* fresh fruit juices, coffee, drinking chocolate, malted drinks, blackcurrant cordials
- *Other:* salt substitutes, humus, nuts, peanut butter, chocolate, milk, yogurt, evaporated and condensed milk, yeast extract, liquorice

Alter cooking methods (as K^+ is very water soluble)

- Use large volumes of water for boiling vegetables (double boiling is no longer recommended as the second boil does not remove a significant amount of additional K^+ but makes many vegetables inedible)
- Chop potatoes and other large vegetables into small to chunks to increase the surface area for K^+ removal
- Soak beans and legumes for at least 24 h prior to cooking and change the water every 4 h
- Rinse vegetables in warm water for a few seconds prior to boiling
- Parboil vegetables before adding them to stews, soups, etc.
- Avoid pressure cooker and microwave cooking (except for re-heating previously cooked food)
- Limit portion size and quantities of fruit and vegetables.

Renal transplantation

Renal transplantation is considered to offer the patient with ERF the best chance of rehabilitation and good quality of life. A patient may have spent a couple of years in the pre-dialysis stage and then several years on dialysis before receiving a transplant. Even with careful management, they will show signs of the long-term metabolic effects of CKD, including:

- anaemia;
- mineral bone disease;
- muscle wasting;
- cardiovascular disease.

Immunosuppressive therapy, used to prolong the life of the transplanted kidney, can exacerbate all of the above conditions as well as creating additional problems (see Box 29.5).

Box 29.5 Immunosuppressive treatment can have many side-effects

- Protein hypercatabolism
- ↑ Appetite leading to obesity
- Hyperlipidaemia
- Glucose intolerance/↑ risk of diabetes (~20% of patients)
- Hypertension
- Hyperkalaemia
- Interference with vitamin D metabolism
- ↑ Cancer risk
- ↑ Infection risk (opportunistic viral and bacterial infections that may ↓ appetite and ↑ nutrient requirements)
- Gum hypertrophy.

Immediately post-transplant

Nutritional care should be the same as for any other post-surgical patient: monitoring blood and urine biochemistry and urine volume, ensuring the return of normal gut function and appetite and meeting requirements with supplements if necessary. The rate at which biochemistry and urine volume return to normal can vary (from a couple of days after surgery to several weeks) and needs to be monitored closely. Treatment varies accordingly from fluid and electrolyte (Na^+ and K^+) restrictions to intravenous support if urine output is excessive and serum electrolyte levels drop below normal.

❗ Dehydration at this stage can damage the new kidney.

Once kidney function has stabilized

The main aims of dietary therapy are to encourage a healthy balanced diet and reduce the risk factors for cardiovascular disease. The incidence of obesity can ↑ dramatically post-transplantation and dietary advice should be given to the patient prior to hospital discharge with regular follow up in outpatient clinics in order to prevent excessive weight gain. Advice should include healthy eating, exercise and other lifestyle improvements

including stress management, avoidance of smoking and exposure to too much sun. As the patient is more susceptible to infections, some advice on food hygiene[1] and safe handling of food is also useful. Barriers to eating a healthy diet may be due to the habits formed whilst adhering to the previous dietary restrictions. Patients who have been on a K^+ restriction may be reluctant at first to ↑ intake of fruit, vegetables and other foods that may have been restricted on the dialysis diet. Conversely, there may be a temptation to over consume some previously restricted foods and this, combined with steroid use, a renewed *joie de vivre* and improved appetite, could contribute to a rapid ↑ in body fat.

Longer-term

Weight management and improvement in lipid levels have been achieved with diet and exercise interventions. These interventions will help reduce the risk of metabolic syndrome and diabetes, although many patients also need medication to control blood lipids. Improved muscle and bone strength may also result from diet and exercise advice. Vitamin D and calcium supplements may be required to improve bone strength (Box 29.6).

Box 29.6 Summary of nutritional management in renal transplantation

- Monitor biochemistry, especially cholesterol, triglycerides, glucose, K^+, bone minerals, PTH, haemoglobin
- Monitor blood pressure control
- Aim for healthy body mass index
- 'The Eatwell Plate' is an appropriate food model to use
- Emphasize eating a good variety of fruit and vegetables
- Encourage high fibre foods
- Encourage fish particularly oily fish, lean meats, and pulses
- Foods high in sugar, saturated fat, and salt should be used sparingly
- Encourage ↓ fat dairy products
- Ensure Ca^{++} requirements are met
- Advise alcohol within usual safe drinking limits
- Be aware of good food hygiene practices
- Encourage physical activity and regular exercise
- Avoid smoking
- Avoid too much sun exposure.

[1] For further information see Food Standards Agency, London, www.food.gov.uk.

Ethnic minority patients with chronic kidney disease

Vegetarian or vegan diets generally provide less protein and may be higher in potassium and phosphate. Protein supplementation may be required, especially in PD.

Diets of ethnic minorities

(See 📖 Chapter 16, 'Minority ethnic communities', p. 306)

- *Fasting:* adequate energy and protein intakes are more difficult to achieve during fasting. For Hindus, pure foods, including fruit, nuts, yogurt, and milk, eaten during this time may be high in potassium and phosphate. Medications may be omitted inappropriately during a fast.
- *Traditional cooking methods:* cooking methods for curries, stews, and stir-fries may conflict with low potassium cooking techniques. Cooking utensils may contain iron, aluminium, or other trace elements, which can accumulate although this is rare.
- *Traditional food items* may be rich in the following:
  - *potassium*: spinach (sag, callaloo), karela, potato pakoras, plantain, yam, cassava, sweet potato, okra, banana, mango, paw-paw, nuts, coconut, sweetmeats, chevda;
  - *phosphate*: lassi, raita, Indian tea, nuts, sweetmeats;
  - *sodium*: chevda, pickles, salt fish and pork, soy sauce, monosodium glutamate.
- *Toxicity:* rarely, some foods may be toxic in renal failure, for example, star fruit (*Averrhoa carambola*), which is eaten especially in Asia, causes severe intoxication in some renal patients leading to intractable hiccups, agitation, muscle weakness, confusion, fits, and can be fatal.

Renal stone disease

Incidence

Renal stone disease (nephrolithiasis or renal calculi) will affect 10% of men and 5% of women in their lifetime. Significant differences in incidence are seen in different populations, suggesting genetic and/or environmental influences such as diet and climate. Populations of the developed world are more at risk with greater incidences seen in Whites > Asian and Hispanic > Black.

Stone formation

- Stone formation can occur anywhere in the kidney, ureter, or bladder.
- The size of stone can vary from microscopic to the large 'staghorn' calculi and can lead to kidney failure if the kidney or urinary tract becomes obstructed.
- Stones vary in composition (Table 29.11).
- ↑ Urinary concentration of calcium, sodium, oxalate, urate and citrate promote stone formation and ↑ concentration in urinary magnesium, pyrophosphate, citrate and nephrocalcin inhibit stone formation.
- Stone formation can also be linked to congenital kidney abnormalities, short bowel syndrome or recurrent infection with urease positive organisms.
- About 50% of stone formers excrete ↑ urinary calcium, >7.5 mmol/day in men or >6.2 mmol/day in women. Hypercalciuria may be due to ↑ absorption of calcium from the gut, calcium resorption from the bone, or ↓ ability of the kidney to reabsorb calcium.
- Stone formation is exacerbated by ↓ urine output volume due to low fluid intake or ↑ losses e.g. sweat and GI tract, e.g. intestinal failure.

Dietary treatment of renal stone disease

The diet of people in the affluent world has been scrutinized with respect to intake of animal protein, sodium, calcium, oxalate and purine. It appears that even with identical dietary intakes, people with a tendency to form stone will form larger crystals than non-stone formers.

- *Fluid:* 2–3 l/day (250 ml every 4 waking hours + 250 ml at meals).
- *Protein:* 1 g protein/kg ideal body weight.
- *Calcium:* restriction is not advised, 700–800 mg/day; hypercalciuria can be ↓ by ↑ alkali load (i.e. fruit/vegetables), dietary fibre, K^+ and PO_4;
- *Sodium:* moderate reduction 90–100 mmol/day.
- *Oxalates:* only 10–15% of urinary oxalate is derived from dietary intake while the rest is formed endogenously (from vitamin C and glycine metabolism); if 24 h urinary oxalate is >440 mmol, check for ↑ oxalate foods (Table 29.12) and megadose intakes of vitamin C; pyridoxine supplements may ↑ stone formation.
- *Purines:* if 24 h urinary uric acid is >4 mmol check for ↑ purine containing foods (Table 29.13); orange juice may help prevent formation of uric acid stones as it is high in citrate without containing too much oxalate.
- *Other:* ↑ intake of potassium, magnesium, fibre, fruit and vegetables is associated with ↓ stone risk; ↑ refined carbohydrate may ↑ risk.

Table 29.11 Main types of renal stone disease

Composition	Incidence (%)	Possible causes
Calcium oxalate: up to 50% may contain calcium hydroxyl phosphate	75	Idiopathic hypercalciuria 1° Hyperparathyroidism ↓ Urine citrate Hyperoxaluria Hyperuricosuria
Magnesium ammonium phosphate (struvite or triple phosphate)	10–20	Bacterial infection
Uric acid	5	Low urine pH Hyperuricosuria
Cystine	1–2	Cysteinuria

Table 29.12 Oxalate-rich foods—bio-availability varies

Drinks	Beer, black tea, cocoa, juices from high oxalate fruits, instant coffee powder, Ovaltine
Fruit	Blackberries, blueberries, gooseberries, kiwi, raspberries, rhubarb, strawberries, tangerines
Vegetables	Beetroot, celery, green beans, leeks, okra, parsley, runner beans, spinach, sweet potato, watercress, yam
Legumes	Baked beans, soy products, e.g. tofu
Grains	Wheat germ, bran
Nuts & seeds	Almonds, cashews, peanuts, pecans, sesame seeds, sunflower seeds
Other	Plain chocolate, soy sauce

Table 29.13 Purine-rich foods

Meat	Liver, kidney, brain, heart, goose, partridge
Fish	Anchovies, crab, herring, mackerel, mussels, roe, sardines, scallops, shrimps, sprats, whitebait
Other	Yeast, meat extract, e.g. Bovril, Oxo

Useful websites

Guidelines

- The Renal Association clinical guidelines. Available at: http://www.renal.org/Clinical/GuidelinesSection/NutritionInCKD.aspx
- DH (2004) National Service Framework for Renal Services. Available at: http://www.dh.gov.uk/en/Healthcare/Longtermconditions/Vascular/Renal/DH_4102636
- National Kidney Foundation—Kidney Disease Outcomes Quality Initiative (KDOQI): Available at: http://www.ajkd.org/content/kdoqiguidelines

Patient information

- Kidney Research UK. Available at: http://www.kidneyresearchuk.org/health/health-information.php
- The Kidney Patient Guide. Available at: http://www.kidneypatientguide.org.uk/site/contents.php
- The Blood Pressure Association (includes information on exercise for those with ↓ mobility). Available at: http://bpa.jamkit.com/Home

Chapter 30

Respiratory disease and cystic fibrosis

Respiratory disease

Asthma

Pathogenesis Evidence suggests that sub-optimum intakes of antioxidant micronutrients, particularly vitamins A and C, contribute to the development of asthma. This is supported by studies showing that people consuming a diet rich in fruit and vegetables have better respiratory health. Furthermore, good quality intervention studies are required to confirm these dietary benefits. In the meantime, a diet compatible with the 'Eatwell Plate' (see 📖 Chapter 2, p. 27) may help reduce risk. Obesity is also associated with ↑ risk of asthma, probably mediated through systemic and airway oxidative stress. Weight loss has been shown to improve respiratory symptoms in overweight patients. An ↑ sodium intake is associated with airway reactivity and ↑ symptoms in patients with asthma; an excessive salt intake should ∴ be avoided.

Food allergy Cow's milk has anecdotally been linked to asthma but objective testing suggests that diet and food allergy is only important in a minority of individuals; in these, food avoidance can improve symptoms and reduce drug therapy and hospital admission (see 📖 Chapter 37 'Food hypersensitivity', p. 730). There is no evidence that feeding infants soya-based, rather than cow's milk formula reduces the risk of having asthma; breastmilk remains the feed of choice for all babies for at least the first 6 months of life (see 📖 Chapter 13 'Breast versus bottle feeding', p. 242).

Chronic obstructive pulmonary disease

Patients with chronic obstructive pulmonary disease (COPD) are frequently malnourished (prevalence 25–80%). This is of concern because undernutrition is associated with poor respiratory function and ↑ susceptibility to infection and, specifically in COPD, is associated with poor prognosis (relative risk of low compared with high BMI in patients with severe COPD is 7.1 [95% CI 3.0–17.1]). Instinctively, providing nutritional support seems an appropriate mode of treatment, although one recent systematic review concluded that nutritional support had no significant effect on anthropometric measurements, lung function, or exercise capacity in patients with stable COPD. However, a wider review of evidence in hospital and community-based COPD patients shows that oral nutrition support can have beneficial effects on respiratory and skeletal muscle strength, walking distance, and well-being in underweight patients who gain >2 kg and, importantly, is not associated with any detrimental effects. Current clinical guidance[1] based on grade D evidence (non-analytical studies and expert opinion) advises that:

- Body mass index (BMI) should be calculated in patients with COPD.
- Patients with BMI <20 or >25 kg/m^2 should be referred to a dietitian.
- If BMI is <20 kg/m^2, patients should be given nutritional supplements to ↑ energy intake and be encouraged to take exercise.
- In older patients attention should also be paid to changes in weight, particularly if the change is more than 3 kg.

[1] NICE (2010). Clinical Guideline 101 Chronic obstructive pulmonary disease http://guidance.nice.org.uk/CG101/Guidance/pdf/English.

Lung cancer

Lung cancer is the second most common malignancy in the UK with an annual incidence of ~40,000. Smoking contributes to 90% of cases. A higher intake of fruit and vegetables has a protective effect which is attributed to their antioxidant content. Trials of antioxidants vitamin A and β-carotene supplements do not yield the same benefits and are, in fact, associated with ↑ incidence of lung cancer and ↑ mortality. Trials of antioxidant vitamin E have demonstrated no positive effects. Eating fruit and vegetables, rather than taking supplements, is thus recommended (see 'Eatwell Plate', in 📖 Chapter 2, p. 27). ❗ Large doses of antioxidant supplements should be discouraged.[2]

Patients with lung cancer are frequently undernourished, especially in the more advanced stages. Nutrition support should be considered in the context of their treatment and prognosis (see 📖 Chapter 24 'Cancer and leukaemia', p. 487).

Lung transplantation

Approximately 150 lung and heart-lung transplants are undertaken in the UK each year. Patients with end-stage lung disease are frequently malnourished. Those with a BMI <17 or >27 kg/m^2 have a greater chance of dying at 90 days after transplant compared to those with BMI between 17 and 25 kg/m^2. Pre-surgical nutritional support has been shown to be effective in increasing body weight in underweight patients; nutritional assessment and advice before transplant may help improve outcome. Diabetes and osteoporosis are common problems following transplantation and pre-emptive dietary advice may help manage these (for comparable advice, see 📖 Chapter 28 'Liver transplantation', p. 630).

Tuberculosis

Worldwide, ~9 million new cases of active tuberculosis (TB) are diagnosed each year and approximately 1.3 million people died from TB in 2008. The prevalence of TB has been increasing in the UK since 1990 after decades of decline[3] (2009: ~9000 individuals diagnosed in the UK, 41% in London, 53% pulmonary TB, 73% born outside UK; 60% aged 15–44 years). Undernutrition is a risk factor for developing TB and weight loss is regarded as a classic symptom of the disease. Patients who are malnourished are at greater risk of dying than those who are not. Vitamin D influences immune response and deficiency is associated with ↑ risk. Patients with TB and those at risk do not require a special diet, but would benefit from a well-balanced diet providing an adequate intake of all macro- and micronutrients.

[2] Cranganu, A., and Camporeale, J. (2009). Nutrition aspects of lung cancer. *Nutr. Clin. Pract.* **24,** 688–700.

[3] NICE (2011). Clinical Guideline 117 tuberculosis, http://guidance.nice.org.uk/nicemedia/live/13422/53638/53638.pdf.

Cystic fibrosis

Approximately 8500 people in the UK have cystic fibrosis (CF), an autosomal recessively inherited disorder that affects the exocrine glands leading to pancreatic insufficiency and chronic lung disease. Weight loss and undernutrition are associated with a worse clinical outcome.

Causes of weight loss and undernutrition in CF

Impaired nutrient absorption Malabsorption occurs in ~90% of patients with CF. Inadequate secretion of pancreatic enzymes should be treated by replacement therapy but not dietary fat restriction (see 📖 Chapter 27, 'Pancreatic enzyme replacement therapy', p. 616). If adequate pancreatic enzymes are taken, there is no need to limit dietary fat in almost all patients. ❗ Fat restriction will have detrimental consequences because of the associated restriction in energy intake. However, in a very small minority of cases where steatorrhoea cannot be controlled adequately in spite of appropriately taken high dose pancreatic supplements, a modest fat restriction should be tried *providing* that an adequate energy intake is maintained and the patient is closely monitored.

Increased requirements Energy needs increase due to ↑ costs of respiration. It is estimated that energy requirements are 120–150% of normal. Protein requirements are also likely to be ↑ as a result of ↑ nitrogen losses via the gut and sputum; an intake of 120% of the reference nutrient intake is recommended (see Appendix 6, 'Dietary reference values', p. 777). Patients ∴ need to consume ↑ energy, ↑ fat, ↑ protein intake plus micronutrient supplementation, see 📖 'Nutritional management', p. 660.

Poor food intake Appetite may be poor due to tiredness and repeated chest infections. ↑ respiratory tract secretions may limit the consumption of some supplement drinks. The nutrient density of the diet should be considered to ensure that requirements are met within the limited quantity of foods consumed.

Nutritional management

This should include the following.

- Regular review by a registered dietitian with experience in this area; nutrient intake is significantly greater in CF patients when they are reviewed at least annually by a dietitian.
- Oral supplements. While some patients may be able to consume sufficient ordinary food to meet their needs, others will benefit from home-made or commercial supplements. (📖 Chapter 25 'Treatment of undernutrition', p. 512).
- Overnight tube feeding. This may be useful for patients who are unable to maintain an adequate oral intake in the long-term or for shorter periods following an exacerbation of respiratory problems. It is particularly beneficial in children where overnight feeding for 6 months is associated with improved nutritional status and catch-up growth.
  - Nasogastric tubes can be passed nightly or a gastrostomy inserted, preferable by endoscopy (see 📖 Chapter 25 'Routes for enteral feeding', p. 518). Feeding regimens should take account of the higher requirements in CF and aim to provide 30–50% of energy needs (check that oral intake is able to provide the rest).

- Energy-dense feeds may be useful, but otherwise general feeding guidelines relevant to the age of the patient should be followed.
- Pancreatic enzyme replacement may be given before feeding commences, but fat content is often tolerated well because the feed is delivered slowly over several hours.
- Insulin may be required if feeding results in hyperglycaemia.
- Encouragement to eat during the day, i.e. between overnight feeds, will help ↑ resumption of oral intake and maintenance of normalcy.

- Fat soluble vitamin status should be checked at least annually in all patients, i.e. plasma levels of vitamins A, D, & E plus prothrombin time to assess adequacy of vitamin K. Most patients will require supplementation, especially in pancreatic insufficiency (Table 30.1).

Table 30.1 Recommendations for daily intake and monitoring of fat soluble vitamins in cystic fibrosis

Vitamin	Age	Intake	Monitoring
A	0–12 months	510 [1,500]	Serum retinol (deficiency <20 μg/dl)
μg [IU]	1–3 year	1,700 [5,000]	Retinol binding protein
	4–8 year	1,700–3,400 [5,000–10,000]	Zinc level
	>8 year/adult	3,400 [10,000]	
D	0–12 months	10 [400]	Serum 25 (OH) vitamin D in late autumn/winter
μg [IU]	>1 year	10–20 [400–800]	
E	0–12 months	40–50	Serum α-tocopherol
mg	1–3 year	80–150	
	4–8 year	100–200	
	>8 year/adult	200–400	
K	0–12 months	0.3–0.5	Prothrombin time
mg	1–8 year	0.3–0.5	Protein induced by vitamin K absence or antagonist II (PIVKA II)
	Adult	2.5–5.0/week	

Adapted from Munck, A. (2010). Nutritional considerations in patients with cystic fibrosis. *Expert Rev. Resp. Med.* **41**, 47–56.

Infants with cystic fibrosis

- Ideally CF babies should be breastfed (associated with better pulmonary function if at least 4 months breastfeeding). They require pancreatic enzymes with each feed (2500–3000 IU lipase per 120 ml milk).
- Alternatively, regular infant formulae can be given, again with pancreatic enzymes.
- Pancreatic enzymes should be mixed with a little breastmilk or formula and given to the infant on a spoon. They should not be given with formula milk in a bottle or feeder.
- Some infants with higher energy requirements may need additional supplementation. This should be undertaken with the advice of a registered dietitian with paediatric experience.

Complicating factors

CF and diabetes mellitus

Due to ↑ survival, up to 30% of CF patients develop diabetes and the dietary advice for the two conditions needs to be reconciled. It is imperative to maintain an adequate energy intake so fat should not be restricted but some saturated fat can be replaced by monounsaturates. As a bulky, ↑ carbohydrate diet may not be practical, foods contributing refined carbohydrate should not be restricted but eaten in conjunction with other items to dissipate the glycaemic effect and insulin prescribed if necessary.

CF and liver disease

Occur in up to 25% of CF patients. Additional problems with malabsorption may occur if bile composition is altered or output is ↓ and this cannot be remedied by ↑ pancreatic enzyme supplementation. If it is ∴ necessary to ↓ fat intake (and this should be avoided if possible), then an adequate energy intake must be maintained using carbohydrate and protein sources. Liver transplantation may be necessary and usually leads to an improvement in pulmonary function, as well as restoration of liver function.

Further information

Dougherty, K.A., Schall, J.I., and Stallings, V.A. (2010). Suboptimal vitamin K status despite supplementation in children and young adults with cystic fibrosis. *Am. J. Clin. Nutr.* **92**, 660–7.

Morton, A.M. (2009). The nutritional challenges of the young adult with cystic fibrosis: transition. *Proc. Nutr. Soc.* **68**, 430–40.

Munck, A. (2010). Nutritional considerations in patients with cystic fibrosis. *Expert Rev. Resp. Med.* **41**, 47–56.

Cystic Fibrosis Trust. Available at: http://www.cftrust.org.uk/aboutcf/publications/consensusdoc/C_3500Nutritional_Management.pdf.

Chapter 31

Human immunodeficiency virus (HIV) infection

Introduction, nutritional goals, and assessment

Introduction

Untreated human immunodeficiency virus (HIV) infection leads to progressive suppression of immune function, eventually rendering the body susceptible to opportunistic infections and tumours. While there is no cure, antiretroviral therapy (ART) is highly effective in suppressing HIV replication. HIV disease is now a chronic condition and causes of death in this population have shifted from traditional AIDS-related illnesses to non-AIDS (Acquired Immune Deficiency Syndrome) events, the most common being atherosclerotic cardiovascular disease, liver disease, end-stage renal disease and non-AIDS–defining malignancies. There are a diverse range of nutritional conditions associated with HIV, reflecting the complexity of the disease and pharmacological management.

- Malnutrition and HIV have similar deleterious effects on immune function (such as reduced CD4 and CD8 lymphocyte cells).
- Decreased nutritional status in people with HIV infection is associated with disease progression, increased morbidity, and reduced survival independent of immunodeficiency and viral load.
- Weight loss, specifically wasting, is an important predictor of HIV progression to AIDS and to death.
- Optimal intake of energy, protein, and micronutrients may help augment immune function.

Nutritional goals

- Prevent and treat unintentional weight and lean tissue loss.
- Manage symptoms and complications associated with HIV disease and treatment.
- Promote good health, adherence to treatment and prevent nutritional deficiencies.

Nutritional assessment

This should be undertaken regularly including:

- Detailed diet history.
- Height.
- Weight.
- Body mass index (BMI).
- Skinfold measurements and circumferences (see 📖 Chapter 4, ‘Anthropometry’, see p. 50) are useful to monitor body composition and shape changes linked to HIV disease and ART.
- Head circumference (infants and children under 2 years).

Unintentional weight and lean tissue loss

This remains a significant complication in the era of effective ART. The aetiology is multifactorial, the main precipitating factors being:

- reduced nutritional intake;
- altered metabolic requirements;
- malabsorption.

Management

Optimization of ART and treatment of underlying conditions and opportunistic infections are priorities. Nutritional status and requirements should be assessed using standard methods.

- Aim to ↑ energy and protein intake.
- Encourage small, frequent, nutritious meals, snacks, and drinks.
- Appropriate use of proprietary energy and protein supplements.
- Symptoms such as nausea, vomiting, diarrhoea, taste changes, and anorexia must be identified and treated (see 📖 Chapter 24, Chemotherapy', p. 490 and 'Radiotherapy', p. 491).
- Artificial nutrition support should be considered if nutritional needs are not met orally despite intervention.
- Nasogastric for short-term support.
- Percutaneous endoscopic gastrostomy (PEG) for longer term intervention (see 📖 Chapter 25, 'Routes for enteral feeding', p. 518).
- Parenteral nutrition is indicated in cases where it is not possible to feed via the gastro-intestinal tract (see 📖 Chapter 25, 'Parenteral nutrition', p. 536).
- Resistance exercise has been successfully used as a safe, cost-effective method of promoting lean body mass.

Cardiovascular risk and complications associated with HIV disease and treatment

A higher prevalence of ↓ bone mineral density, vitamin D deficiency, impaired glucose tolerance and ↑ cardiovascular risk has been reported in HIV positive people compared to individuals who are HIV negative.

Known metabolic and morphological disturbances, associated with HIV infection and treatment, may include:

- *Dyslipidaemia:* ↑ total and LDL cholesterol and triglycerides, ↓ HDL cholesterol.
- Insulin resistance and hyperglycaemia.
- Bone demineralization.
- Visceral, breast, and dorso-cervical fat accumulation.
- Lipoatrophy: loss of subcutaneous fat from limbs, buttocks, and face.

Management

Careful choice of ART, nutritional screening and assessment, and appropriate nutritional advice may help prevent long-term complications.

- Patients should have regular physical, biochemical and detailed monitoring of dietary intake for signs of development of metabolic side-effects and for risk of developing coronary heart disease and bone demineralization.
- Advice based on the 'Eatwell plate' (📖 Chapter 2, p. 27).
- Those with hyperlipidaemia or elevated CVD risk should receive more intensive dietary advice.
- Exercise should be encouraged as it may benefit metabolic variables and abdominal shape.
- Limited evidence suggests that ↑ fibre diets are associated with a ↓ risk of developing central fat deposition.
- Additional modifiable risk factors to address include smoking, hypertension and obesity.
- Dietary treatment of diabetes should follow current guidelines (see 📖 Chapter 22, 'Goals and principles of dietary management', p. 448).
- Weight-reducing advice may help reduce visceral adiposity.

Achieving good health and nutritional status

Good dietary intake and a healthy lifestyle may be particularly important for people living with HIV as they may have a higher risk of ill-health compared to those not infected. Good nutrition and a regular meal pattern may help maintain a healthy body weight, avoid long-term complications and promote good adherence to treatment.

Additional dietary issues

Prevention of mother-to-child transmission of HIV

Most children become infected with HIV through mother-to-child transmission (MTCT) during pregnancy, delivery or breastfeeding. Prevention of mother to child transmission programmes have significantly reduced MTCT of HIV to <1%.

In the UK and other well-resourced countries, where there is access to safe, clean water and where milk substitutes are affordable and easily available, avoidance of breastfeeding by women living with HIV is recommended to prevent transmission of HIV via this route. ❗ This advice does not apply in countries who do not have uninterrupted access to infant formula milk and clean water and where other specific conditions are not met. According to WHO Guidelines:

- Exclusive breastfeeding is recommended for infants born to women living with HIV for the first six months of life
- Complementary foods should be introduced at age 6 months and breastfeeding should continue for 12 months in environments where replacement feeding is not appropriate.
- Breastfeeding should only stop when a nutritionally adequate and safe diet can be provided without breastmilk.

Children living with human immune virus

In children, HIV disease has a more rapid progression to AIDS, compared to adults, and their immature immune systems make them more vulnerable to opportunistic infections. Children infected with HIV may have increased energy requirements of around 10% and while recovering from illness this may increase by 50–100% above requirements of healthy uninfected children. Conversely, others may be well-nourished and at risk of becoming obese.

Maintaining adequate growth is a priority. Nutritional screening and assessment, including height, weight, anthropometry and blood lipids, should be part of routine care and care plans should include the following considerations:

- Encourage healthy eating appropriate to age and nutritional status.
- Medication, including interactions with food.
- Disease state.
- Growth and development.
- Activity level.
- Social and financial aspects including housing, cooking facilities.
- Physical and mental health of other family members.
- Confidentiality issues that may prevent families from making use of local health services.
- Cultural background, including traditional foods.

A multidisciplinary approach to care planning is essential.

Food and drug interactions

Presence or absence of food in the gut may affect drug absorption or modify risk of side-effects. Some ART regimens involve food restrictions

and requirements. It is important to refer to the manufacturer's current information. See also 🕮 Chapter 38, 'Drug–nutrient interactions', p. 738.

Alternative therapies and drug interactions

While the majority of alternative treatments are generally safe, there are potential interactions between some herbal and botanical products with ART and other medications that may have serious consequences for treatment success. Clinicians should routinely ask patients about their use of herbal remedies, vitamins and minerals and alternative treatments.

Food and water safety

Food and water borne infection is more common in the immunocompromised host. Good food hygiene[1] and avoidance of high-risk foods (e.g. raw/undercooked eggs, unpasteurized milk products, raw/undercooked meat and fish) are advisable.

When food and water borne infections do occur, it is important that patients see a health worker without delay in order to minimize illness and avoid weight loss and nutritional impairment.

Further reading

Pribram, V. (2010). *Nutrition and HIV*. Wiley-Blackwell, Oxford.

[1] For further information see Food Standards Agency, London, 🖱 www.food.gov.uk.

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Chapter 32

Nutrition in mental health

Introduction

Nutrition interacts with and influences mental health in a comparable way to physical health: a 'healthy and varied diet' can help promote mental well-being whilst an inadequate or excessive intake of food or specific nutrients can have a detrimental effect on mental health (Figure 32.1). The duration and impact of nutritional effects vary depending on the life stage.

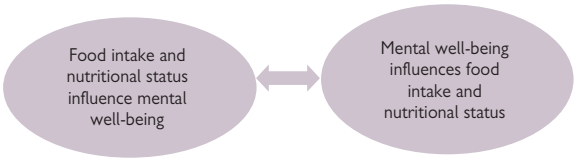

Fig. 32.1 Relationship between well-being and nutritional status.

It is estimated that one in four people in the UK will experience mental health problems over the course of a year. The interrelationship between mental health and nutrition includes a diverse range of topics ranging from those close to 'normal' healthy behaviour to the 'extremes' of mental ill health (see Table 32.1).

Table 32.1 Relationship between mood and eating*

Mood disorder and symptoms	Potential nutrition consequences
Depression	
Loss of appetite	Inadequate/inappropriate intake
Apathy and disinterest in food	Compromised nutritional status
Tiredness—unable to cook	Weight loss/gain
Loss of thirst sensation	Tiredness/lack of concentration
Food craving/erratic eating habits	Dehydration
	Constipation
Anxiety	
Restlessness/hyperactivity	↑ Energy expenditure
Dry mouth	Inadequate/excessive intake
Nausea, vomiting, diarrhoea	Difficulty chewing and swallowing
Loss of appetite	Compromised nutritional status
Food refusal	Weight loss/gain
Comfort eating	Tiredness/lack of concentration

* Note. Many of the nutritional consequences will contribute to the symptoms and potentially exacerbate them, e.g. tiredness in depression is associated with poor food intake → inadequate ingestion of energy and nutrients → further tiredness.

Pharmacotherapy in mental health

Drugs used in treating mental health problems may influence food intake and/or nutritional status. It should not be assumed that every patient taking medication will experience any or all of the side-effects associated with specific drugs. When side-effects arise, they are sometimes managed by adjusting the dose or changing prescription to a similar preparation that may be better tolerated. However, as some pharmacotherapy is long-term, e.g. taken for many years, there may be nutritional implications that require intervention (see Box 32.1).

Box 32.1 Examples of possible nutrition-related side-effects associated with selected drugs used to treat mental illness

Antidepressants

- Tricyclic, e.g. dosulepin, → dry mouth, sour metallic taste, constipation
- 5-Hydroxytryptamine re-uptake inhibitors, e.g. fluoxetine, → anorexia, nausea, and vomiting (usually mild) may occur in first 10 days but tend to resolve
- Monoamine oxidase (MAO) inhibitors, e.g. phenelzine, → patients taking these drugs should avoid foods containing high levels of tyramine, e.g. mature cheese, yeast extracts, soya bean products, pickled herring, and certain wine (see 🕮 Chapter 38, 'Drug–nutrient interactions', p. 738)

Antipsychotics

- Atypical antipsychotics, e.g. clozapine, olanzapine, → ↑ appetite, weight gain, diabetogenic
- Thioxanthenes, e.g. flupentixol decanoate (used as depot injection), → ↑ appetite, weight gain

Mood stabilizers

- Lithium salts, e.g. lithium carbonate, → nausea, metallic taste (~ mild, controlled by adjusting dose); serum electrolytes must be checked (see 🕮 Chapter 38, 'Drug–nutrient interactions', p. 738)

Anticonvulsants

- Barbiturates, e.g. phenobarbital*, → ↓ vitamin D levels, ↓ folate levels; e.g. phenytoin* → ↓ vitamin D absorption, ↑ turnover, & ↓ absorption of folate

* No longer 1st choice of treatment but many patients continue to take it.

Who can contribute to nutritional care in mental health?

The vast majority of people with mental health problems live in the community, some autonomously and others requiring considerable support. Obtaining, preparing, and eating a well-balanced diet can be a challenge and poor diet can exacerbate both short-term symptoms and the risk of chronic health problems associated with mental illness. In addition to family carers, health professionals in primary care and the community mental health team should be aware of nutritional needs and facilitate support. Specialist training in nutrition is rarely required and dietitians are well-placed to guide other healthcare professionals and support workers in how to optimize their clients' nutritional status. In addition to considering the health aspects associated with the nutrients supplied by food, the pleasure of eating and the empowerment associated with preparing an edible meal can also make a valuable contribution.

Nutrition in specific mental health conditions

Depression

In addition to increased sadness and anxiety, major depression is associated with loss of appetite.

- Low levels of neurotransmitters, e.g. serotonin, dopamine, noradrenaline and γ-amino butyric acid (GABA), are observed in depression. Dietary sources of neurotransmitter-precursors, e.g. tryptophan, tyrosine and phenylalanine may facilitate transmitter production with antidepressant effects. Systematic review[1] indicates supplements of 5-hydroxy tryptophan or tryptophan are better than placebos at alleviating depression but insufficient evidence is available to recommend supplementation. Eating a 'normal' varied diet will provide physiological doses.
- Omega-3 fatty acids may also play a role in neurotransmission via their conversion into prostaglandins and leukotrienes or through affecting signal transduction in brain cells. Meta-analysis[2] has found evidence of benefit from omega-3 supplementation (daily doses ~0.1–6.0 g of n-3) in people diagnosed with depression (standardized difference 0.10, 95% CI 0.02, 0.17, $p = 0.01$), but less effect on those who have depressed mood. See Table 23.3 for fish providing omega-3s.
- People with depression are more likely to have a ↓ intake and ↓ plasma markers of B vitamins. This may be a consequence of poor intake 2° to depressed appetite or co-morbidities rather than causative. Intervention studies are required to identify any potential benefit from supplements.

Bipolar disorder

Bipolar is a complex psychiatric disorder usually treated with mood-stabilizing medication.

- Omega-3 fatty acids play a key role in maintaining 'fluidity' of cell membranes which influences neurotransmitter receptor function. Addressing relative depletion of omega-3s has been investigated as a potential treatment for bipolar disorder. Systematic review[3] of clinical trials has indicated that omega-3 supplementation is safe and may be useful as adjunctive therapy for depressive but not for manic symptoms. Further studies are required before recommendations can be made.
- A number of studies have investigated the effects of a range of water soluble vitamins and amino acid supplements in bipolar disorder but without definitive evidence.

[1] Shaw, K.A., Turner, J., and Del Mar, C., (2009). Tryptophan and 5-hydroxytryptophan for depression. *Cochrane Database System. Rev.* CD003198.

[2] Appleton, K.M., Rogers, P.J., and Ness, A.R. (2010). Updated systematic review and meta-analysis of the effects of n-3 long-chain polyunsaturated fatty acids on depressed mood. *Am. J. Clin. Nutr.* **91,** 757–70.

[3] Montgomery, P.,and Tichardson, A.J. (2009). Omega-3 fatty acids for bipolar disorder. *Cochrane Database System. Rev.* CD005169.pub2.

Schizophrenia

Patients with schizophrenia usually suffer from hallucinations, paranoia, delusions and malfunctioning speech or thought.

- Impaired amino acid metabolism, and specifically reduced serotonin synthesis, has been associated with the pathophysiology. Limited evidence indicates that amino acids supplements may reduce some schizophrenic symptoms without adverse effects.
- Several studies have investigated the effects of omega-3 fatty acids in patients with schizophrenia but systematic review[4] of these has concluded that there is still insufficient good quality and independent evidence on which to base recommendations for supplementation.
- Weight gain is associated with antipsychotic drugs prescribed for schizophrenia (see 🕮 this chapter 'Pharmacotherapy in mental health', p. 673) and NICE guidance[5] recommends that this should be considered by service users and healthcare professionals when choosing medication.

Obsessive compulsive disorder

Obsessive compulsive disorder (OCD) causes recurring stressful thoughts followed by related compulsions (actions) which are repeated in an uncontrolled way.

- Selective serotonin re-uptake inhibitors (SSRIs) provide effective pharmacological treatment of OCD and ∴ in theory, foods which ↑ serotonin levels may also provide some benefit. There is no evidence to support this at present.

[4] Irving, C., Mumby-Croft, R., and Joy, L.A. (2010). Polyunsaturated fatty acid supplementation for schizophrenia. *Cochrane Database System. Rev.* CD001257.pub2.
[5] 🖱 http://www.nice.org.uk/nicemedia/live/11786/43608/43608.pdf.

Developmental disorders

Autism spectrum disorders

People with autism spectrum disorders (ASD) can have an intellectual ability ranging from a severe learning disability to being academically 'main stream'; ~10% may also have special skills or abilities. Asperger's syndrome is used to describe those with ASD who have an ability to function at a higher level. The characteristics of the conditions vary between individuals and with time but can be summarized as:

- Difficulties with communication.
- Difficulties with social interaction.
- Difficulties with behavior, interests and activities.

Pathogenesis

The exact cause of ASD is unknown, but a combination of genetic and environmental factors are thought to contribute to changes in brain development. It is unknown whether nutrition is causally implicated.

Nutrition as treatment

The role of diet has been investigated as a possible treatment of ASD, particularly as nutritional status plays an important role in normal brain development.

- *Micronutrient supplementation:* the potential benefits of vitamin B_6 and magnesium supplements have been investigated in 33 trials. However, systematic review[1] has concluded that these do not provide sufficient evidence on which to base recommendations and that further large, well-designed studies are needed.
- *Diets focusing on possible GI co-morbidity:* links between gastrointestinal (GI) tract symptoms and autism have led to evaluation of diets which might alleviate these including gluten- and casein-free diets and food elimination diets. Although there is some evidence[2, 3] to support a link between GI epithelial changes and altered immune response in ASD, the dietary benefits are predominantly anecdotal or from small or methodologically limited studies. At present, these do not provide sufficient evidence on which to base recommendations for dietary modification and further large, well-designed studies are needed.
- *Omega-3 fatty acids:* on the basis of their role in brain development and contribution to cell membrane integrity, >100 studies have investigated the role of omega-3s in ASD. However, on systematic review[4] this number was reduced to one randomized controlled trial which showed a small, but non-significant improvement associated with ~1.5 g/day over 6 weeks. Again, this is insufficient for the basis of recommendations and further large, well-designed studies are needed.

[1] Nye, C., and Brice, A. (2009). Combined vitamin B_6-magnesium treatment in autism spectrum disorder. *Cochrane Database System. Rev.* CD003497.pub2.
[2] Isherwood, E. and Thomas, K. (2008). *Dietary management of autism spectrum disorder*. British Dietetic Association, Birmingham.
[3] Srinivasan, P. (2009). A review of dietary interventions in autism. *Ann. Clin. Psychol.* **21,** 237–47.
[4] Bent, S., Bertoglio, K., and Hendren, R.L. (2009). Omega-3 fatty acids for autistic spectrum disorder: A systematic review. *J. Autism Dev. Disord.* **39,** 1145–54.

Attention deficit hyperactivity disorder (ADHD)

ADHD, or hyperkinetic disorder, is a syndrome characterized by hyperactivity, impulsivity and inattention. People with ADHD may exhibit all of these symptoms or predominantly more of one and less of another. Symptoms vary in severity and only those with significant impairment meet criteria for a diagnosis of ADHD. Symptoms of ADHD can overlap with symptoms of other related disorders.

Pathogenesis

The exact cause of ADHD is unclear but a combination of genetic and environmental factors are thought to contribute to changes in brain development. High coffee intake *in utero* has been suggested as a contributory cause but epidemiological studies have not identified this as an independent risk factor.

Nutrition as treatment

The role of diet has been investigated as a possible treatment of ADHD with little success:

- *Artificial colouring and additives:* many studies have investigated the benefits from eliminating these from the diet. Current guidance recommends that this should not be a routine treatment.[5] However, clinical assessment should include questions about food and drink and possible links to behavior. If these are reported, an intake/behavior diary should be kept and then if necessary, a referral made to a dietitian. Further dietary management, e.g. specific dietary elimination, should be jointly managed by dietitian, mental health specialist/paediatrician and patient/carer and young person.
- *Omega-3 (and other) fatty acids:* have been investigated in ADHD because of their role in brain development and contribution to cell membrane integrity. Although some studies have reported improvements in behaviour, the consensus[5,6] is that there remains insufficient evidence to support supplementation and that further well-designed and long-term studies are needed.
- *Weight loss or poor weight gain:* may arise in adults and children with ADHD if food intake is poor, hyperactivity results in energy expenditure exceeding intake or in association with some medication, e.g. methylphenidate, atomoxetine, or dexamfetamine. Routine monitoring of weight and, in children, plotting height and weight on growth charts is required. Taking medication with or after food or changing meal time to avoid peak drug-action may also help.

[5] http://www.nice.org.uk/nicemedia/live/12061/42059/42059.pdf.
[6] Gadoth, N. (2008). On fish oil and omega-3 supplementation in children: The role of such supplementation on attention and cognitive dysfunction. *Brain Devel.* **30**, 309–20.

Eating disorders

- Defined as persistent disturbance of eating (± behaviour) that impairs physical health or psychosocial functioning or both and that is not 2° to any other medical or psychiatric disorder.
- Include anorexia nervosa, bulimia nervosa, and binge eating disorder.
- Individuals who do not fall within strict diagnostic criteria are described as having an atypical eating disorder or disordered eating.

Pathogenesis includes

- *Genetic factors:* estimated hereditability 50–83%.
- *Biological factors:* starvation impacts directly on brain and is associated with behavioural and psychosocial impairment. Complex integration of appetite control, motivation to seek food and eat and self-regulation may also contribute.
- *Environmental factors:* may include events from conception onwards (stress in pregnancy, prenatal complications, prematurity) to societal pressures and concepts of 'fatness'/'thinness'.

***Management across all eating disorders*[1]**

- Assessment and co-ordination of care.
- Providing good information and support.
- Getting help early.
- Management of physical aspects, e.g. diabetes, pregnancy, laxative use, vomiting, dental health.
- Specific consideration of children and adolescents.
- Identification and screening in primary care and non-mental health settings.

Anorexia nervosa

- ICD-10 descriptor: anorexia nervosa (AN) is characterized by deliberate weight loss, induced and sustained by the patient. Occurs most commonly in adolescent girls and young women, but adolescent boys and young men may also be affected, as may children approaching puberty and older women up to the menopause. Associated with a specific psychopathology whereby a dread of fatness and flabbiness of body contour persists as an intrusive overvalued idea, and the patients impose a low weight threshold on themselves. There is usually undernutrition of varying severity with secondary endocrine and metabolic changes and disturbances of bodily function. The symptoms include restricted dietary choice, excessive exercise, induced vomiting and purgation, and use of appetite suppressants and diuretics.
- Life time prevalence is 0.9% for women and 0.3% for men.

Management

Guidance is mainly based on C grade evidence[1]

- Assessment and management in primary care including (but not only) body mass index, rate or weight loss, objective physical signs, laboratory tests; annual review by GP is required if not under 2° care.

[1] http://www.nice.org.uk/nicemedia/live/10932/29218/29218.pdf.

- Psychological interventions accompanied by regular monitoring of physical state. Therapies to consider include cognitive analytic therapy, cognitive behaviour therapy, interpersonal psychotherapy, focal psychodynamic therapy and family interventions.
- Aim for outpatient management which should last at least 6 months.
- Dietary counseling alone should not be sole treatment.
- For inpatients, provide structured, symptom-focused treatment regime with expectation of weight gain. Carefully monitor physical status on re-feeding (see 📖 Chapter 25 'Refeeding syndrome', p. 544). Provide psychological treatment but not rigid behavior modification programmes.
- Post-hospitalization, continue outpatient psychological treatment and physical monitoring for at least 12 months.
- For children and adolescents, offer family interventions but provide appointments for patients separate from family/carers. Involvement of siblings should be considered. Hospitalization should balance urgent physical needs with educational and social aspects.
- Pharmacological interventions. Limited evidence base and medication should not be used as sole treatment. Consider drugs side effects, especially cardiac, due to compromised cardiac function in AN monitor with ECG.
- Aim for weight gain of 0.5–1.0 kg/week in inpatients or 0.5 kg in outpatients. Will require additional 3500–7000 kcal/week (500–1000 extra per day). Oral multi-vitamin/multi-mineral supplement is recommended during weight restoration. Parenteral nutrition is not appropriate unless GI tract dysfunction.
- Feeding against the patient's will. Only as a 'last resort' and in the context of the Mental Capacity Act 2005. Healthcare professionals without specialist experience who are considering this or compulsory admission, should seek advice.

Bulimia nervosa

- *ICD-10 descriptor:* bulimia nervosa (BN) is characterized by repeated episodes of overeating and an excessive preoccupation with the control of body weight, leading to a pattern of overeating followed by vomiting or use of purgatives. This disorder shares many psychological features with AN, including over-concern with body shape and weight. Repeated vomiting is likely to give rise to disturbances of body electrolytes and physical complications. There is often, but not always, a history of earlier AN, the interval ranging from a few months to several years.
- Life time prevalence is 1.5% for women and 0.5% for men.

Management

Guidance is based on A, B and C grade evidence:[1]

- As a first step, encourage to follow evidence-based self-help programme supported by healthcare professional.
- Specifically adapted cognitive behavioural therapy (CBT-BN) should be offered with 16–20 sessions over 4–5 months.

- Consider other psychological treatment if patient does not want or does not respond to CBT-BN, e.g. interpersonal psychotherapy lasting 8–12 months.
- In addition or alternatively, offer antidepressant medication. First choice is selective serotonin re-uptake inhibitors (SSRI), e.g. fluoxetine in higher dose than for depression (60 mg daily). No other medication is recommended in BN.
- Physical effects should be monitored, e.g. fluid and electrolyte balance in vomiting and excessive laxative use. If treatment of electrolyte disturbance is required, utilize oral route unless GI tract dysfunction.
- Great majority of patients should be managed as outpatients but those of risk of suicide or self-harm may require more intensive care. If admission is required, it should be to an experienced unit.
- In children and adolescents, management should be adapted to their needs and include family if appropriate.

Atypical eating disorders including binge eating disorder

- *ICD-10 descriptor:* disorders that include some of the features of BN but overall clinical picture does not justify diagnosis, e.g. there may be recurrent bouts of overeating and overuse of purgatives without significant weight change or the typical over-concern about body shape and weight may be absent.
- Life time prevalence is 3.5% for women and 2.0% for men.

Management

Guidance is based on A, B and C grade evidence:[1]

- Management should follow the guidance for the eating disorder closest to the individual's clinical condition.
- As a first step, encourage to follow evidence-based self-help programme supported by healthcare professional.
- Specifically adapted cognitive behavioural therapy (CBT-BED) should be offered to adults. Other psychological treatments, e.g. interpersonal psychotherapy for binge eating disorder (BED) and modified dialectical behaviour therapy may be offered to adults with persistent BED. Suitably adapted programmes should be offered to adolescents.
- Inform patients that all psychological treatments for BED have limited effect of body weight. Consider concurrent or consecutive intervention for the management of co-morbid obesity.
- In addition or alternatively, offer a trial of antidepressant medication, e.g. an SSRI (see 📖 'Bulimia nervosa', p. 681).

Further reading

Treasure, J., Claudino, A.M., and Zucker, N. (2010). Eating disorders. *Lancet* **375**, 583–93.

Dementia

See Box 32.2 for factors influencing eating and drinking in dementia.

Dementia affects

- 700,000 people in the UK.
- 2% of people aged between 65 and 75 years.
- 20% of those >80 years.

Causes of dementia

- Alzheimer's disease;
- vascular disease;
- Lewy body disease;
- Huntington's disease;
- AIDS;
- head injury;
- Prion disease (e.g. CJD);
- multiple sclerosis;
- Wernicke–Korsakoff syndrome;
- syphilis.

Box 32.2 Factors influencing eating and drinking in dementia

- Poor memory: forgetting to eat or that they have eaten, forgetting to shop or the names of foods to buy
- Poor co-ordination: inability to put food on to cutlery, move food into the mouth, peel or unwrap food
- Inability to sequence activities needed to prepare meals
- Drug side-effects: dry mouth, drowsiness, constipation, dysphagia
- Poor concentration: easily distracted from meals by noise and other activity
- Tremor: spilling drinks, food
- Eating slowly: food becomes unappetizing or removed by carer
- Poor vision/confusion: food not recognized
- Agitation and restlessness: increases energy requirements while reducing opportunities to eat and drink
- Hallucinations: reluctance or refusal to eat food that appears to contain foreign bodies
- Tooth/mouth problems: pain or discomfort, ill-fitting dentures, tooth decay, altered taste
- Choking/swallowing problems: food may be hoarded in the mouth, spat out, taken out or aspirated causing chest infections
- 'Sun-downing': reduced cognitive function in the late afternoon/early evening so meals at this time may not be eaten
- Depression: a common additional diagnosis causing poor appetite and reluctance to eat
- Professional and lay carers may restrict diets because of additional diagnoses, e.g. diabetes, high cholesterol, diverticulitis, obesity. This may or may not be appropriate.

Improving nutrition

- Screening for nutritional risk, e.g. using MUST (see 📖 Chapter 25 'Malnutrition universal screening tool', p. 504) and dysphagia.
- Monitoring weight routinely; thinness is common in dementia because food intake is low, not because it is part of the illness.
- Maintaining physical activity to help maintain appetite and energy intake.
- Tailoring support to what the individual needs: help with shopping, cooking, company at mealtimes, verbal or physical prompts.
- Using soft and texture modified foods only when really necessary.
- Asking family carers for advice and information.
- Maintaining independence by offering help not interference.
- Offering snacks: some older people develop 'grazing habit'. Ensure snacks are nutritious so that total intake is not compromised.
- Providing choice by allowing people to select from plates of food that can be eaten immediately.
- Avoiding patterned crockery, tablecloths, etc., which may cause visual confusion at mealtimes and distract from the food.
- Putting drinks into clear glasses to make them easier to see.
- Allowing time for meals: hurried meals may cause agitation and distress.
- Limiting noise, distractions and other activities at mealtimes.
- Ensuring adequate lighting so that food can be seen properly.
- Talking about food and encourage eating by chatting about the meal.
- Ensuring adequate resources for catering services in institutional settings and in social care packages for people at home.
- Ensuring appropriate training for all staff involved in dementia care.

Increasing nutrient intake

- Using whole milk or Channel Island milk (rather than skimmed or semi skimmed milk) in all cooking and for drinks.
- Using sugar (rather than artificial sweeteners) in cooking and drinks.
- Making a cooked breakfast available. People with dementia may eat better early in the day.
- Including fried foods, cakes and traditional puddings on menus.
- Using alcohol in moderation to stimulate the appetite. This may be offered on its own, or added to other drinks before meals.
- Making food available at night for those who sleep poorly.
- Adding butter, margarine or grated cheese to mashed potato or other vegetables.
- Adding cream, white sauce, butter or margarine (rather than water or gravy) to food that needs to be pureed.
- Offering high energy snacks between meals (e.g. cake, biscuits, ice cream, instant desserts, trifle, chocolate, sandwiches).

Finger foods

Finger foods (see Table 32.2) may help people who cannot remember how to use cutlery. They may help people maintain independence and dignity by allowing people to feed themselves and the greater interaction with food may increase intake. Finger food menus must be analysed for nutritional adequacy.

Table 32.2 Examples of finger foods

Starchy and cereal	Protein-rich	Dairy	Fruit and vegetables	Energy dense
Buttered rolls	Chicken nuggets	Cheese cubes	Apple slices	Biscuits
Chips	Fish cakes	Sliced cheese	Banana pieces	Chocolate
Crumpets	Fish fingers	Yogurt-covered raisins	Carrot sticks	Crisps
Potato cakes	Hard-boiled egg		Celery sticks	Ice cream cones
Roast potatoes	Meatballs		Cherry tomatoes	Jam tarts
Tea cakes	Samosas		Grapes	Slices of cake
Toast fingers	Sandwiches		Orange segments	
	Sausages			

Soft diets

If a person with dementia is unable to chew, care should be taken to ensure that food provided supplies an adequate energy, protein, and micronutrient intake (see 📖 Chapter 23, 'Dysphagia', p. 480). Simply liquidizing ordinary food is rarely adequate and oral nutrition supplements should be considered (see Table 25.5).

Mental Capacity Act 2005

Overview

This Act provides a framework to empower and protect people with limited or no ability to make decisions for themselves. It defines who can make decisions for them and in which situations, and also describes the process of how this should be done. It facilitates forward planning by allowing people with capacity to anticipate a future time when they are not able to make decisions. The Act covers major decisions such as property and affairs, healthcare and where they live as well as day-to-day decisions about what to eat and personal care. It also provides guidance on the use of a Lasting Power of Attorney, Independent Mental Capacity Advocate and advance decisions concerning life sustaining treatments.

Five key principles

- Every adult has the right to make his or her own decisions and must be assumed to have capacity to make them unless it is proved otherwise.
- A person must be given all practicable help before anyone treats them as not being able to make their own decisions.
- Just because an individual makes what might be seen as an unwise decision, they should not be treated as lacking capacity to make that decision.
- Anything done or any decision made on behalf of a person who lacks capacity must be done in their best interests.
- Anything done for or on behalf of a person who lacks capacity should be the least restrictive of their basic rights and freedoms.

Further reading

http://www.publicguardian.gov.uk/mca/mca.htm.
http://www.opsi.gov.uk/acts/acts2005/ukpga_20050009_en_1.

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Chapter 33

Nutrition in neurological conditions

Multiple sclerosis

Multiple sclerosis (MS) is an autoimmune disorder of the central nervous system with a UK prevalence of ~1.4 in 1000. Damage is caused to the myelin sheath surrounding nerves, thus impairing the conduction of impulses. The condition varies from a relapsing/remitting pattern (~80% patients) to a progressive form that may be fatal within a few years. Nutritional considerations relate to:

- the cause of the condition;
- possible effects on its progression;
- specific nutritional concerns that might arise in some people with MS.

Pathogenesis

Genetic susceptibility and environmental factors have been implicated. A number of dietary factors have been investigated but convincing evidence is limited:

- Population studies have identified an association between a high prevalence of MS and ↑ saturated fat and ↓ polyunsaturated fatty acids intakes (myelin is composed of ~70% fatty acids, including ~ a third of polyunsaturates). This is not supported by case-controlled studies.
- A possible link between ↑ MS risk and ↓ dietary vitamin D intake or ↓ exposure to sunlight in childhood.

Prospective studies are needed.

Disease progression

More than 50% of people with MS take dietary supplements or modify their food intake using a range of interventions including polyunsaturated fatty acid (PUFA), antioxidant and vitamin supplementation and gluten- or milk-free diets. Evidence to support any positive effects is very limited. Systematic review[1] has identified that only six studies investigating polyunsaturated fatty acids have been adequately undertaken and the results of these do not show any benefit or harm. More good quality studies are needed.

In the absence of evidence, it is therefore recommended that people with MS should eat a variety of foods from the major food groups in quantities to maintain a healthy weight (see 📖 Chapter 2, 'The Eatwell plate', p 27). More specific advice may be needed by those with particular symptoms or weight changes, consider referral to a registered dietitian.

Specific nutritional concerns

Fatigue, loss of balance, weakness, numbness, tingling, and bladder problems are among the most common symptoms experienced by MSc patients. Difficulty in swallowing is relatively uncommon except in the very late stages but may have a profound impact on food intake and nutritional status.

- Routine nutritional assessment will help to identify patients whose nutritional intake is suboptimum before depletion results in clinical impairment. Regular (3–6-monthly) monitoring of weight and BMI will be sufficient in most cases.

[1] Farinotti, M., Simi, S., Di Pietrantonj, C., *et al.* (2009). Dietary interventions for multiple sclerosis. *Cochrane Database System. Rev.* CD004192.pub2.

- Practical support, e.g. help with shopping or meal preparation, may be sufficient to help some patients 'normalize' their intake.
- Nutritional supplements may help to increase nutrient intake where sufficient food cannot be consumed (see 🕮 Chapter 25, 'Treatment of undernutrition', p. 512).
- Overweight is common, ~40% of patients diagnosed 10–13 years, and related to reduced mobility and ↑ fatigue. A moderate energy-restricted diet that provides all other nutrient requirements is advisable (see 🕮 Chapter 21, 'Weight management: overview', p. 418).
- Constipation, reported in ~40%, may be alleviated by ↑ fibre intake and drinking sufficient fluid, >2l/day (see 🕮 Chapter 26, 'Constipation', p. 602).
- Swallowing difficulties should be evaluated by a speech and language therapist and dietitian with expertise in this area (see 🕮 Chapter 23, 'Dysphagia', p. 480).
- If an adequate nutritional intake cannot be maintained orally, feeding via a PEG may help improve quality of life (see 🕮 Chapter 25. 'Routes for enteral feeding', p. 518). If adequacy of intake is a concern, early intervention may help avert complications associated with under nutrition.

Alternative diets and multiple sclerosis

- Patients with multiple sclerosis (MS) are offered a variety of allegedly therapeutic diets through the popular press and other media.
- Some include specific dietary restrictions that may compromise the adequacy of their nutrient intake.
- Other regimes involve the purchase of specific food items or supplements that are not prescribable and often expensive.
- At present, there is little evidence to support dietary manipulation other than that described in 'Specific nutritional concerns', p. 690.
- Patients may need advice about the potential harms and benefits associated with some alternative diets (Box 33.1).

Box 33.1 Questions to ask about alternative diets advised for MS[2]

- Is it based on scientific evidence or just promoted by enthusiasts?
- Will the diet be worse than the symptoms?
- Is the diet nutritionally adequate?
- Are the ingredients easily available to buy?
- How much does it cost?
- Will it be difficult to prepare or cook the food?
- Is it recommended by neurologists and dietitians?

Further reading

Habek, M., Hojsak, I., and Brinar, V.V. (2010). Nutrition in multiple sclerosis. *Clin. Neurol. Neurosurg.* **112**, 616–20.

[2] 🖱 http://www.mstrust.org.uk/downloads/diet.pdf.

Motor neurone disease

Motor neurone disease (MND), or amyotrophic lateral sclerosis, is a group of related progressive disorders involving the degeneration of the motor neurones and leading to muscle weakness and wasting. Sensory neurones, e.g. taste, are not affected but difficulty chewing and swallowing may arise. The prevalence in the UK is ~6 in 100 000 and 50% of patients have a life expectancy of <3years at diagnosis.[1]

Undernutrition

Inadequate nutrient intake often leads to poor nutritional status (~20%). This may further impair muscle function and is associated with ↓ survival.

Causes

- Dysphagia: lip and tongue dysfunction, palatal incompetence, impaired swallow reflex, pharyngeal weakness, and reduced laryngeal elevation.
- Arm weakness: dependence on others to be fed.
- Social consequences: difficulty in eating and excessive salivation may inhibit eating with other people.

Treatment

- Assessment of nutritional status by a dietitian (see 📖 Chapter 4, 'Nutrition assessment', p. 33).
- Evaluation of swallow and appropriately textured diet by speech and language therapist and dietitian—good coordination is essential (see 📖 Chapter 23, 'Dysphagia', p. 480).
- Gastrostomy insertion (endoscopically or radiologically) can relieve pressure to eat. Attention to feed delivery time is required to minimise disruption to sleep and feeding in public.[2] Insertion is recommended if:
  - >10% loss of body weight despite supplementation;
  - unsafe swallow or bulbar symptoms;
  - life expectancy >3 m;
  - able to provide consent and manage feeds (or carer who can).

Overweight

A small number of patients, particularly those whose mobility is compromised by leg muscle weakness, are overweight. A modest energy restriction that does not compromise other nutrient intakes is advised (see 📖 Chapter 21, 'Weight management: overview', p. 418).

[1] Mitchell, J.D., and Borasio, G.D. (2007). Amyotrophic lateral sclerosis. *Lancet* **369**, 2031–41.
[2] Rio, A., Ellis, C., Shaw, C., *et al.* (2010). Nutritional factors associated with survival following enteral tube feeding in patients with motor neurone disease. *J. Hum. Nutr. Dietet.* **23**, 408–15.

Antioxidants

The role of antioxidants in treating motor neurone disease (MND), i.e. to combat the oxidative stress contributing to disease progression, has been examined in a number of studies, but a recent systematic review[3] revealed no significant benefits or contraindications. Whilst there is no specific evidence to support the use of supplements, diets of altered texture that are provided to patients with swallowing difficulties are often relatively ↓ in antioxidants and this could be addressed by the inclusion of suitably prepared (i.e. fresh and not overcooked) fruit and vegetables.

[3] Orrell, R.W., Lane, R.J.M., and Ross, M. (2008). Antioxidant treatment for amyotrophic lateral sclerosis or motor neurone disease. *Cochrane Database System. Rev.* CD002829.pub4.

Parkinson's disease

Parkinson's disease (PDis) is a chronic progressive neurological condition with a UK prevalence of 150–200 per 100,000. Although it is associated with older people, the mean age at onset is 55 years with 1 in 10 people aged >80 years affected. Symptoms including hypokinesis (reduced movement and fatigue), rigidity, tremor, and depression can contribute to a poor food intake and impair nutritional status, particularly in the later stages.

Pathogenesis

The causes of PDis are still unclear although probably include a genetic component. Many studies have investigated possible nutritional causes of PDis but provide limited conclusive evidence.[1] Avoiding excessive energy intake may be neuro-protective but this needs to be carefully balanced against ↑ energy needs 2° to motor symptoms and ↓ intake due to eating difficulties described below.

Nutritional management of symptoms

- *Constipation* may arise from a poor overall food intake, a ↓ fibre diet as a result of texture modification, or as a side-effect of anti-parkinsonian medication. Increasing dietary fibre and an adequate fluid intake (>2 l/day) is advisable. Fibre can be provided for patients requiring a soft/puree diet as oat porridge, pureed or mashed fruits including bananas, prunes, and dates, and thickened lentil-type soups.
- *Weight loss* will result from an inadequate intake possibly as a result of declining ability to shop or prepare food, increasing tremor that makes self-feeding difficult or swallowing difficulties. Energy requirements may be ↑ due to motor symptoms. An evaluation of the patient's physical status and nutritional needs will help identify how to best address undernutrition (see 🕮 Chapter 25, 'Undernutrition', p. 508).
- *Swallowing difficulties* are common and occur in up to 84% of patients, although in most they are relatively mild and do not impair food intake until a later stage. The patient's ability to swallow should be evaluated by a speech and language therapist and dietitian with expertise in this area to coordinate advice and an appropriately textured diet (see 🕮 Chapter 23, 'Texture modification', p. 480).
- *Dry mouth* may arise as a side-effect of medication. Moist meals served with appropriate sauce may help. Sharp flavour, e.g. lemon and grapefruit, may stimulate saliva.
- *Physical difficulties* in eating including spillages may limit intake. Occupational therapist assessment may identify helpful approaches including the use of non-slip mats, adapted cutlery, two-handled cups and stay-warm plates if meal times are prolonged. Finger foods may also help (see 🕮 Chapter 32, 'Dementia', p. 684).

Other nutritional management

Protein restriction The potential competition between circulating amino acids and PDis medication, L-dopa, led to investigation of diets providing

[1] Gaenslen, A., Gasser, T., and Berg, D. (2008). Nutrition and the risk for Parkinson's disease. A review of the literature. *J Neural Transm* **115**, 703–713.

↓ protein diets (<10 g/day) during the day. Although pre-1993 studies looked promising, the long-term nutritional effects have not been examined. There is insufficient evidence to recommend restricting dietary protein as a means of treating PDis and ☹ this may have serious adverse effects on nutritional status. However, patients experiencing fluctuating symptoms may benefit from manipulating the timing of protein consumption without restricting total intake[2] by:

- avoiding taking their medication with high protein meals;
- taking protein-rich foods in smaller quantities, but more often, i.e. six small meals/snacks daily;
- eating a greater proportion of their dietary protein in the evening.

Antioxidants Free radicals are implicated in the neurological damage of PDis, which has generated interest in the therapeutic value of antioxidants. NICE guidance[3] recommends that vitamin E supplements are not used, based on studies from the 1990s. However, a more recent review[1] of studies investigating the potential therapeutic effects of dietary antioxidants (i.e. from food, not supplements) including vitamin E concluded these are suggestive of potential benefit although currently provide insufficient evidence to translate into dietary recommendations. A well-balanced diet including five portions of fruit and vegetables per day will help provide a good baseline intake of antioxidants.

[2] 🖱 http://www.parkinsons.org.uk/PDF/B126_theprofessionalsguidetoparkinsons.pdf.
[3] National Collaborating Centre for Chronic Conditions (2006) Parkinson's disease: national clinical guideline for diagnosis and management in primary and secondary care. London: Royal College of Physicians http://www.nice.org.uk/nicemedia/live/10984/30087/30087.pdf.

Alzheimer's disease

Alzheimer's disease (AD) is the most common form of dementia and affects ~0.5 million people in the UK. This section provides a very brief summary of the limited nutrition and dietary-related evidence in the prevention and treatment of AD. See 🕮 Chapter 32, 'Dementia', p. 684 for practical advice about supporting people with AD.

- *Aluminium:* high levels of dietary aluminium were suggested as a possible risk factor for AD in the 1960s. This is not supported by evidence.
- *Folic acid ± vitamin B_{12}:* systematic review[1] has concluded that supplementation does not benefit unselected older people but may help improve cognition in older people with high homocysteine levels, i.e. with poor folate intake (see 🕮 Chapter 6, 'Folate', p. 112).
- *Omega-3 fatty acids:* increasing evidence (epidemiological, biological and observational studies but not randomised controlled trials) suggests that omega-3s have a protective effect. However, systematic review[2] has concluded that there is insufficient evidence at present to support the supplementation.
- *Thiamine:* systematic review[3] has concluded that there is no evidence that thiamine is effective in either preventing of treating AD.
- *Vitamin E:* systematic review[4] has concluded that there is no evidence that vitamin E is effective in either preventing of treating AD.

So, what is the optimum diet in AD?

In the absence of other evidence, a varied diet which includes foods from the major food groups in quantities compatible with a healthy weight should be recommended for people with AD. The key to optimizing the nutritional intake of people with AD may lie in the practical aspects of food provision, see 🕮 Chapter 32, 'Dementia', p. 684.

[1] Malouf, R., and Grimley Evans, J. (2009). Folic acid with or without B12 for the prevention and treatment of healthy elderly and demented people. *Cochrane Database System. Rev.* CD004514.pub2.
[2] Lim, W.S., Gammack, J.K., Van Niekerk, J.K., *et al.* (2009). Omega 3 fatty acid for the prevention of dementia. *Cochrane Database System. Rev.* CD005379.pub2.
[3] Rodriguez, J.L., Qizilbash, N., López-Arrieta, J., *et al.* (2010). Thiamine for Alzheimer's disease. *Cochrane Database System. Rev.* CD001498.pub2.
[4] Isaac, M.G.E.K.N., Quinn, R., and Tabet, N. (2008). Vitamin E for Alzheimer's disease and mild cognitive impairment. *Cochrane Database System. Rev.* CD002854.pub2.

Dietary treatments for epilepsy

The ketogenic diet (KD) is a high fat, restricted carbohydrate regime that has been used as a treatment for epilepsy since the 1920s. It has been shown to be successful in many observational studies, with over half of treated children showing seizure reduction greater than 50%, including 10–15% becoming seizure-free. A recent randomised controlled trial found seizure frequency after 3 months to be significantly lower in the KD group compared to controls. The KD is designed to induce a similar metabolic response to starvation, with the ketone bodies acetoacetate and β-hydroxybutyrate becoming the primary energy source for the brain in the absence of adequate glucose supply. Although the precise mechanism of action is unclear, initiation and maintenance of this state of ketosis is important for optimal seizure control. Other diets successfully used in epilepsy treatment are the modified Atkins diet (MAD) and the low glycaemic index treatment (LGIT).

Diet type

- *Classical KD:* used since the 1920s. Based on a ratio of fat to carbohydrate and protein, usually 4:1 (90% dietary energy from fat). Fat is mainly from foods, such as cream, butter, oil, and mayonnaise. Carbohydrate is usually limited to small servings of vegetables and/or fruits. Protein is based on minimum requirements for growth. An individual dietary prescription is calculated by an experienced dietitian; recipes are used with all meals and snacks at the correct ketogenic ratio and food must be weighed to ensure dietary accuracy.
- *Medium chain triglyceride (MCT) KD:* developed in the 1970s, the addition of MCT increases ketosis, thus allowing more carbohydrate and protein. The traditional MCT diet provides 60% energy from MCT; a modified version uses 30% energy MCT and an extra 30% from fat in foods. MCT is given as an oil or emulsion (Liquigen, SHS), both available on prescription. Although an individualised prescription must be again calculated by the dietitian, and all food weighed, exchange lists rather than recipes are often used.
- *MAD:* first reported in 2003, this diet restricts carbohydrates and encourages high fat foods, but does not limit or measure protein or total calories, or weigh food.
- *LGIT:* first reported in 2005, this is more generous in carbohydrates, but only those with a glycaemic index of less than 50 are allowed. Protein, fat, and calories are monitored, but not as strictly as on a KD. Food is not weighed, but based on portion sizes.
- Full vitamin, mineral and trace element supplementation is necessary on all diets to avoid nutritional deficiencies.

Indications and contra-indications for dietary treatments

- *Age:* the diets are mainly used to treat childhood epilepsy, but can also be successful in infants, although will require a more cautious approach to initiation and monitoring. The less restrictive MAD or LGIT may be more suitable for adolescents and adults
- *Seizure type:* traditionally used to treat generalised seizures, but no current evidence to show special benefits on any one type of seizure or syndrome.
- *Medications:* generally not used until at least two anti-epileptic medications have failed. Can be used alongside other anti-epileptic

medication, although initiation should be done with caution in patients taking topiramate due to increased risk of acidosis and excess ketosis.
- *Dietary restriction:* Can be used for both oral and nasogastric (NG) tube (gastrostomy) fed patients. Can be combined with dairy-free, gluten-free and vegetarian diets. A vegan KD would be difficult to implement without the use of a prescribable source of protein, due to the necessary carbohydrate restrictions.
- The diets should not be used in an individual with inborn errors of fat metabolism (β-oxidation defects) or disorders which require a high dietary carbohydrate content as treatment or a history of hyperlipidaemia or renal stones.
- They should be used with caution if also taking diuretics or medications that increase risk of acidosis. Concomitant steroid use may limit ketosis and there will be limited success if pre-existing behavioural feeding problems.

Initiation

Diets can be started at home, without a fast, if carefully monitored by the hospital team. Adverse effects on initiation are more common with the KD and could include excess ketosis, acidosis, hypoglycaemia, vomiting, diarrhoea and food refusal. Tolerance to the KD should be built up gradually by starting at a lower ratio (classical diet) or reduced amount of MCT (MCT diet); full diet can be achieved within a week or two.

Monitoring

- Dipsticks used to measure urine ketone (acetoacetate) levels initially twice daily. Can ↑ frequency once diet established, aiming for high readings (8–16 mmol/l). Finger-prick blood ketone (β-hydroxybutyrate) monitors have recently been developed and may improve accuracy of monitoring. Some centres also monitor blood glucose levels during KD initiation and fine-tuning.
- Routine serum biochemistry every 3–6 months, including plasma lipids, nutritional indices and carnitine.
- Urine tested for haematuria every 3 months, also calcium-creatinine ratio. Renal ultrasound may be needed if stones suspected.
- Regular measures of weight and height to ensure adequate growth.

Complications

- GI symptoms reported with KD use; commonly constipation; occasionally vomiting, diarrhoea and abdominal pain.
- Hyperlipidaemia is common, although long-term effects on cardio-vascular system undetermined.
- *Renal stones:* reported in 5–8% of KD users.
- *Growth problems:* risk of compromised linear growth in younger children on the KD, may be related to ↓ protein intake.
- *Increased infections:* reported in 2–4% of KD users, although no specific immunodeficiency determined.
- *Other:* literature reports on the KD include bleeding abnormalities and bruising, cardiac complications, pancreatitis, hypoproteinaemia, and potentiation of valproate toxicity.

Duration and discontinuation

- A 3-month trial of treatment is recommended. If there is no improvement by then despite appropriate dietary fine-tuning, it should be discontinued.
- If a diet is successful, it will usually be continued for at least two years, but the period of time should be individualized based on patient response, not on specific guidelines.
- Discontinuation must be undertaken cautiously. The longer it has been used and the more successful it has been in seizure treatment, the more gradual should be the change back to a normal diet. This will be done by a stepwise process which may take weeks or months.

Further reading

Neal, E.G. and Cross, J.H. (2010). Efficacy of dietary treatments for epilepsy. *J. Hum. Nutr. Diet.* **23**: 113–19.

Chapter 34

Rheumatology, dermatology, and bone health

Osteoarthritis

It is estimated that ~8.5 million people in the UK are affected by osteoarthritis (OA). This is the most common reason for the >50,000 hip replacements undertaken each year. These numbers are likely to increase as the population ages as this condition primarily affects people aged over 40 years. The hands, knees, hips, and feet are most commonly affected.

Nutritional risk factors

- Obesity is the most important, potentially modifiable risk factor for developing OA in weight-bearing joints, e.g. ↑ BMI by 2 kg/m^2 increases relative risk of knee OA by 1.36. Non-weight bearing joints are also affected by obesity, mediated by inflammatory factors secreted by adipose tissue.
- People with ↓ serum vitamin C and D and ↓ vitamin K intake have ↑ risk of OA; antioxidant vitamin C plays a role in collagen synthesis.

Nutritional advice

- No special diet is indicated in OA and a varied intake compatible with the 'Eatwell Plate' (see 📖 Chapter 2, p. 27) is advisable.
- Weight loss is recommended for those with a BMI ≥25 kg/m^2. Wherever possible, dietary advice should be combined with exercise (see 📖 Chapter 21 'Obesity', p. 411), which is considered the single most important intervention in OA.
- Glucosamine (glucose with an amino group) is a shell-fish derived compound described as a 'nutritional supplement'. Although some studies have indicated that it may help ↓ pain, recent systematic review[1] has concluded that non-branded glucosamine provided no benefit.
- No clinical trials have reported on the potential anti-inflammatory effects of *n*-3 fatty acids (eicosapentaenoic and decosahexaenoic) in osteoarthritis. *In vitro* studies using osteoarthritic models have demonstrated benefit from both *n*-3 and *n*-6 fatty acids, but results are inconsistent and further studies are needed.
- Although often avoided in OA, there is no evidence of benefit from ↓ intake of 'acid' foods, e.g. tomatoes and citrus fruit, and these provide antioxidants which are likely to be therapeutic.[2] However, there is no good evidence of benefit from antioxidant supplements, i.e. vitamins A, C, E, and selenium, individually or in combination.[3]
- Anecdotal reports of improvements with cider vinegar and/or honey have not been tested.
- Patients with advanced OA may have difficulty in shopping or preparing food and as a result their intake and nutritional status may fall. Appropriate support is required.

[1] Towheed, T., Maxwell, L., Anastassiades, T.P., *et al.* (2009). Glucosamine therapy for treating osteoarthritis. *Cochrane Database System. Rev.* CD002946.pub2.
[2] Raymond, M.P., and Pattison, D.J. (2008). Dietary manipulation in musculoskeletal conditions. *Best Pract. Res. Clin. Rheumatol.* **22,** 535–61.
[3] Canter, P.H., Wider, B., and Ernst, E. (2007). The antioxidant vitamins A, C, E and selenium in the treatment of arthritis: a systematic review of randomized clinical trials. *Rheumatology* **46,** 1223–33.

Rheumatoid arthritis

This chronic, autoimmune condition affects ~350,000 people in the UK. Joint inflammation causes swelling, pain, muscle weakness, and functional impairment with ~10% of sufferers experiencing severe disability <5 years after diagnosis. Rheumatoid arthritis (RA) is associated with a greater risk of cardiovascular disease and an estimated reduction in life expectancy of 3–10 years.

Nutritional risk factors

- Systematic review has shown that diet may play a role in the aetiology of RA. A diet providing olive oil, oil-rich fish, fruit, and vegetables may be protective while people with low serum antioxidant levels have ↑ risk.
- There is no evidence that high coffee intake increases risk of RA, but drinking coffee is frequently associated with cigarette smoking, which is an independent risk factor.

Nutritional issues

- Some patients with RA have poor nutritional intake 2° to loss of appetite and difficulty in preparing food, especially during periods of inflammatory exacerbation.
- Total energy expenditure in RA is lower than in matched controls, mainly because of a reduction in energy expended through physical activity. Basal metabolic rate may increase during inflammatory exacerbations but only if expressed per kg lean body mass.

Nutritional advice

- Patients with RA do not need a special or restricted diet but should aim to eat a nutritionally adequate intake, e.g. based on the 'Eatwell Plate' (see 📖 Chapter 2, p. 27).
- Attempts should be made to optimize weight by gentle reduction if overweight or by addressing undernutrition (see 📖 Chapter 21, 'Weight management', p. 418, 📖 Chapter 25, 'Treatment of undernutrition', p. 512).
- Cochrane systematic review[1] has concluded there is insufficient evidence on which to base specific dietary advice. Individual studies have shown some benefit associated with vegetarian and Mediterranean diets although unintentional weight loss may be a concern.
- NICE guidance[2] also concludes that there is no good evidence for dietary modification and suggests that people with RA should be encouraged to follow a Mediterranean-type diet (see Box 34.1).
- In spite of these conclusions, three meta-analyses[3] have shown benefits from the anti-inflammatory effects of omega–3 fatty acids. The best dietary sources of *n*-3 fatty acids are oil-rich fish including mackerel, salmon, and sardines (see Table 23.3).

[1] Hagen, K.B., Byfuglien, M.G., Falzon, L., *et al.* (2009). Dietary interventions for rheumatoid arthritis. *Cochrane Database of Systematic Reviews*. DOI: 10.1002/14651858.CD006400.pub2.
[2] 🖱 http://www.nice.org.uk/nicemedia/live/12131/43327/43327.pdf
[3] Raymond, M.P., and Pattison, D.J. (2008). Dietary manipulation in musculoskeletal conditions. *Best Pract. Res. Clin. Rheumatol.* **22,** 535–61.

- Antioxidants—although ↑ intake is protective against developing RA, the treatment effects of vitamin C, E, and selenium supplementation have been disappointing. Dietary sources have not been evaluated, but it seems reasonable to recommend a diet that provides good food sources of antioxidant micronutrients, not least because of their benefits in relation to cardiovascular disease. Five portions of fruit and vegetables daily will provide this.
- Folic acid supplementation (<5 mg/week) is beneficial[4] in patients treated with folate antagonist, methotrexate. Vitamin B_{12} status should be checked before starting.
- Exclusion/allergy diets are frequently followed by RA patients. These may include eliminating meat, dairy products, or 'acidic' foods or periods of fasting. Evaluative studies, mostly of limited quality, have reported mixed results, but provide inconclusive evidence. Patients should be advised to eat a well-balanced diet that provides good sources of the micronutrients described above and only restrict their intake if there is evidence of benefit. Review by a registered dietitian will help ensure that nutritional adequacy is maintained.
- Calcium and vitamin D status. Corticosteroids are an effective treatment for RA but patients are susceptible to steroid-induced bone disease (vertebral bone density 85–95% of control values). There is no evidence of benefit from Ca or vitamin D supplementation but adequate amounts should be consumed from food in the diet.
- Difficulties in obtaining, preparing, and eating food should not be underestimated in patients whose hands or jaws are particularly affected and may have an adverse effect on intake. Advice and practical support from an occupational therapist and dietitian may help overcome this.

Box 34.1 A Mediterranean diet is not a single entity, but includes …

- More oily fish and less meat
- Fresh fruit and vegetables
- Olive oil rather than saturated animal fats
- Wholegrains
- Legumes (pulses), nuts and seeds
- Red wine in moderate amounts

[4] Ortiz, Z., Shea, B., Suarez Almazor, M., *et al.* (2009). Folic acid and folinic acid for reducing side effects in patients receiving methotrexate for rheumatoid arthritis. *Cochrane Database System. Rev.* CD000951.

Gout

Gout is a metabolic disorder manifest as acute joint disease. It is caused by the deposition of urate crystals in the joints, tendons, and tissues leading to inflammation and severe pain.

Pathogenesis and nutritional issues

- High serum urate levels are associated with ↑ deposition, but do not always lead to gout. They may arise from:
  - metabolism from endogenous purines;
  - metabolism from dietary purines, e.g. from offal meat, fish, yeast extract, beer, and some vegetables;
  - reduced urinary excretion.
- Obesity and excessive alcohol intake (acute and chronic) is associated with ↑ endogenous urate production and ↓ urinary excretion.
- 95% of patients with gout also have hyperinsulinaemia.
- 25–60% of patients with gout also have hyperlipidaemia.
- Raised serum urate levels are considered a marker of insulin resistance.

Nutritional advice

- Losing excess body weight is the first priority. This should be undertaken by a modest ↓ dietary energy and ↑ exercise. Crash dieting may worsen hyperuricaemia and precipitate an attack so sudden weight loss should be avoided. Aim for a reduction of 0.5–1.0 kg/week.
- Saturated fat should be replaced by mono- or polyunsaturates, especially if hyperlipidaemia is present.
- An energy-restricted diet with ↓ fat (~30% energy), ↓ carbohydrate (~40% energy), and ↑ protein (~30% energy) has been shown to be compatible with weight loss and improvement in gout symptoms. However, further studies are required before it can be recommended as the optimum diet.
- Alcohol intake, especially from beer and spirits, should be reduced to moderate levels (see 📖 Chapter 9 'Alcohol', p. 186).
- Beef, pork, lamb and seafood are associated with ↑ serum uric acid so their intake should be reduced.
- Although pulses and nuts contain purine, ↑ intake is not associated with ↑ gout risk so their intake should be encouraged.
- Sugar-sweetened soft drinks should be reduced as fructose content is associated with ↑ serum uric acid. Added-fructose should be avoided but not fructose occurring naturally in fruit and vegetables because of the other health benefits associated with these foods.
- Coffee consumption may have some benefit. However, introduction of coffee or large quantities may trigger an attack (similar to introduction of allopurinol) so care is advisable initially.
- Low fat dairy products are associated with ↓ serum uric acid levels and ↓ gout risk as well as contributing to heart-healthy diet (see 📖Chapter 23, 'Cardiovascular disease', p. 465).
- Vitamin C supplements are associated with ↓ future gout risk. Dietary sources from fruit and vegetable may also be protective.

- There is no evidence at present that omega-3 or omega-6 fatty acids supplements yield any benefit in gout.
- There is no evidence that antioxidant supplements are beneficial.

Further information

Choi, H.K. (2010). A prescription for lifestyle change in patients with hyperuricaemia and gout. *Curr. Opin. Rheumatol.* **20,** 165–72.

Systemic lupus erythematosus

Systemic lupus erythematosus (SLE) is a chronic autoimmune condition where antibodies attack the connective tissues. People of all ages can be affected but it is most common in women of child-bearing age, and has a higher incidence in Black and Asian than White women.

Nutritional issues and advice

- There is no good evidence that nutrition is implicated in the pathogenesis of SLE.
- SLE is associated with ↑ risk of cardiovascular disease. A cardio-protective diet of low saturated fat, ↑ fruit and vegetables and oily fish is recommended to patients who are well and have a good appetite.
- The anti-inflammatory effects of omega–3 fatty acids may be beneficial. Dietary supplementation with ≡ 500 mg eicosapentaenoic acid and 350 mg decosahexaenoic acid is associated with a significant ↓ in systemic lupus activity measure.[1] This is equivalent to eating oily fish twice per week, e.g. 2 x 170 g (6 oz) fresh salmon/mackerel (see Table 23.3).
- Corticosteroids may be used in long-term treatment and SLE patients taking them have ↑ risk of osteoporosis. They should be advised to consume an adequate intake of dietary calcium and vitamin D and increase weight-bearing exercise. One pint (600 ml) of semi-skimmed/whole milk provides the reference nutrient intake for women aged 19–50 years, e.g. 700 mg Ca.
- Some patients with SLE may develop renal failure and may need specific dietary modification. Individual advice should be given by a renal dietitian.
- The health of patients with SLE can vary from relatively well to an acutely ill, hypercatabolic state. Nutritional support may be required if food intake is compromised and should be instigated promptly because poor nutritional status is associated with a worse outcome (↑ in systemic lupus activity measure).

[1] Duffy, E.M., *et al.* (2004). The clinical effect of dietary supplementation with omega-3 fish oils and/ or copper in systematic lupus erythematosus. *J. Rheumatol.* **31**, 1551–6.

Atopic eczema

Atopic eczema is a chronic inflammatory itchy skin condition which usually arises in childhood and in most cases follows a pattern of flares and remission. It is often associated with asthma and allergic rhinitis. A diagnosis of food allergy should be considered in children with atopic eczema who have reacted to a food with symptoms or when symptoms are not controlled by optimum management, especially if associated with GI tract symptoms, e.g. colic, vomiting and altered bowel habit. A diet history and details of dietary modification should be included in diagnostic investigations.

NICE guidance[1] includes specific direction about diet:

- A trial replacement of cow's milk formula should be offered to infants with moderate-severe eczema which is not controlled by emollients and mild topical corticosteroids. This should be replaced by either extensively hydrolysed protein formula or amino acid formula for 6–8 weeks.
- Children who follow a cow's milk free diet for longer than 8 weeks should be referred to a dietitian.
- Children with suspected cow's milk allergy should not be given milk from other animals, i.e. goats or sheep, or partially unhydrolysed protein formulae.
- Soya protein should be used as an alternative to cow's milk only in infants aged >6 months.
- The potential effect of maternal diet on eczema in breast fed infants is unknown.
- If food allergy is strongly suspected, an exclusion diet should be carried out with advice from a registered dietitian.

In addition …

- Infants with suspected egg allergy who have positive specific IgE to eggs may benefit from an egg-free diet.[2]
- There is no evidence that probiotics are beneficial in eczema but may be associated with adverse effects.[3]

See 🕮 Chapter 37 'Food hypersensitivity', p. 730.

[1] 🖱 http://www.nice.org.uk/nicemedia/live/11901/38597/38597.pdf.
[2] Bath-Hextall, F.J., Delamere, F.M., and Williams, H.C. (2008). Dietary exclusions for established atopic eczema. *Cochrane Database of Systematic Reviews*. DOI: 10.1002/14651858.CD005203.pub2.
[3] Boyle, R.J., Bath-Hextall, F.J., Leonardi-Bee, J., *et al.* (2008). Probiotics for treating eczema. *Cochrane Database System. Rev.* CD006135.pub2.

Epidermolysis bullosa

Epidermolysis bullosa (EB) is a rare genetic condition which causes severe blistering of fragile skin resulting in wounds, disability and a heightened risk of skin cancer. It is estimated that approximately 5,000 people in the UK have EB. There are different forms with varying severity of symptoms and these may include damage to GI tract membranes resulting in difficulty eating, strictures and subsequent poor nutritional status and restricted growth (see Box 34.2 for the practical aspects of nutrition and food).

Requirements

- Nutrient requirements may be ↑ to:
  - compensate for nutrients loss via open wounds;
  - optimize healing of skin lesions;
  - support the immune system in combating infection via broken skin;
  - assist in normal GI tract function and ↓ constipation.
- Energy requirements may be reduced if physical activity/mobility is low;
- ↑ Fluid may be required due to skin losses.

Box 34.2 Practical aspects of nutrition and food in EB

- Encourage breast feeding in infants
- Normal food is appropriate providing that it can be eaten and sufficient quantity consumed
- Nutrient-dense foods may help provide requirements if intake is poor
- If activity/mobility is limited, ↓ excessive energy that might → undesirable weight gain (but energy-dense foods may be needed if ↓ intake)
- Ensure adequate fluid and fibre intake to minimize constipation, which may damage fragile anal skin (be aware that fibre-rich foods may be filling and limit other intake)
- Spicy, crunchy and acidic foods may be painful to eat while smooth, cool/warm foods may be more acceptable
- If pureed foods are needed, ensure they are freshly prepared to minimise nutrient losses and that nutrient density is not compromised by addition of excessive fluid when processed
- Micronutrient supplementation may be required if intake is poor but limited information is available on requirements
- Height/length and weight should be monitored on nationally appropriate growth charts, taking account of the individual child's illness
- Infants and children should be referred to a paediatric dietitian for individual advice within the context of a multidisciplinary healthcare team

Further information

Haynes, L. (2010). Nutrition for children with epidermolysis bullosa. *Dermatol. Clin.* **28**, 289–301.

http://www.debra.org.uk/publications.html

Osteoporosis

Osteoporosis, thinning of the bones, affects ~3 million people in the UK, predominantly those >50 years, and is associated with ~230 000 bone fractures annually.

Nutritional risk factors

- Generally poor diet, including inadequate calcium and vitamin D intake.
- High alcohol intake.
- Malabsorption, e.g. coeliac disease, Crohn's disease.

However, most risk factors are not nutrition-related: early menopause, family history, treatment with corticosteroids, immobility.

Prevention through an optimum diet

- Peak bone mass is reached in adolescence and young adulthood, i.e. decades before most people are concerned about osteoporosis. It cannot be ↑ in later life so good early bone health is essential.
- Calcium intake should meet reference nutrient intakes (mg/day) given in Table 34.1. It is estimated that ~10% of boys and ~20% of girls fail to meet these recommendations (thus reducing their chance of achieving peak bone mass—see 🕮 Chapter 14 'Nutrient deficiencies in children', p. 286) and that up to 16% of women have a Ca intake <400 mg/day. An inadequate dietary intake will accelerate age-related bone loss and contribute to osteoporosis. See Table 34.2 for good dietary sources.
- Vitamin D is required in Ca metabolism. It is obtained from:
  - Adequate exposure to sunlight, e.g. face and arms, 30 min/day in direct sunlight between April and October. There is recent concern that many people in Europe do not achieve this and as a result have a poor vitamin D status;
  - Dietary sources. There are no reference nutrient intake values for the ages of 1–65 years in the UK (see 🕮 Appendix 6, p. 780). Most European countries recommend 5–10 μg/day. For good food sources, see Table 34.3.
- Excessive alcohol intake should be avoided. Safe limits are ≤21 units/week for men; ≤14 units/week for women.
- Weight-bearing exercise including stair-climbing and 10 min brisk walking daily is recommended.
- Recent studies have suggested that a low vitamin K intake and high salt, protein, and vitamin A intakes are detrimental to bone health. These issues are complex and at present there is insufficient information on which to base recommendations. A varied diet based on the 'Eatwell Plate' (see 🕮 Chapter 2, p. 27) is likely to be safe and contribute to good bone health.
- Maternal nutrition may influence the bone health of the next generation and, therefore, prevention of osteoporosis requires long-term public health strategies.

Table 34.1 Reference nutrient intakes (mg/day) of calcium*

Age	Male	Female
7–10 years	550	550
11–18 years	1000	800
>18 years	700	700

* For younger children see Appendix 6, p. 780.

Table 34.2 Sources of calcium

Providing ~200 mg Ca		Providing ~50 mg Ca	
Source	**Weight (g)**	**Source**	**Weight (g)**
Milk*, cupful	170	White bread	30
Yogurt, small carton	130	Wholemeal bread	50
Cheese, e.g. cheddar	30	Baked beans	100
Cheese spread	40	Muesli, Swiss	50
Sardines, canned with bones	40	Spinach	30
Sesame seeds	30	Orange	100

* Whole, semi-skimmed, or skimmed.

Whole milk and full fat cheese contribute saturated fat and ∴ selecting a lower fat alternative would provide a healthier option.

Table 34.3 Sources of vitamin D

Providing ~5 µg vitamin D		Providing ~5 µg vitamin D*	
Source	**Weight (g)**	**Source**	**Weight (g)**
Cod liver oil	2	Ghee	260
Herring, grilled	30	Pork chop, grilled	450
Salmon, tinned/steamed	50	Butter	550
Pilchards, tinned	60	Whole egg	600
Margarine (all types)	60	Lamb, roasted	650
Egg yolk	100	Beef, roasted	700

*The right-hand column shows quantities >> normal portions and are not recommended because of the high quantity of associated saturated fat.

Nutritional management

- When osteoporosis is diagnosed, management should include review of dietary adequacy.
- Supplementation with calcium and vitamin D has varying effects with five recent meta-analyses reaching different conclusions.[1] This variation probably arises from differences in adherence to long-term supplementation (Ca <10% adherence at 1 year) and effects on patients who are replete/deplete.
- One meta-analysis[2] of 15 trials (>8,000 patients) concluded that Ca supplementation has a modest effect on ↑ bone density and marginal effect on ↓ fractures but is also associated with ↑ in serious adverse cardio-vascular events so should not be the first line of treatment. Achieving an adequate Ca intake from dietary sources may be preferable providing that food sources are not also high in saturated fat.
  - If supplements are required, calcium is available as tablets, effervescent tablets, chewable tablets, granules, and syrup. As the required dose is large, the tablets are usually of considerable size. Major side-effects are rare but some gastrointestinal symptoms may occur. Splitting the daily does into smaller quantities and altering the time of dosing, e.g. before or after food, may help.
  - Dietary Ca absorption is enhanced by lactose and casein phospholipids which are present in milk. Oxalic acid (i.e. from rhubarb and spinach) and phytate (i.e. from the husk of grains, nuts and seeds), impair calcium absorption.
- Protein supplements should be provided for older patients with fractures who are undernourished but are unnecessary in those who are replete where they may ↑ urinary Ca excretion.[3]
- Recent studies indicate that vitamin K supplementation is associated with ↓ urinary Ca so may provide a future treatment option.

[1] Rizzoli, R., Bruyere, O., Cannata-Andia, J.B., *et al.* (2009). Management of osteoporosis in the elderly. *Curr. Med. Res. Opin.* **25**, 2373–87.
[2] Bolland, M.J., Avenell, A., Baron, J.A., *et al.* (2010). Effect of calcium supplements on risk of myocardial infarction and cardiovascular events: meta-analysis. *Br. Med. J.* **341,** c3691.
[3] Earl, S., Cole, Z.A., Holroyd, C., *et al.* (2010). Dietary management of osteoporosis throughout the life course. *Proc. Nutr. Soc.* **69,** 25–33.

Chapter 35

Palliative care

Palliative care

Palliative care is the approach taken to provide comfort and quality of life to people with a life-threatening illness and their families. When curative treatment is not possible, palliative care should be implemented at an early stage in the disease process. The aim is to alleviate symptoms and to support physical, emotional, spiritual and cultural needs. Although palliative care encompasses end of life care, it should be noted that many people live with an incurable condition for many months or years. Throughout this time, optimizing an individual's nutritional status is essential if physical function and quality of life are to be maintained.

Factors contributing to nutritional risk

Undernutrition is common in patients receiving palliative care. Reasons for deterioration in nutritional status are often multi-dimensional. Factors which can contribute include:

- *The disease process:* certain diseases such as cancer and motor neurone disease can alter the body's metabolism, resulting in an increase in nutritional requirements.
- *Symptom burden:* adverse physical symptoms such as nausea, early satiety, dysphagia, taste changes, pain and weakness can impact greatly on appetite and enjoyment of food. As a consequence, oral intake is decreased and nutritional requirements for macronutrients and micronutrients are not met.
- *Treatment:* treatments such as chemotherapy and radiotherapy may be given to palliate the disease. The side effects of these treatments often affect oral intake (see 📖 Chapter 24, 'Cancer and leukaemia', p. 487). In addition, prescribed medication and poly-pharmacy can also influence appetite and intake.
- *Psychosocial implications:* for the patient and their family, the psychosocial impact of being diagnosed with an incurable condition is significant. Psychological symptoms such as anxiety and depression may directly influence appetite. In addition, financial pressures, isolation or a loss of independence around activities of daily living may lead to practical difficulties associated with access to food.

For the individual, the physical and psychological consequences of nutritional requirements not being met manifest as weight loss and malnutrition. The implications of this have been discussed in 📖 Chapter 25, 'Undernutrition', p. 508. The clinical and economical pressures in terms of increased morbidity, poorer treatment outcomes and longer hospital stays must also be considered.

Nutritional management in palliative care

Screening should be used to identify those patients receiving palliative care who are at nutritional risk. In those susceptible to malnutrition, a full assessment should be completed to determine the appropriate nutritional management strategy. It must be emphasized that nutritional support should be considered in all patients where malnutrition has been identified as a concern.

Oral nutrition support

Oral nutrition, with the appropriate practical assistance to enable feeding, should be the first stage of providing nutritional support. Provision of food and fluids is a basic duty of care and, unless identified as unsafe, should be continued to the end of life. Points to consider include the following:

- Correct the correctable. Underlying symptoms or psychosocial issues which could be improved should be addressed.
- Advice regarding food fortification, meal pattern and the use of sip feeds and modular nutritional supplements should be discussed and prescribed on an individual basis.
- Previously imposed dietary restrictions, for example to treat dyslipidaemia or diabetes, may need to be relaxed.
- Good oral hygiene and mouth care is important and can help to reduce the risk of infections.
- Supportive care should be provided for the family and carers who may be struggling to understand and accept the difficulties and challenges around eating and meal times.
- A balance between encouragement and acceptance of the individual's inability to eat and drink is important.
- In some cases a reduction in oral intake may be a natural part of the dying process.

Artificial nutrition support

Where nutritional requirements fail to be met orally, consideration should be given to the initiation of artificial nutrition support. A decision should be made based on the likely prognosis, treatment plan and, where known, the patient's wishes. Both enteral and parenteral feeding have potential complications that need to be considered and weighed up against the likely benefits of feeding.

Healthcare ethics

The ethical considerations of the provision of artificial nutrition support in patients receiving palliative care continue to be an area of much debate. In healthcare ethics, four key principles exist which together form a framework within which ethical dilemmas should be discussed. These four key principles are:

- *Autonomy:* a patient's right to self-determination.
- *Non-maleficence:* avoidance of doing harm.
- *Beneficence:* providing benefit.
- *Justice:* fair and equal treatment for all.

When a decision is to be made regarding the use of artificial nutrition support, the following points should also be considered:

- In law, artificial nutritional support is viewed as a medical treatment and as such, can be withheld, given or withdrawn.
- In patients whose future remains uncertain, a time-limited trial to determine the benefits of feeding may be appropriate.
- Decisions should be made in a timely manner to avoid an irreversible deterioration in nutritional status.
- Informed consent must be obtained from the patient.

The Mental Capacity Act (2005)

Some patients, for example those who are unconscious or with conditions which cause confusion or drowsiness, may lack the capacity to make an informed decision regarding aspects of their healthcare treatment, including the provision of artificial nutrition support. The Mental Capacity Act (2005) for England and Wales provides a legal framework for people aged 16 or above to protect and empower them in this decision making process. It also provides guidance on the use of a Lasting Power of Attorney, Independent Mental Capacity Advocate and advance decisions concerning life sustaining treatments. Further details are described in 📖 Chapter 32, 'Nutrition in mental health', p. 671.

Palliative care summary

In all cases, nutritional support should be considered and provided on an individual basis. To ensure effective outcomes, good communication, multiprofessional working and ongoing support and monitoring are essential.

Further information

Department of Health (2005) The Mental Capacity Act. Available at: 🖱 http://www.dh.gov.uk/mentalcapacityact.

Ellershaw, J., and Ward, C. (2003). Care of the dying patient: the last hours or days of life. *Br. Med. J.* **326**, 30–4.

NICE (2004). *Improving supportive and palliative care for adults with cancer*. Available at: 🖱 http://www.nice.org.uk/csgsp.

NICE (2011). *End of life care*. Due Nov 2011. Details available at: 🖱 http://www.nice.org.uk/guidance/qualitystandards/indevelopment/endoflifecare.jsp.

Chapter 36

Inherited metabolic disorders

Definitions and management

Definitions

- *Metabolism:* cellular biochemical reactions that occur within the body. This involves the breakdown (catabolism) and formation (anabolism) of chemical compounds.
- *Metabolic pathways:* sequence of chemical reactions of metabolism.
- *Enzymes:* proteins that control the chemical reactions (or steps) in a metabolic pathway.
- *Cofactors:* 'helper molecules' that assist the protein in the biochemical reaction. These are often vitamins.

Inherited metabolic disorders (IMD) are due to deficient activity of an enzyme (or occasionally multiple enzymes) in a metabolic pathway. The deficiency 'blocks' the metabolic pathway and the clinical consequences of this arise because:

- substrates prior to the 'block' accumulate and can be toxic;
- essential products beyond the 'block' are not formed;
- other compounds may be formed via alternative pathways which may be toxic.

Patients can present at any age: as neonates, throughout childhood, and in adulthood. The severity of the disorder may vary widely depending upon the degree of enzyme deficiency. IMD occur in many pathways of amino acid, carbohydrate, lipid, and vitamin metabolism.

Treatment

Based upon an understanding of the biochemistry. The mainstays of therapy are the following:

- Therapeutic diet (see Table 36.1) to:
  - limit the intake of substrate that cannot be catabolized;
  - provide the product that cannot be formed.
- Large doses of cofactor vitamins.
- Medicines that conjugate with toxic metabolites so that the product is excreted in urine.
- Enzyme replacement therapy is possible in a few disorders.
- Liver transplant for some disorders.

IMD are rare and complex so it is essential patients are managed in a specialist metabolic centre by a multidisciplinary team including metabolic consultants, dietitians and nurses, with supporting specialized laboratory services.

Further reading

For detailed information on presentation, medical and dietary management and outcome of IMD see:

Fernandes, J., Saudubray, J.M., van den Berghe, G., *et al.* (2006). *Inborn metabolic diseases: diagnosis and treatment*, 4th edn. Springer Medizin, Verlag, Heidelberg.

Dixon, M. (2007). Inborn errors of metabolism. In V. Shaw and M. Lawson (eds) *Clinical paediatric dietetics*, 3rd edn. Blackwell Science, Oxford.

Newborn screening for inherited metabolic disorders

Newborns have been screened in UK for phenylketonuria since 1968, for MCADD in England since 2004, and Northern Ireland, 2009. Discussions are underway to expand newborn screening to include other IMD. Earlier detection will help improve outcome in many. For detailed information see: Expanded newborn screening; a review of the evidence can be found at: www.phgfoundation.org.

Table 36.1 Summary of dietary management of some inherited metabolic disorders

Disorder	Dietary management
Amino acid disorders	
Classical phenylketonuria	Low phenylalanine diet + phenylalanine-free, tyrosine-enriched amino acid supplement
Maple syrup urine disease*	Low leucine, isoleucine, valine diet + leucine, isoleucine, valine-free amino acid supplement Isoleucine and valine supplements may be needed Amino acid supplement given during illness
Tyrosinaemia type I and II	Low protein diet (to limit tyrosine intake) + tyrosine, phenylalanine-free amino acid supplement
Classical homocystinuria	Low methionine diet + methionine-free, cystine-enriched amino acid supplement
Organic acidaemias*	
Methylmalonic acidaemia* Propionic acidaemia*	Low protein diet (to limit methionine, threonine, valine, isoleucine intake) + methionine, threonine, valine, isoleucine-free amino acid supplement sometimes recommended
Isovaleric acidaemia*	Low protein diet (to limit isoleucine intake)
Glutaric aciduria type I*	Low lysine or low protein diet (to limit lysine intake) + lysine-free, tryptophan-reduced amino acids Amino acid supplement given during illness
Urea cycle disorders*	
Ornithine carbamoyl transferase deficiency* Citrullinaemia* Argininosuccinic aciduria*	Low protein diet (to limit waste nitrogen for excretion) + l-arginine supplements. Essential amino acid supplements may be needed
Carbohydrate disorders	
Classical galactosaemia	Minimal galactose and lactose diet. Infant soya milk substitute. Calcium and vitamin D supplements may be needed for children
Glycogen storage disease type I* and type III*	Frequent supply of exogenous glucose, provided as continuous overnight tube feeds and either 2-hourly daytime feeds or uncooked cornstarch Type III—milder disorders require less intensive dietary treatment

Table 36.1 (*Contd.*)

Disorder	Dietary management
Fatty acid oxidation disorders*	
Very long chain acyl-CoA dehydrogenase deficiency*	Minimal long chain fat, ↑ CHO diet and medium chain triglyceride supplements. Frequent daytime feeding and continuous overnight tube feeds or uncooked cornstarch
Long chain 3-hydroxyacyl-CoA dehydrogenase deficiency*	
Medium chain acyl-CoA dehydrogenase deficiency*	Emergency regimen during illness
Lipid disorders	
Familial hypercholesterolaemia	Healthy eating—restricted saturated fat, replace with poly- and monounsaturated fat
Type I hyperlipidaemia	Very low long chain fat diet (to tolerance). Medium chain triglycerides can be given
Abetaliproteinaemia	Very low long chain fat diet. Vitamin A&E supplements. Medium chain triglycerides not recommended

*Disorders requiring emergency regimen during metabolic stress such as intercurrent illnesses, e.g. colds, ear infections, etc. (see 📖 'Emergency regimens for IMD', this chapter, p. 724 for more detailed information).

Emergency regimens for inherited metabolic disorders

Metabolic stress, e.g. intercurrent infections combined with poor oral intake and fasting, anaesthesia, or surgery can precipitate severe metabolic decompensation in some IMD. Decompensation is due to catabolism with concomitant ↑ production of toxic metabolites. An emergency regimen (ER) of glucose polymer solution is given to provide energy and help minimize the effects of catabolism. The basic ER is similar for all disorders:

- Glucose polymer solution is given orally or via tube 2–3-hourly day and night.
- Carbohydrate concentration of these and volume given depends on age and weight (see Table 36.2).
- Glucose polymers can be flavoured to improve palatability.
- If an oral rehydration solution is prescribed for treatment of gastroenteritis, additional glucose polymer needs to be added to provide a final concentration of 10–12% carbohydrate. More concentrated solutions may exacerbate diarrhoea.
- For some disorders, additional specific therapy is given such as drugs to promote excretion of toxic metabolites or amino acids to promote anabolism.
- If the ER is not tolerated an admission to the local hospital for tube feeding of ER or stabilization with IV fluids (10% dextrose) is often necessary.
- Parents should be given explicit, hand-held, written ER instructions that explain the disorder, hospital management and provide contact details for the specialist metabolic centre.
- For detailed information on emergency regimen protocols for use in hospital see: www.bimdg.org.uk.
- ER solutions are not nutritionally complete and prolonged use can result in protein malnutrition. The child's usual diet should at least start to be reintroduced after 24–48 h of ER.

Table 36.2 Emergency regimens—composition and fluid volume for age

Age	Glucose polymer concentration (% CHO)	Energy (kcal/100 ml)	Suggested daily fluid volume given as 2–3-hourly drinks
0–6 months	10	40	150 ml/kg
7–12 months	10	40	120 ml/kg (up to 1200 ml/day maximum)
1–2 years	15	60	11–20 kg: 100 ml/kg for first 10 kg + 50 ml/kg for next 10kg
2–10 years	20	80	≥20kg: 100 ml/kg for first 10 kg + 50 ml/kg for next 10 kg + 25 ml/kg thereafter (up to 2500 ml/day maximum)
>10 years	25	100	

Phenylketonuria

Phenylketonuria (PKU) is due to a deficiency of the enzyme phenylalanine hydroxylase that converts the essential amino acid phenylalanine to tyrosine. Phenylalanine (Phe) accumulates in plasma and is neurotoxic. Tyrosine, which is essential for the synthesis of protein and the catecholamine neurotransmitters, becomes deficient. Untreated, patients will develop severe mental retardation. Newborn screening for PKU was established in the UK in 1968. Patients are treated with a low Phe diet that is continued throughout childhood; some adults also remain on diet. During pregnancy, a low Phe diet is essential to prevent damage to the unborn baby.

Low Phe diet—main principles

- Restrict intake of dietary protein to maintain plasma Phe concentrations within recommended reference range for age (Table 36.3).
- Provide daily Phe allowance. Daily Phe intake varies between patients and depends upon the level of enzyme activity:
  - Phe prescribed is based on plasma Phe concentrations;
  - Phe intake is measured using a system of 50 mg Phe exchanges or 1 g protein exchanges if Phe content of food is unknown;
  - Phe is provided in breast milk/infant formula for babies or low protein foods, e.g. potato, pasta, for older infants and children;
  - Phe intake is divided evenly across the day.
- Give a Phe-free amino acid supplement (protein substitute) with added tyrosine (essential because Phe restriction limits natural protein intake to below that needed for normal growth):
  - Generous intakes of Phe-free amino acids are recommended: 0–2 years =3.0 g/kg body weight/day; 3–10 years = 2.0 g/kg/day. Amino acid supplement is given 3–4 x throughout the day combined with some of the measured Phe foods.
  - A range of age-dependent prescribable amino acid supplements are available. These vary in nutrient composition and presentation, e.g. infant formula, gels, juice or milk-type drinks which need reconstitution, Tetrapak ready-made drinks, tablets, capsules (see 🕮 Chapter 38, 'Prescription of nutritional products', p. 745, and *British National Formulary*).
  - Flavourings need to be added to improve palatability/acceptability of some of these products, particularly older formulations.
- Give a vitamin and mineral supplement to meet normal dietary requirements. Some amino acid supplements provide adequate amounts of vitamins and minerals; if not, a separate supplement will be needed.
- Provide adequate energy intake for growth from a combination of:
  - naturally very ↓ protein foods (e.g. pure fats, sugar, fruit, some vegetables);
  - special ↓ protein, prescribable manufactured foods, e.g. bread and flour mixes, pasta, rice, biscuits, crackers, chocolate, snack pots, cereals.

Low Phe diet—monitoring

A low Phe diet is monitored by regular measurement of plasma Phe concentrations. See Table 36.3 for frequency of monitoring and recommended plasma Phe concentrations at different ages. Parents collect blood samples for Phe analysis (usually on to a Guthrie card and send by 1st class post to the biochemistry laboratory). Ideally, blood should be taken at the same time, in the morning before the amino acid supplement. Parents need to be promptly advised of any necessary dietary changes depending on plasma Phe results.

Reasons for high plasma Phe concentrations:

- intercurrent illnesses;
- too much dietary protein/Phe;
- insufficient amino acid supplement;
- unintentional use of non-PKU amino acid supplement or gluten-free, rather than low protein manufactured foods.

Reasons for low plasma Phe concentrations:

- inadequate intake of protein/Phe;
- growth spurt;
- ↑ requirement post-illness.

Table 36.3 Recommended reference ranges for plasma Phe concentrations and frequency of Phe monitoring in PKU*

Age (years)	Plasma Phe (μmol/l)	Frequency of monitoring
0–4	120–360	Weekly
5–10	120–480	Fortnightly
>11	120–700	Monthly

*Medical Research Council Working Party on Phenylketonuria (1993). Recommendations on the dietary management of phenylketonuria. *Arch. Dis. Child.* **68**, 426–7.

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Chapter 37

Food hypersensitivity

Food hypersensitivity

Classification

Food hypersensitivity (FHS) reactions can be categorized as immune-mediated (food allergies) and non-immune mediated (food intolerances).

IgE-mediated food hypersensitivity

- Includes classic primary and secondary sensitization to foods, causing a spectrum of symptoms from mild oropharyngeal symptoms through to anaphylaxis.
- Characterized by immediate symptoms.
- Prevalence of 4–8% in childhood and 1.3–4% in adults.

Non-IgE and non-immune mediated FHS

- Non-IgE food allergy includes coeliac disease, food-induced proctitis and entercolitis (FPIES) and eosinophilic oesophagitis.
- Non-immune mediated FHS includes enzymatic reactions such as lactose intolerance, pharmacological reactions such as hypersensitivity to vaso-active amines and salicylates and reactions of unknown aetiology such as those to food additives.
- Onset of symptoms for many conditions within this FHS spectrum is usually delayed.
- Actual prevalence for many conditions is unknown, except for well-characterized disorders such as lactose intolerance and coeliac disease.
- 30% or more of the population perceive themselves or their child to have FHS, whereas the actual prevalence varies greatly according to the condition.

Diagnosis

- *Clinical history:* the cornerstone of diagnosis should include information on symptom type and speed of onset, suspect foods, current diet and food exclusions and other factors such the presence of other allergies such as asthma or rhinitis, whether exercise, alcohol or aspirin were involved in the reactions and the past medical history and family history of allergy.
- *Skin prick test (SPT):* good first line test for all FHS. Is inexpensive, immediate, 95% negative predictive value, good sensitivity. A skin wheal over a certain size is predictive at 95 or 100% level for milk, egg and peanuts. Poor specificity and positive predictive value; using fresh foods can improve this. There is debate over whether SPT is suitable if the patient has taken antihistamines or has reported anaphylaxis. SPT may not be possible in extensive eczema where there is insufficient clear skin available to allow testing.
- *Serum IgE:* good for validating SPT or if SPT are not possible. Has good negative predictive value and sensitivity with cut off levels that are highly positively predictive for some foods. Is first choice of test if patient has taken antihistamines or experienced an anaphylactic reaction. However, no immediate result and not a good predictor for wheat (unless checking specifically for the wheat allergen omega 5 gliadin), soy, fruits and vegetables.

- *Diagnostic diets:* 4-week avoidance of a food group, food additive or naturally occurring substance in food followed by open or blinded challenge if symptoms improve. Useful if discordance between tests and clinical history and essential if no suitable tests available. Total exclusion, or 'few foods' diets rarely used for IgE-mediated FHS but may be useful for other FHS. Unsupervised elimination diets may be unsafe and diagnostic diets should be managed by a dietitian to ensure dietary adequacy. No need for diagnostic avoidance diet if test result is strongly concordant with clinical history.
- *Food challenge:* the gold standard of diagnosis. Is used to establish a diagnosis for IgE-mediated FHS if discordance between history, test results and diagnostic diet. Definitive diagnostic test for most non-IgE mediated and non-immune mediated FHS. Can also be used to check whether a childhood food allergy has resolved. Speed of onset and symptom severity normally dictate whether a challenge should be carried out in the hospital setting and whether it should be open or blinded.

There are many non-validated tests available that claim to diagnose FHS. Patients should be discouraged from using the results of such tests as a basis for implementing dietary change.

Management

The main management for any FHS reaction is avoidance of the trigger food(s). For most foods, the avoidance advice will be similar but degree may vary depending on the severity of symptoms and type of reaction.

Cow's milk

- Prevalence is 1.9–3.2%, although the allergy resolves in 75% of cases, especially in those who can tolerate baked milk and usually before adulthood.
- Diet excludes all cow and other mammalian milks such as goat or sheep milk; also milk components such as milk solids, lactose, casein, and whey; foods containing milk e.g. flavoured crisps, baked beans and breakfast cereals.
- Infants 0–6 months should be exclusively breastfed or given an extensively hydrolysed or amino acid formula.
- Commence weaning at 6 months; can include infant formula soy milk and soy foods at this stage, if soya is tolerated.
- Milk substitutes fortified with energy, calcium, and B vitamins can be used in >5 years; rice milk, oat milk, goat's milk, and unfortified soy milk are not suitable.
- Calcium and vitamin D supplements may be required by both children and adults.
- Nutritional adequacy of milk-free diets that do not include a milk replacement may be compromised.
- Milk can trigger other FHS such as lactose intolerance and non-IgE-mediated food allergy such as FPIES and eczema; it has also been linked anecdotally to asthma and irritable bowel syndrome.

Eggs

- Affects 1.6–3.2% of children and most commonly presents in the first year of life.
- 50% of children will become tolerant to egg, and many of those with persisting allergy will be able to tolerate cooked egg.
- High level of cross-reactivity to other avian eggs such as duck and goose eggs; there is also cross-reactivity to allergens in chicken meat.
- Cooking reduces the allergenicity of eggs but raw or loosely cooked egg may still be present in foods e.g. royal icing, marzipan, meringue (Pavlova), mayonnaise, Yorkshire pudding.

Fish and shellfish

- Affects children and is the commonest food allergy in adults; it is usually lifelong.
- There is high cross reactivity between different fish species, including white and oily fish and fresh and sea water fish and between crustaceans (prawns, lobster and crab) and molluscs (mussels, squid, oysters, snails) but no cross-reactivity between fish and shellfish (crustaceans or molluscs).

- It is prudent for people to avoid all seafood if they are allergic to fish or shellfish due to cross contamination.
- Some allergens in fish can be denatured by intense heat, such as during the canning process, but allergens in shellfish are very robust and not destroyed by heat. Fish allergens may be present in cooking vapours.
- Certain species of fish such as tuna and mackerel, and fish that is not completely fresh, can contain high levels of histamine which can precipitate a pseudo-allergy called scombroid poisoning, which may be mistaken for allergy.

Wheat and other grains

- Wheat allergy is more common in children than adults. Wheat is however implicated in a form of food allergy that does affect adults, known as food-dependent, exercise-induced anaphylaxis (FDEIA) where the reaction only occurs when the trigger food is eaten in conjunction with exercise; wheat is the most common precipitant.
- SPT and serum IgE are poor predictors of wheat allergy; a better choice is to test for the presence of IgE antibodies to the wheat allergen omega-5 gliadin which is highly predictive of wheat allergy in children and FDEIA triggered by wheat.
- There is a high degree of cross-reactivity between wheat and grass pollen, so that 80% positive SPT to wheat have no clinical significance in people with a grass pollen allergy.
- Wheat is also involved in other FHS reactions such as coeliac disease (see 🕮 Chapter 26, 'Coeliac disease', p. 592) and often reported to precipitate symptoms in people with irritable bowel syndrome.
- Corn/maize, barley/malt and rice have all been reported to cause food allergy although less so than some other cereals.

Fruit and vegetables

- Can be primary allergy with sensitization mediated though ingestion, but more commonly involved in cross-reactions between tree pollen and plant foods, known as Pollen Food Syndrome (PFS), part of a spectrum of conditions called Oral Allergy Syndrome.
- PFS affects 6–47% of pollen-sensitized individuals, although up to 90% of those sensitized to birch pollen can have PFS.
- Symptoms are most commonly triggered by apples, stone fruits and tree nuts, with immediate symptoms on consuming the raw fruit or vegetable in its raw state.
- Allergens from other pollens and latex can also cross-react to plant foods; there can also be cross-reactions between plant foods.

Peanuts and tree nuts

- Can develop at any age, but most commonly in childhood; 20% of peanut allergy resolves, but less than 10% of tree nut allergy.
- Peanuts most commonly reported food to provoke anaphylaxis.
- Better labelling; declaration of ingredients and nut trace warnings may help decrease accidental exposure.

- Up to 60% of peanut allergic individuals are sensitized to other nuts but this may not be manifest clinically.
- Prevalence of allergy to some nuts increasing, e.g. to cashew and Brazil nuts.
- Usual dietary advice is to avoid all nuts and foods containing nuts, even if only one nut is reported to be a trigger food, due to contamination and poor recognition of different nuts; there is usually no need to avoid other legumes in the case of peanut allergy, unless symptoms have been reported.
- Foods most likely to contain nuts include pastries, biscuits, cereals, ice-cream, pesto (which commonly contains cashew nuts), oriental and Asian cuisine, named nut oils such as hazelnut oil; refined oils containing peanut oil are usually allowed.
- Sesame seeds are the commonest seed allergens, although reactions to mustard seed and sunflower seed are becoming more common.
- Seeds have similar allergens to tree nuts and peanuts and cross-reactions may occur.

Food additives

- Commonly perceived to cause FHS, but are likely to only affect less than 0.1% of the population.
- People who have asthma and urticaria are more likely to report hypersensitivity to certain food additives.
- The following additives are the most likely to be implicated in FHS:
  - *Colourings:* both natural food colourings such as carmine (cochineal), annatto, turmeric and saffron and synthetic azo dyes such as tartrazine (E102) and sunset yellow (E110).
  - *Preservatives:* benzoates (E210–219)—beer, jam, fruit products, pickled foods, yoghurt, salad cream, berries, prunes and spices such as cinnamon and cloves; sulphites (E220–E227)—wine, cider, lager, fruit juices and squashes, meat products, dried fruits and vegetables.
  - *Flavour enhancers:* monosodium glutamate—soups, sauces, dried noodle snacks, gravy, ready meals, and Chinese food.

Naturally occurring food hypersensitivity triggers

- *Salicylates:* occur naturally in plant foods, and have a similar chemical structure and properties to aspirin; foods containing the most salicylate include coffee, tea, wine, dried herbs and spices, black pepper, oil of wintergreen, spearmint, certain fruits and vegetables.
- *Vaso-active amines:* can cause a type of FHS, which mimics IgE-mediated FHS; histamine, the commonest vaso-active amine, is found in red wine, strong and blue cheeses, oily fish especially tuna and mackerel, spinach, aubergines, and pork products.

Food labels

EU directive (2006/142/EC) requires that the foods listed in Table 37.1 be declared on the label of all packaged food if they have been deliberately added to the product, however small the amount:

Table 37.1 Foods to be listed on packaging under EU directive 2006/142/EC

Celery	Molluscs (including mussels, scallops, squid and oysters)
Cereals containing gluten (including wheat, rye, barley, oats and spelt)	Mustard
Crustaceans (including prawns, lobster, crab)	Peanuts
Eggs	Sesame seeds
Fish	Soya and products containing soy
Lupin flour	Sulphites and SO_2 >10 mg/kg
Milk and milk products including lactose	Tree nuts i.e. almond, hazelnut, walnut, cashew, pecan, brazil, pistachio, macadamia

See 🕮 Chapter 8, 'Food labelling', p. 166.

Further information

Skypala, I., and Venter, C. (2009). *Food hypersensitivity: diagnosing and managing food allergies*. Wiley-Blackwell, Oxford.

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Chapter 38

Drug–nutrient interactions and prescription of nutritional products

Drug–nutrient interactions

Practitioners should consider any possibility of interactions between food or enteric feed given to patients and prescribed or over-the-counter medication. Both micro- and macronutrients may interact with drug therapy and these interactions may be positive (enhancing the effects of the medication leading to medication toxicity) or detrimental (leading to a failure of therapy or drug ineffectiveness).

Herbal medicines, e.g. St. John's wort, gingko, dong quai, may also interact with prescribed medication or interfere with the patient's condition.

The risk of nutrient-drug interactions is greatest in those taking more than one medication, children and elderly people. This raises particular concern about drugs with a narrow therapeutic index, i.e. those that require careful monitoring, for example:

- lithium;
- monoamine oxidase inhibitors (MAOI);
- phenytoin;
- theophylline;
- warfarin.

Food, enteral feeds and herbal medicines can affect the main pharmacokinetic processes—absorption, distribution, metabolism and excretion of drug therapy. Examples of each are given in Tables 38.1–3.

► Medication is best taken with a glass of water, as acidity of fruit juices, tea and coffee can alter drug properties and its pH balance.

Interactions leading to alterations in drug therapy

The absorption of certain drugs may be delayed and reduced by the presence of food in the stomach or, in some cases, enhanced. Taking certain medication with food can reduce gastric irritation or damage. For example, it is advisable to take ibuprofen, naproxen, and diclofenac with or after food. It is for this reason that cautionary medication labels such as 'with or after food', 'an hour before food or an empty stomach', should be followed.

Table 38.1 Absorption drug-food/nutrient interactions*

Drug/class	Food and/or nutrient	Effect of interaction/advice
Bismuth Flucloxacillin Phenoxymethylpenicillin Rifampicin	Presence of food in the stomach	Absorption delayed and ↓ Advise to take on an empty stomach or 1 h before food
Ciprofloxacin Norfloxacin Tetracyclines	Milk and dairy products	Absorption ↓ Advise to leave 2-h gap between drug and dairy consumption

(*Continued*)

Table 38.1 (*Contd.*)

Drug/class	Food and/or nutrient	Effect of interaction/advice
Theophylline	High protein diet High carbohydrate diet	Bioavailability ↓ by high protein diets Bioavailability ↑ by high carbohydrate diets
Ciprofloxacin Digoxin Phenytoin Rifampicin Tetracycline Theophylline	Enteral feeds	Absorption ↓ Leave time gap between giving medication and enteral feed

Table 38.2 Metabolism drug-food/nutrient interactions*

Drug/class	Food and/or nutrient	Effect of interaction/advice
Amiodarone Buspirone Calcium channel blockers Carbamazepine Ciclosporin Colchicine Corticosteroids Coumarins Digoxin Fexofenadine Simvastatin	Grapefruit juice	Metabolism of drug altered due to liver enzyme, cytochrome P450, stimulation Advise to follow manufacturer's instructions
Warfarin	Large amounts of brussels sprouts, green vegetables, cabbage, lettuce, green tea, excess quantities of ice cream, avocado, mango	Drug metabolism ↑ and anticoagulant effect of warfarin ↓
Warfarin	Cranberry juice	Anticoagulant effect of warfarin ↑ ❗ fatal incident reported
Warfarin	High doses of vitamin E	Anticoagulant effect of warfarin ↑
Levodopa Phenobarbital, phenytoin	Vitamin B_6 (pyridoxine)	Effects of levodopa ↓ by concurrent supplementation of pyridoxine Advise to avoid vitamin B_6

Table 38.3 Excretion drug–food/nutrient interactions*

Drug/class	Food and/or nutrient	Effect of interaction/advice
Lithium	Salt (sodium containing)	Excretion of lithium affected by ↑ or ↓ sodium intake
		Advise that once stabilized on lithium, patients should keep sodium intake stable

*These lists are not exhaustive—reader should consult Stockley's Drug Interactions or British National Formulary for further details.

Warfarin and vitamin K containing foods

The anticoagulant, warfarin, is a vitamin K antagonist and alterations in vitamin K intake may affect the levels of warfarin and its anticoagulant effect. For example, consumption of large amounts of beetroot, green leafy vegetables (spinach, brussels sprouts, lettuce), green tea, mango, large quantities of ice-cream and alcohol may ↓ anticoagulant effects of warfarin. Once patients are stabilized on warfarin they should avoid major variations in the consumption of these foods and diet overall.

Potentiation of drug action

Inhibition of drug metabolism by nutrients may enhance drug effects. For example:

- Cranberry juice has been implicated in death through enhancing the effects of warfarin leading to fatal bleeding.
- Grapefruit juice may enhance the actions of calcium-channel blockers (especially felodipine, nimodipine, nicardipine and verapamil) used in hypertension, angina and arrhythmias. It may also potentiate the effects of simvastatin by reducing its metabolism. Amlodipine can occasionally interact with grapefruit juice.
- Grapefruit juice may enhance the actions of the antihistamine fexofenadine and the immunosuppressant ciclosporin. Increase in plasma drug concentrations of those drugs can lead to drug toxicity.
- Fish oils inhibit platelet aggregation and may ↑ bleeding when used together with anti-platelet drugs (aspirin) and anticoagulant, warfarin.
- High doses of vitamin E enhance the anticoagulant effects of warfarin.
- Dietary sodium influences the excretion of lithium (used in bipolar affective disorder) so that increasing salt intake may ↓ plasma lithium whilst salt-restricted diets may ↑ plasma lithium to toxic levels.

Interactions limiting therapy

- Diuretics such as bendroflumethiazide and furosemide used for hypertension and chronic heart failure cause salt and water excretion. Their therapeutic effects may be reduced by consuming a high salt intake.
- The antibiotics, tetracycline and quinolones (such as ciprofloxacin) should not be taken at the same time as antacids, milk or substances containing zinc, iron or calcium salts as these can lead to ↓ absorption and ↓ effectiveness of the antibiotic. Separate intake by 2 h.

- The therapeutic actions of levodopa for the management of Parkinson's disease may be ↓ by supplements of vitamin B_6 (pyridoxine). This does not occur when the levodopa is combined with a dopa-decarboxylase inhibitor, e.g. carbidopa.

❗ Interactions with potentially serious events

- Warfarin and vitamin K containing foods: see Table 38.2.
- *Warfarin and cranberry juice:* see Table 38.2.
- *Grapefruit juice and fexofenadine or ciclosporin:* see Table 38.2.
- *Angiotensin converting enzyme (ACE) inhibitors/angiotensin II receptor antagonists and potassium supplements or salt substitutes:* ACE inhibitors (e.g. ramipril, enalapril) and angiotensin II receptor antagonists (e.g. losartan) may cause K^+ retention and this may be exacerbated by K^+ supplements or K^+-containing salt substitutes causing hyperkalaemia.
- *MAOI and tyramine:* patients taking MAOI (e.g. phenelzine, moclobemide) for depression (rarely used nowadays) should avoid tyramine-containing foods (e.g. mature cheese, yeast extracts, soya bean products, pickled herring, red wine) as this may lead to the 'cheese reaction' with a severe increase in blood pressure and palpitations.
- *Isotretinoin and vitamin A:* isotretetinoin is a retinoid used in the treatment of acne and should not be used with vitamin A supplements due to the risk of a vitamin A overdose.

Drug therapies requiring nutritional supplements

Certain drug therapies may require nutritional supplements to limit adverse effects.

- *Corticosteroids:* long-term treatment with oral corticosteroids (e.g. prednisolone) or potentially high dose inhaled corticosteroids is a risk factor for the development of osteoporosis and calcium supplements may be recommended.
- *Methotrexate:* this is a folate-antagonist used in rheumatoid arthritis, Crohn's disease, anticancer chemotherapy and psoriasis. Folic acid 5 mg/day should be prescribed as appropriate prevention of megaloblastic anaemia.
- *Isoniazid:* this antibiotic used in treatment of tuberculosis may have anti-vitamin B_6 effects leading to peripheral neuropathy and so 10mg/day pyridoxine is recommended.
- *Anti-epileptic drugs in pregnancy:* many antiepileptic drugs are associated with birth defects and folic acid 5 mg/day is prescribed to ↓ risk of neural tube defects. Carbamazepine, phenytoin and phenobarbital are associated with risk of neonatal bleeding (including intracranial bleeds) and vitamin K is given to the mother from the 36th week of pregnancy and to the baby at birth.
- *Proguanil:* if this anti-malarial drug is prescribed in pregnancy, supplementation of folic acid 5 mg/day is required.

Drug therapy leading to nutritional deficiencies

Certain drug treatments may reduce the absorption of nutrients.

- *Cholestyramine and orlistat:* cholestyramine (for hyperlipidaemia or jaundice) and orlistat (for obesity) may both ↓ the absorption of fat-soluble vitamins (A, D, E, and K). Supplements may be required and should be taken at a different time to the drug.

- *Anti-epileptic drugs:* enzyme-inducing antiepileptic drugs (e.g. carbamazepine, phenytoin) may induce the metabolism of vitamin D and may be overcome by vitamin D supplementation.
- Diuretics such as bendroflumethiazide and furosemide, especially when used at higher doses for chronic heart failure, may cause hypokalaemia and K^+ supplements or foods rich in K^+ such as bananas may be recommended.
- Metformin (for diabetes) may ↓ absorption of vitamin B_{12}.
- *Insulin and sulphonylureas:* e.g. gliclazide and glibenclamide, (for diabetes) may lead to hypoglycaemia if meals are skipped or insufficient (see 🕮 Chapter 22, 'Hypoglycaemia', p. 457).
- *Methotrexate and folic acid:* see above.

Alcohol and drugs

- Alcohol may enhance the action of many drugs acting on the brain (e.g. antidepressants, benzodiazepines, and antiepileptic drugs) leading to impaired mental ability and increased sedation.
- It is a misconception that all antibiotics interact with alcohol. Of the commonly used agents, there is a significant interaction with metronidazole and tinidazole, which leads to a severe reaction including nausea, vomiting and flushing.
- Even small amounts of alcohol consumed with disulfiram (used in treatment of alcohol dependence) will cause a reaction resulting in facial flushing, tachycardia, giddiness, hypotension and potentially collapse. Some oral medicines and mouthwashes bought over the counter contain sufficient alcohol to precipitate this reaction.
- Major changes in alcohol intake will affect anticoagulant effect of warfarin.

Nutritional status and drug therapy

- Dehydration will enhance the actions of diuretics and other anti-hypertensives and may ↑ the risks of falls in the elderly.
- Low dietary protein intake as well as disease processes may cause hypoalbuminaemia. Many drugs are bound to plasma proteins; this does not necessarily lead to major changes in therapy although correction may be required in therapeutic drug monitoring.
- Enteral tube feeding provides the opportunity for drugs to interact with constituents of the feed. When changing from a tablet to a liquid preparation (e.g. digoxin) the bioavailability may be altered and the dose of drug may need to be changed. ❗ Clinically significant interaction exists between enteral feeds and the following drugs: phenytoin, theophylline, digoxin, ciprofloxacin, tetracyclines, and rifampicin. See 🕮 Chapter 25, 'Enteral feeding and drugs', p. 534).

Metabolic effects of drugs

- Some drugs may alter plasma lipid or glucose levels.
- Drugs that may lead to dyslipidaemia: β-blockers, corticosteroids, thiazide diuretics, anabolic steroids, certain anti-HIV drugs, retinoids, and combined oral contraceptives.
- Drugs that may affect glucose tolerance: thiazide diuretics, corticosteroids.

Effects of drug treatment on appetite and feeding

Some drug treatments may affect appetite (Table 38.4) and thus influence intake.

Table 38.4 Examples of drugs causing changes in appetite

Drug	Appetite ↑ / weight ↑	Appetite ↓ /nausea + vomiting
Anti-diabetic *Sulphonylurea, insulin*	✓	
Anti-emetic *Chlorpromazine*	✓	
Anti-epileptic *Sodium valproate*	✓	
Anti-manic *Lithium*	✓	
Anti-psychotic *Olanzapine, metazapine*	✓	
Digoxin		✓
Corticosteroids *Prednisolone, dexamethasone*	✓	
Antidepressants *SSRIs*		✓
Tricyclics	✓	

SSRI—selective serotonin re-uptake inhibitors.

Some drugs may adversely influence nutritional intake by causing:

- *Taste disturbances:* with ACE Inhibitors, calcium-channel blockers, anticancer chemotherapy drugs.
- *Dry mouth:* antimuscarinic side-effects (for example with tricyclic antidepressants).
- *Oral mucositis:* this is a common side-effect of anticancer drugs (especially with alkylating agents, methotrexate, and fluorouracil) where interference with cell division leads to oral ulceration. This may be exacerbated by poor oral hygiene. Saline mouthwashes are often used for relief and, in the case of fluorouracil, sucking ice while it is infused is recommended.
- *Nausea and vomiting:* digoxin, anticancer chemotherapy, opioids (such as morphine), certain drugs for Parkinson's disease, SSRIs, erythromycin, theophylline.
- *Gastric irritation:* use of non-steroidal anti-inflammatory drugs in particular are associated with gastric damage and ulceration.

Common herb–drug interactions

There are an increasing number of herbal products available with potential to interact with prescribed medication (see Table 38.5 for the most common). The use of herbal drug products is prevalent amongst cancer patients, elderly people, children and adults who are trying to reduce their body weight but is often unreported to health care professionals.

For full details on herb-to-drug interaction, consult Stockley's Herbal Medicines Interactions.

As with conventional medicines, pharmacokinetic and pharmacodynamic type interactions can occur between herbal product and drugs leading to either enhancement or antagonism of drug effect.

St John's wort (*Hypericum perforatum*) used commonly as an antidepressant, is an important liver enzyme inducer and may reduce the effects of a number of drugs. Concomitant treatment should be avoided and patient GP or pharmacist should be consulted.

Table 38.5 Most common herb-drug interactions*

Herb	Interacts with	Comment
St John's wort	Antidepressants	Plasma drug concentration ↓ → ↓ therapeutic effect
	Anti-virals: HIV protease inhibitors *(atazanavir, indinavir, nelfinavir, ritonavir, saquinavir)*	↓ Therapeutic effect
	HIV non-nucleoside reverse transcriptase inhibitors *(efavirenz, nevirapine)*	↓ Therapeutic effect
	Anti-convulsants *(carbamazepine, phenobarbital, phenytoin)*	↓ Control of seizures
	Ciclosporin, tacrolimus	↑ Risk of transplant rejection
	Digoxin	↓ Therapeutic effect
	Oral combined contraceptives, oestrogen-containing patches and vaginal rings	↓ Therapeutic effect
	Voriconazole	↓ Therapeutic effect
	Theophylline	↓ Asthma control
	Warfarin	↓ Anticoagulant effect
	SSRIs *(fluoxetine, paroxetine, sertraline)*	↑ Serotonergic effects
	Triptans *(sumatriptan, naratriptan, rizatriptan, zolmitriptan)*	↓ Therapeutic effect
Ginkgo biloba	Anticoagulant *(warfarin)* Antiplatelet *(aspirin)*	↑ Risk of bleeding
Ginseng	Anticoagulant *(warfarin)*	↑ Risk of bleeding
Dong quai	Anticoagulant *(warfarin)*	↑ Risk of bleeding
Echinacea	Immuno-suppressant	Possible immune-stimulation
Saw palmetto	Anticoagulant *(warfarin)*	Altered anticoagulation

* List is not exhaustive. The reader should consult Stockley's Herbal Medicines Interactions or British National Formulary for further details.

Prescription of nutritional products

All nutritional products within the UK can be bought without a prescription, i.e. none are classified as *prescription-only medication*, which can only be obtained if prescribed by a medical practitioner or other specified health-care professional. However, some nutritional products may be prescribed for specific conditions and are then categorized as drugs, rather than food. This facility is important for patients with chronic conditions who may need expensive special products over a long period of time, e.g. coeliac disease where gluten-free products are required.

Prescribable nutrition products are listed in the *British National Formulary*, Appendix 7, borderline substances as foods that may be prescribed for clinical conditions, alphabetically by brand name of products in the following categories:

- A7.1 Enteral feeds (non-disease specific).
- A7.2 Nutritional supplements (non-disease specific).
- A7.3 Specialized formulas.
- A7.4 Feed supplements.
- A7.5 Feed additives.
- A7.6 Foods for special diets.
- A7.7 Nutritional supplements for metabolic diseases.

Doctors prescribing such products are advised to:

- Endorse the prescription with 'ACBS' i.e. prescribed in accordance with the guidelines from the Advisory Committee on Borderline Substances.
- Ensure that the patient will be adequately monitored in taking the products and that, where necessary, expert hospital supervision, usually by a dietitian, will be available. Good communication between healthcare professionals and patients is required to optimize the products provided and the cost to the prescribing budget.

Further information

The British National Formulary is published twice yearly in March and September. http://bnf.org/ London: BMJ Group and Pharmaceutical Press.

Williamson, E., Driver, S., Baxter, K., (eds) (2009). *Stockley's Herbal Medicines Interactions.* Pharmaceutical Press, London.

Baxter, K. (ed.) (2010). *Stockley's Drug Interactions* . Pharmaceutical Press, London.

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Appendices

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Appendix 1

Weights and measures

Volume

1fl oz = 28.41 ml
1 pint = 568.3 ml
1 litre = 1.76 pint

Table A1.1 Approximate volume conversion

fl oz/pint	ml/l	ml/l	fl oz/pt
1 fl oz	28 ml	50 ml	1.75 fl oz
¼ pint (5 fl oz)	142 ml	100 ml	3.5 fl oz
½pint	284 ml	200 ml	7 fl oz
1 pint	568 ml	500 ml	8.8 fl oz
2 pints	1.1 l	1 l	1.76 pints
3 pints	1.7 l		
4 pints	2.3 l		
5 pints	2.8 l		

Mass/weight

1 ounce = 28.35 g
1 pound (16 oz) = 454 g (0.45 kg)
1 stone (14 lb) = 6.35 kg
1 g = 0.0352 ounces
1 kg = 2.2 pounds

Table A1.2 Approximate weight conversion

g to oz		oz to g	
g	**oz**	**oz**	**g**
1	0.04	1	28
10	0.35	2	57
15	0.53	3	85
20	0.71	4	113
30	1.06	5	142
40	1.41	6	170
50	1.76	7	198
60	2.12	8	227
70	2.47	9	255
80	2.82	10	284
90	3.17	11	312
100	3.53	12	340
		13	368
		14	397
		15	425
		16	454

Appendix 2

Anthropometrics

Length/height conversions

1 inch = 2.54 cm
1 foot (12 in) = 30.48 cm
1 yard (36 in) = 91.44 cm
1 cm = 0.394 inch
1 m = 39.37 inches

Table A2.1 Approximate length conversions

Inches to centimeters		Centimetres to inches	
in	**cm**	**cm**	**in**
1	2.54	1	0.39
2	5.08	2	0.79
3	7.62	3	1.18
4	10.16	4	1.57
5	12.70	5	1.97
6	15.25	6	2.36
7	17.78	7	2.76
8	2.32	8	3.15
9	22.86	9	3.54
10	25.40	10	3.94
20	50.80	20	7.87
30	76.20	30	11.81
40	101.60	40	15.75
50	127.00	50	19.69
60	152.40	60	23.62
70	177.80	70	27.56
80	203.20	80	31.50
90	228.60	90	35.43
100	254.0	100	39.37

Table A2.2 Approximate height conversions

m	ft and in	m	ft and in
1.22	4′0″	1.6	5′3″
1.23	4′½″	1.61	5′3½″
1.24	4′1″	1.63	5′4″
1.26	4′1 ½″	1.64	5′4½″
1.27	4′2″	1.65	5′5 ″
1.28	4′2 ½″	1.66	5′5½″
1.29	4′3″	1.68	5′6″
1.31	4′3 ½.″	1.69	5′6½″
1.32	4′4″	1.7	5′7″
1.33	4′4½″	1.71	5′7 ½″
1.35	4′5″	1.73	5′8″
1.36	4′5½″	1.74	5′8 ½″
1.37	4′6″	1.75	5′9″
1.38	4′6½″	1.76	5′9 ½″
1.4	4′7″	1.78	5′10″
1.41	4′7 ½″	1.79	5′10½″
1.42	4′8″	1.8	5′11 ″
1.43	4′8 ½″	1.82	5′11½″
1.45	4′9″		
1.46	4′9 ½″	1.83	6′0″
1.47	4′10″	1.84	6′0 ½″
1.49	4′10½″	1.85	6′1″
1.5	4′11 ″	1.87	6′1 ½″
1.51	4′11½″	1.88	6′2″
		1.89	6′2 ½″
1.52	5′0″	1.9	6′3″
1.54	5′0 ½″	1.92	6′3½″
1.55	5′1″	1.93	6′4 ″
1.56	5′1 ½″	1.94	6′4½″
1.57	5′2″	1.96	6′5″
1.59	5′2 ½″	1.97	6′5½″
		1.98	6′6″

Mass/weight conversions

1 oz = 28.35 g
1 lb = 454 g or 0.45 kg
1 g = 0.0352 oz
1 kg = 2.2 lb

Table A2.3 Approximate weight conversions

kg	st	lb	kg	st	lb	kg	st	lb	kg	st	lb
0.5		1	44	6	13	83	13	1	122	19	3
1		2	45	7	1	84	13	3	123	19	
1.5		3	46	7	3	85	13	6	124	19	7
2		4	47	7	6	86	13	7	125	19	10
2.5		6	48	7	8	87	13	10	126	19	11
3		7	49	7	10	88	13	11	127	20	0
3.5		8	50	7	13	89	14	0	128	20	1
4		9	51	8	0	90	14	3	129	20	5
4.5		10	52	8	3	91	14	4	130	20	7
5		11	53	8	4	92	14	7	131	20	8
5.5		12	54	8	7	93	14	8	132	20	11
6		13	55	8	10	94	14	11	133	20	13
			56	8	11	95	14	13	134	21	1
10	1	8	57	9	0	96	15	1	135	21	3
15	2	6	58	9	1	97	15	4	136	21	6
20	3	1	59	9	4	98	15	6	137	21	8
21	3	4	60	9	6	99	15	8	138	21	10
22	3	7	61	9	8	100	15	10	139	21	13
23	3	8	62	9	11	101	15	13	140	22	0
24	3	11	63	9	13	102	16	1	141	22	3
25	3	13	64	10	1	103	16	3	142	22	5
26	4	1	65	10	3	104	16	6	143	22	7
27	4	3	66	10	6	105	16	7	144	22	10
28	4	6	67	10	7	106	16	10	145	22	11
29	4	8	68	10	10	107	16	11	146	23	0

Table A2.3 (*Contd.*)

kg	st	lb	kg	st	lb	kg	st	lb	kg	st	lb
30	4	10	69	10	13	108	17	0	147	23	1
31	4	13	70	11	0	109	17	3	148	23	5
32	5	0	71	11	3	110	17	5	149	23	6
33	5	3	72	11	4	111	17	7	150	23	8
34	5	6	73	11	7	112	17	8	151	23	11
35	5	7	74	11	8	113	17	11	152	23	13
36	5	10	75	11	11	114	17	13	153	24	1
37	5	11	76	12	0	115	18	1	154	24	3
38	6	0	77	12	1	116	18	5	155	24	6
39	6	1	78	12	5	117	18	6	156	24	7
40	6	3	79	12	6	118	18	8	17	24	10
41	6	7	80	12	8	119	18	10	158	24	13
42	6	8	81	12	10	120	18	13	159	25	0
43	6	11	82	12	13	121	19	0	160	25	3

Body mass index

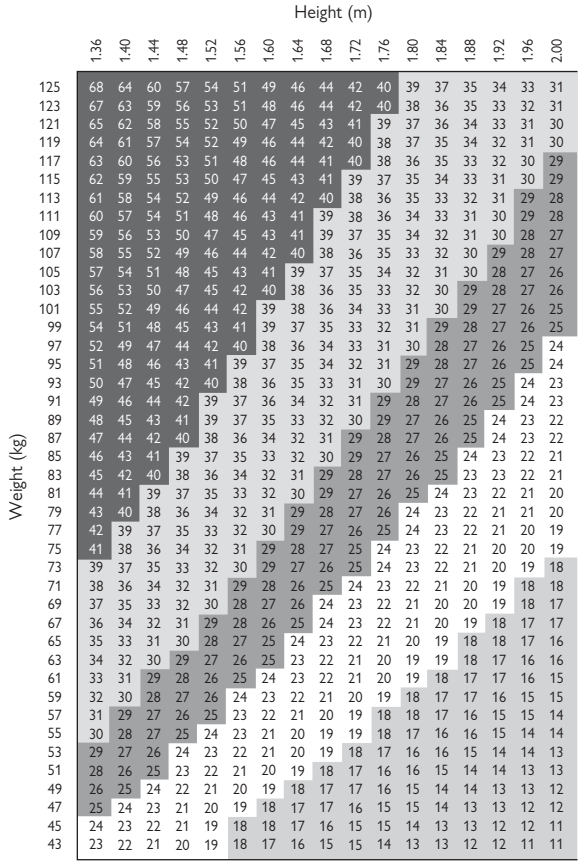

Fig. A2.1 Adult BMI ready reckoner.

Table A2.4 WHO cut-offs for BMI in adults

BMI (kg/m^2)	Weight status	Risk of co-morbidities
Below 18.5	Underweight	Low
18.5–24.9	Normal	Average
25.0–29.9	Overweight	Increased
30.0–39.9	Obese	Moderate–severe
Above 40	Very obese	Severe

Table A2.5 International cut-off points for body mass index for overweight and obesity between 2 and 18 years*

Age (years)	Body mass index 25 kg/m^2		Body mass index 30 kg/m^2	
	Males	Females	Males	Females
2	18.41	18.02	20.09	19.81
2.5	18.13	17.76	19.80	19.55
3	17.89	17.56	19.57	19.36
3.5	17.69	17.40	19.39	1923
4	17.55	17.28	19.29	19.15
4.5	17.47	17.19	19.26	19.12
5	17.42	17.15	19.30	19.17
5.5	17.45	17.20	19.47	19.34
6	17.55	17.34	19.78	19.65
6.5	17.71	17.53	20.23	20.0
7	17.92	17.75	20.63	20.51
7.5	18.16	18.03	21.09	21.01
8	18.44	18.35	21.6	21.57
8.5	18.76	18.69	22.17	22.18
9	19.10	19.07	22.77	22.81
9.5	19.46	19.45	23.39	23.46
10	19.84	19.86	24.00	24.11
10.5	20.20	20.29	24.57	24.77
11	20.55	20.74	25.10	5.42
11.5	20.89	21.20	25.58	26.05
12	21.22	21.68	26.02	26.67
12.5	21.56	22.1	26.43	27.24
13	21.91	22.58	26.84	27.76
13.5	22.27	22.98	27.25	28.20
14	22.62	23.34	27.63	28.57
14.5	22.96	23.66	27.98	28.87
15	23.29	23.94	28.30	29.11
15.5	23.60	24.17	28.60	29.29

Table A2.5 (*Contd.*)

Age (years)	Body mass index 25 kg/m^2		Body mass index 30 kg/m^2	
	Males	Females	Males	Females
16	23.90	24.37	28.88	29.43
16.5	24.19	24.54	29.14	29.56
17	24.46	24.70	29.41	29.69
17.5	24.73	24.85	29.70	29.84
18	25	25	30	30

* From Cole TJ, Bellizzi MC, Flegal KM, *et al.* (2000). *Br. Med. J.* **320** (7244), 1240, Table 4. 6th May 2000 edition. Amended with permission from the BMJ publishing group.

Waist circumference cut-offs for risk of metabolic complications, and mindex and demiquet measures of adiposity

Table A2.6 WHO waist circumference cut offs and risk of associated metabolic complications

	Increased risk	Substantially increased risk
♂	≥94 cm	≥102 cm
♀	≥80 cm	≥88 cm

Mindex and demiquet

Measures of adiposity >64 years using demispan as proxy for height:

Mindex (♀) = wt (kg)/demispan (m)

Demiquet (♂) = wt (kg)/demispan (m^2)

Table A2.7 Deciles for mindex (♀)*

	10	20	30	40	50	60	70	0	90
64–74 years	68.3	73.3	77.8	82.2	84.8	88.4	92.3	99.9	110.6
75+ years	63.1	68.4	73.6	78.1	81.7	85.3	88.4	94.6	102.2

* Reprinted from Lehmann, B., Bassey, E. J., Morgana, K. (1991). Normal values for weight, skeletal size and body mass indices in 890 men and women aged over 65 years. *Clin. Nutr.* **1**, 18–23. Lehman Copyright (1991) with permission from Elsevier.

Table A2.8 Deciles for demiquet (♂)*

	10	20	30	40	50	60	70	80	90
64–74 y	87.6	96.1	99.6	102.4	106.7	111.6	117.1	123.7	130.7
75+y	84.5	92.8	98.9	103.1	106.3	109.1	113.4	119.3	125.3

* Reprinted from Lehmann, B., Bassey, E. J., Morgana, K. (1991). Normal values for weight, skeletal size and body mass indices in 890 men and women aged over 65 years. *Clin. Nutr.* **1**, 18–23. Lehman Copyright (1991) with permission from Elsevier.

Table A2.9 Mid-arm circumference (MAC) (cm)*

Age group (y)	Percentile						
	5th	10th	25th	50th	75th	90th	95th
♂							
18–74	26.4	27.6	29.6	31.7	33.9	36.0	37.3
18–24	25.7	27.1	28.7	30.7	32.9	35.5	37.4
25–34	27.0	28.2	30.0	32.0	34.4	36.5	37.6
35–44	27.8	28.7	30.7	32.7	34.8	36.3	37.1
45–54	26.7	27.8	30.0	32.0	34.2	36.2	37.6
55–64	25.6	27.3	29.6	31.7	33.4	35.2	36.6
65–74	25.3	26.5	28.5	30.7	32.4	34.4	35.5
♀							
18–74	23.2	24.3	26.2	28.7	31.9	35.2	37.8
18–24	22.1	23.0	24.5	26.4	28.8	31.7	34.3
25–34	23.3	24.2	25.7	27.8	30.4	34.1	37.2
35–44	24.1	25.2	26.8	29.2	32.2	36.2	38.5
45–54	24.3	25.7	27.5	30.3	32.9	36.8	39.3
55–64	23.9	25.1	27.7	30.2	33.3	36.3	38.2
65–74	23.8	25.2	27.4	29.9	32.5	35.3	37.2

* Bishop, C.W., Bowen, P.E., and Ritchley, S.L. (1981). Norms for nutritional assessment of American adults by upper arm anthropometry. *Am. J. Clin. Dietet.* **34,** 2530–9.

Upper arm anthropometry

Table A2.10 Mid-arm muscle circumference (MAMC) (cm)*

Age group (y)	Percentile						
	5th	10th	25th	50th	75th	90th	95th
♂							
18–74	23.8	24.8	26.3	27.9	29.6	31.4	32.5
18–24	23.5	24.4	25.8	27.2	28.9	30.8	32.3
25–34	24.2	25.3	26.5	28.0	30.0	31.7	32.9
35–44	25.0	25.6	27.1	28.7	30.3	32.1	33.0
45–54	24.0	24.9	26.5	28.1	29.8	31.5	32.6
55–64	22.0	24.4	26.2	27.9	29.6	31.0	31.8
65–74	22.5	23.7	25.4	26.9	28.5	29.9	30.7
♀							
18–74	18.4	19.0	20.2	21.8	23.6	25.8	27.4
18–24	17.7	18.5	19.4	20.6	22.1	23.6	24.9
25–34	18.3	18.9	20.0	21.4	22.9	24.9	26.6
35–44	18.5	19.2	20.6	22.0	24.0	26.1	27.4
45–54	18.8	19.5	20.7	22.2	24.3	26.6	27.8
55–64	18.6	19.5	20.8	22.6	24.4	26.3	28.1
65–74	18.6	19.5	20.8	22.5	24.4	26.5	28.1

* Bishop, C.W., Bowen, P.E., and Ritchley, S.L. (1981). Norms for nutritional assessment of American adults by upper arm anthropometry. *Am. J. Clin. Dietet.* **34,** 2530–9.

Table A2.11 Triceps skin-fold thickness (cm)*

Age group (y)	Percentile						
	5th	10th	25th	50th	75th	90th	95th
♂							
18–74	4.5	6.0	8.0	11.0	15.0	20.0	23.0
18–24	4.0	5.0	7.0	9.5	14.0	20.0	23.0
25–34	4.5	5.5	8.0	12.0	16.0	21.5	24.0
35–44	5.0	6.0	8.5	12.0	15.5	20.0	23.0
45–54	5.0	6.0	8.0	11.0	15.0	20.0	25.5
55–64	5.0	6.0	8.0	11.0	14.0	18.0	21.5
65–74	4.5	5.5	8.0	11.0	15.0	19.0	22.0
♀							
18–74	11.0	13.0	17.0	22.0	28.0	24.0	37.0
18–24	9.4	11.0	14.0	18.0	24.0	30.0	34.0
25–34	10.5	12.0	16.0	21.0	26.5	33.5	37.0
35–44	12.0	14.0	18.0	23.0	29.5	35.5	39.0
45–54	13.0	15.0	20.0	25.0	30.0	36.0	40.0
55–64	11.0	14.0	19.0	25.0	30.5	35.0	39.0
65–74	11.5	14.0	18.0	23.0	28.0	33.0	36.0

*Bishop, C.W., Bowen, P.E., and Ritchley, S.L. (1981). Norms for nutritional assessment of American adults by upper arm anthropometry. *Am. J. Clin. Dietet.* **34**, 2530–9.

Child growth foundation charts

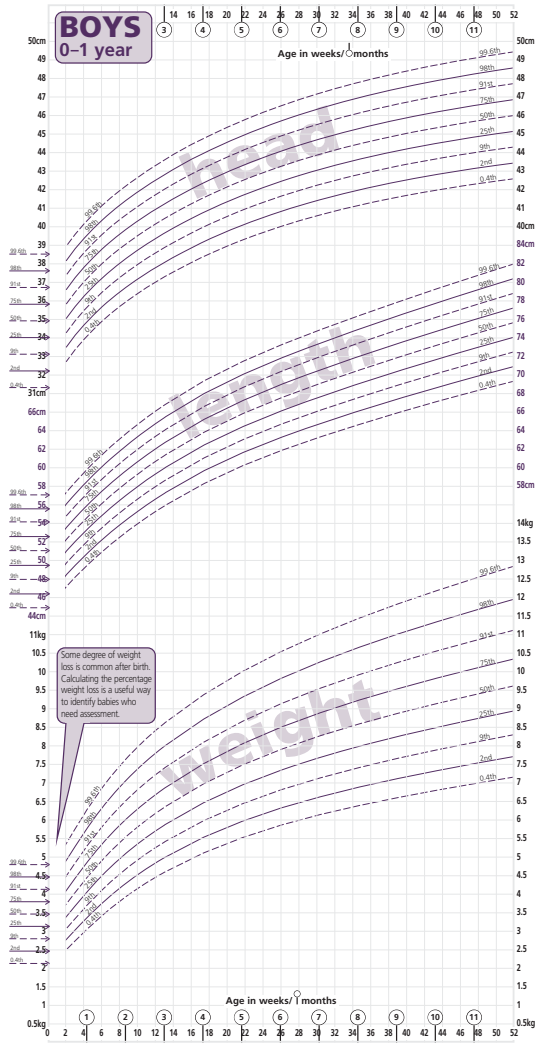

Fig. A2.2 UK-WHO growth chart for boys, 0–1 years. Reproduced with the kind permission of the Department of Health.

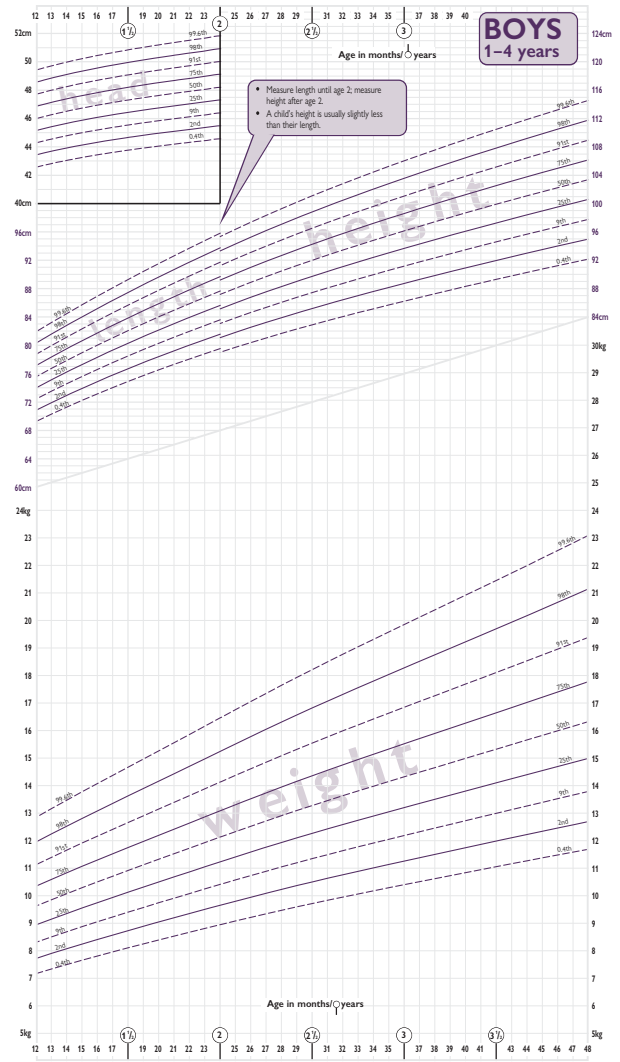

Fig. A2.3 UK-WHO growth chart for boys 1–4 years. Reproduced with the kind permission of the Department of Health.

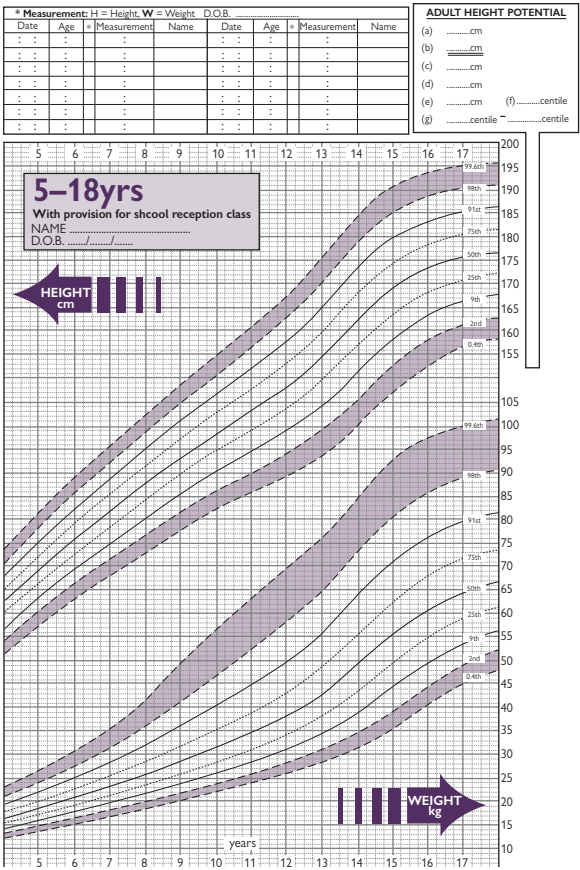

Fig. A2.4 Child Growth Foundation 9-centile growth chart for boys 5–18 years. Reproduced with the kind permission of the Child Growth Foundation.

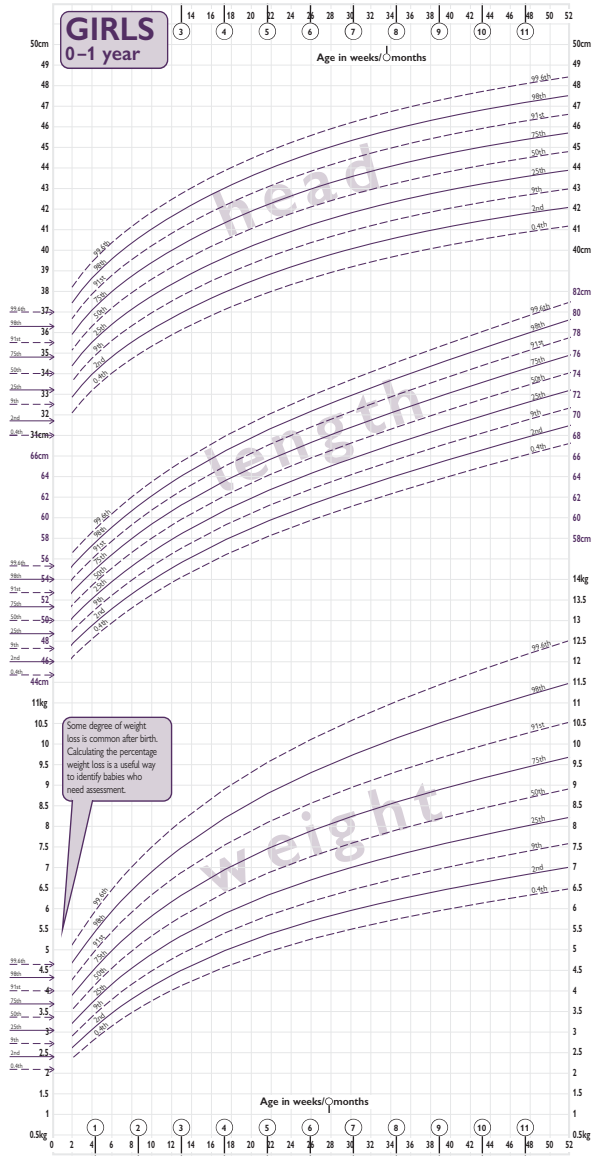

Fig. A2.5 UK-WHO growth chart for girls 0–1 year. Reproduced with the kind permission of the Department of Health.

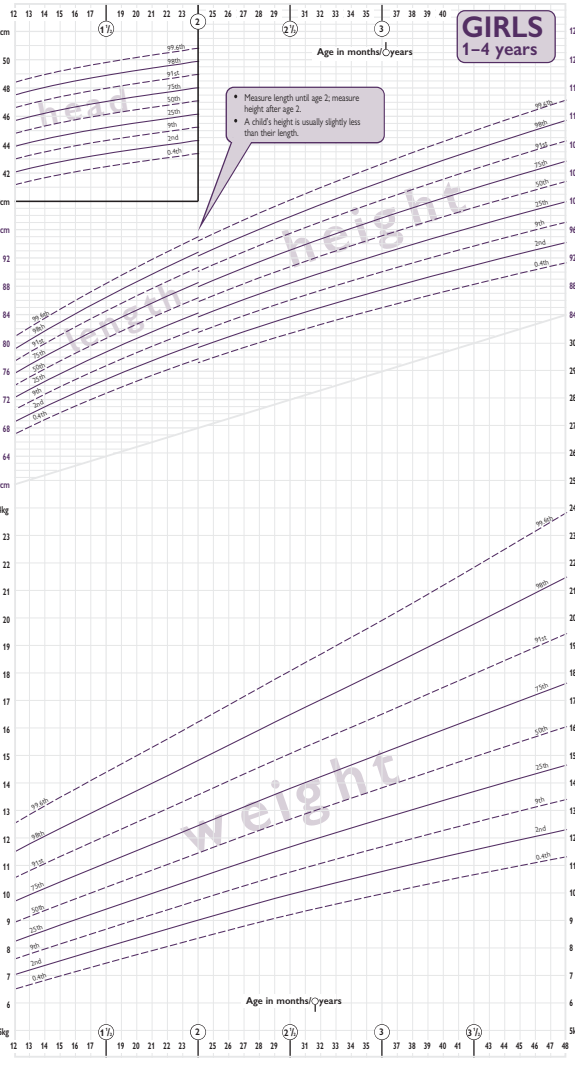

Fig. A2.6 UK-WHO growth chart for girls 1–4 years. Reproduced with the kind permission of the Department of Health.

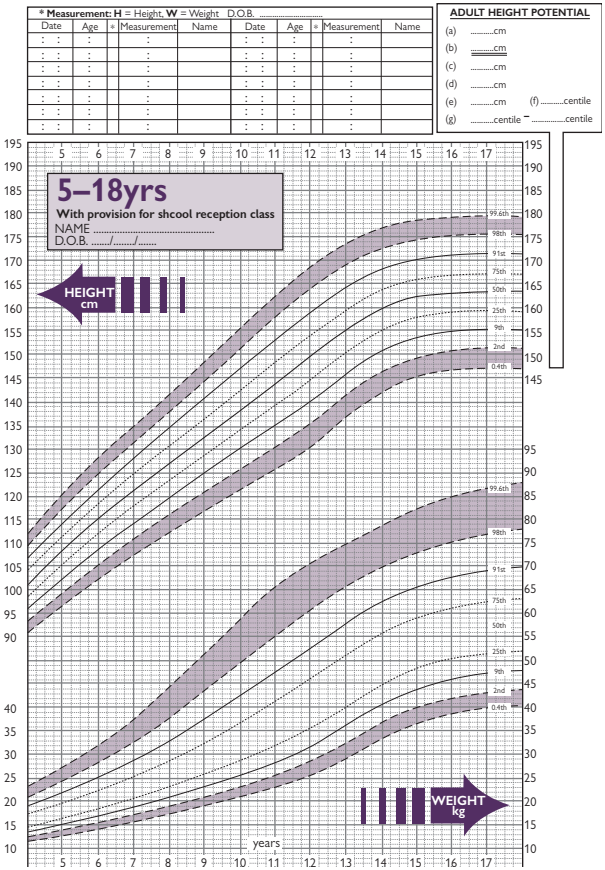

Fig. A2.7 UK-WHO growth chart for girls 5–18 years. Reproduced with the kind permission of the Child Growth Foundation.

Appendix 3

Conversion factors

Dietary energy

Units used in energy balance

1000 J = 1 kJ
1000 kJ = 1 MJ
1 kcal = 4.184 kJ (The Royal Society (London) recommended conversion factor)
1 kJ = 0.239 kcal
1 W = 1 J/s
0.06 W = 1 kJ/min
86.4 W = 1 kJ.24 h

Table A3.1 Nutrient energy yields

Nutrient	Energy yield per gram	
	kcal	kJ
Protein	4	17
Carbohydrate	3.75	16
Fat	9	37
Alcohol	7	29
Medium chain triglyceride (MCT)	8.4	35

Protein/nitrogen

Dietary protein/dietary nitrogen

Dietary protein (g) = dietary nitrogen (g) × 6.25[1]
Dietary nitrogen (g) = dietary protein (g) ÷ 6.25[1]

[1] This conversion factor is only appropriate for a mixture of foods. For milk or cereals alone, the factors 6.4 or 5.7 should be used.

Vitamin A

The active vitamin A content of a diet is usually expressed in retinol equivalents.
1 μg retinol equivalent = 1 μg retinol or 6 μg β carotene
1 IU vitamin A = 0.3 μg retinol or 0.6 μg β carotene

Vitamin D

1 μg vitamin D = 40 IU
1 IU = 0.025 μg vitamin D

Nicotinic acid/tryptophan

1 mg nicotinic acid = 60 mg tryptophan

Nicotinic acid content mg equivalents = nicotinic acid (mg) + (tryptophan (mg)/60

Mineral content of compounds and solutions

Table A3.2 Mineral content of compounds and solutions

Solution/compound	Mineral content	
1 g sodium chloride	393 mg Na	17 mmol Na
1 g sodium bicarbonate	273 mg Na	12 mmol Na
1 g potassium bicarbonate	524 mg K	13.4 mmol K
1 g calcium chloride (hydrated)	273 mg C	7 mmol Ca
1 g calcium carbonate	400 mg Ca	10 mmol Ca
1 g calcium gluconate	93 mg Ca	2.3 mmol Ca
1 l normal saline	3450 mg Na	150 mmol Na

Appendix 4

Energy expenditure prediction equations

The following equations are used as a basis for calculating energy expenditure (DRVs):[1] They have been derived from a large number of studies,[2] including the classic work of Harris and Benedict (1919),[3] which measured energy expenditure in healthy subjects. In clinical practice, these are often referred to as the Schofield equations.

Table A4.1 Equations for estimating basal metabolic rate from weight (MJ/24 h)*

Age range (years)	♂	♀
10–18	0.074W + 2.754	0.056W + 2.898
18–30	0.063W + 2.896	0.062W + 2.036
30–60	0.048W + 3.653	0.034W + 3.538
>60	0.049W + 2.459	0.038W + 2.755

*W = body weight in kg.

Table A4.2 Equations for estimating basal metabolic rate from weight (kcal/24 h)*

Age range (years)	♂	♀
10–18	17.69W + 659	13.39W + 693
18–30	15.06W + 692	14.83W + 487
30–60	11.48W + 874	8.13W + 846
>60	11.72W + 588	9.09W + 659

*W = body weight in kg.

[1] Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. HMSO, London.
[2] Schofield, W.N. (1985). Predicting basal metabolic rate, new standards and review of previous work. *Hum. Nutr. Clin. Nutr.* **39C** (Suppl. 1), 5–41.
[3] Harris, J.A. and Benedict, F.G. (1919). *A biometric study of basal metabolism in man*. Carnegie Institution of Washington, Washington DC.

Notes on tables

- The equations in Table A4.2 were derived from those in Table A4.1 using a conversion factor of 1 kcal = 0.004182 MJ.
- The equations published for children aged 10–18 years are rarely used for those aged less than 15 years.
- The equations published for men and women aged >60 years are derived from data from only 50 and 38 individuals, respectively, so are least robust. All other equations are derived from data from between 300 and 2900 individuals.

Table A4.3 Calculated physical activity level (PAL) values for light, moderate, and heavy activity (occupational and non-occupational)*

Non occupational activity level	Occupational activity level					
	Light		Moderate		Heavy	
	♂	♀	♂	♀	♂	♀
Sedentary	1.4	1.4	1.6	1.5	1.7	1.5
Moderately active	1.5	1.5	1.7	1.6	1.8	1.6
Very active	1.6	1.6	1.8	1.7	1.9	1.7

*Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. HMSO, London.

Appendix 5

Clinical chemistry reference ranges

❗ The values given are for guidance purposes only. Values will vary between laboratories. Check normal ranges in use at applicable setting before making clinical decisions.

Table A5.1 Adult normal values*

Substance	Value	Substance	Value
Albumin	32–50 g/l	Red Cell Count	
Bicarbonate	20–29 mmol/l	Males	$4.5–6.5\times10^{12}$/l
Bilirubin	<17 μmol/l	Females	$3.8–5.8\times10^{12}$/l
Calcium	2.15–2.55 mmol/l	Mean cell haemoglobin (MCH)	27–32 pg
Chloride	97–107 mmol/l	Mean cell volume (MCV)	77–95 fl
Total cholesterol	<5 mmol/l	Mean cell haemoglobin conc	32–36 g/dl
Creatinine	60–125 mmol/l	White blood count (WBC)	$4.0–11.0\times10^{9}$/l
Glucose (fasting)	<6.1 mmol/l	Neutrophils	$2.0–7.5\times10^{9}$/l
Phosphate	0.7–1.5 mmol/l	Eosinophils	$0.04–0.4\times10^{9}$/l
Magnesium	0.7–1.0 mmol/l	Monocytes	$0.2–0.8\times10^{9}$/l
Osmolality	278–305 mosmol/kg	Basophils	$0.0–0.1\times10^{9}$/l
Potassium	3.5–5.0 mmol/l	Lymphocytes	$1.5–4.5\times10^{9}$/l
Sodium	135–150 mmol/l	Platelets	$150–400\times10^{9}$/l
Total protein	63–80 g/l	Erythrocyte sedimentation rate	2–12 mm/ 1st hour
Triglycerides	0.55–1.90 mmol/l	Ferritin (varies with age)	14–200 μg/l
Urate	0.14–0.46 mmol/l	Pre-menopausal women	14–148 μg/l
Urea	3.0–6.5 mmol/	Serum B_{12}	150–700 ng/l
Haemoglobin		Serum folate	2.0–11.0 μg/l
Male	13.0–18.0 g/dl	Red cell folate	150–700 μg/l
Female	11.5–16.5 g/dl	Prothrombin time (PT)	12–14 s
Newborn male	16.0–21.0 g/dl	Activated partial thromboplastin time (APTT)	26.0 33.5 s
2–6 month female	10.5–12.5 g/dl		
Haematocrit (PCV)		Thrombin time (TT)	± 3s of control
Male	0.40–0.52		
Female	0.36 0.47		

* Adapted from Provan, J. (2005) *Oxford handbook of clinical and laboratory investigation*, 2nd edn. By permission of Oxford University Press, Oxford.

Table A5.2 Adult normal urine levels

Substance	Value
Albumin	< 20 mg/24 h
Calcium	<7.5 mmol/24 h
Creatinine	9–15 mmol/24 h
Phosphate	15–50 mmol/24 h
Osmolality	50–1500 mosmol/24 h
Potassium	14–120 mmol/24 h
Protein	< 150 mg/24 h
Sodium	100–250 mmol/24 h
Urate	<3.0 mmol/24 h
Urea	250–600 mmol/24 h

Table A5.3 Adult normal faecal values

Substance	Value
Faecal fat	<18 mmol/24 h
Nitrogen	70–140 mmol/24 h

Appendix 6

Dietary reference values (DRVs)

Estimated average requirements

Table A6.1 Estimated average requirements(EAR; MJ/day) according to body weight and physical activity level (PAL)*

Body weight (kg)	BMR† MJ/day	PAL				
		1.4	1.5	1.6	1.8	2.0
♂						
30	4.97	7.0	7.5	8.0	9.0	9.9
35	5.34	7.5	8.0	8.6	9.6	10.7
40	5.71	8.0	8.6	9.1	10.3	11.4
45	6.08	8.5	9.1	9.7	11.0	12.2
50	6.45	9.0	9.7	10.3	11.6	12.9
55	6.82	9.6	10.2	10.9	12.3	13.6
60	7.19	10.1	10.8	11.5	12.9	14.4
65	7.56	10.6	11.3	12.1	13.6	15.1
♀						
30	4.58	6.4	6.9	7.3	8.2	9.2
35	4.86	6.8	7.3	7.8	8.7	9.7
40	5.14	7.2	7.7	8.2	9.2	10.3
45	5.42	7.6	8.1	8.7	9.8	10.8
50	5.70	8.0	8.5	9.1	10.3	11.4
55	5.98	8.4	9.0	9.6	10.8	12.
60	6.26	8.8	9.4	10.0	11.3	12.5

*Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. HMSO, London.

†BMR, Basal metabolic rate calculated as per Table A4.1.

Table A6.2 Estimated average requirements (EARs) for energy of children 0–18 years*

Age	EAR MJ/d (kcal/day)	
	♂	♀
0–3 months	2.28 (545)	2.16 (515)
4–6 months	2.89 (690)	2.69 (645)
7–9 months	3.44 (825)	3.20 (765)
10–12 months	3.85 (920)	3.61 (865)
1–3 years	5.15 (1 230)	4.86 (1 165)
4–6 years	7.16 (1 715)	6.46 (1 545)
7–10 years	8.24 (1 970)	7.28 (1 740)
11–14 years	9.27 (2 220)	7.72 (1 845)
15–18 years	11.51 (2 775)	8.83 (2 110)

*Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. HMSO, London.

Reference nutrient intakes

Table A6.3 Reference nutrient intakes for protein (RNI)*

Age	Weight (kg)	RNI (g/day)
0–3 months	5.9	12.5
4–6 months	7.7	12.7
7–9 months	8.8	13.7
10–12 months	9.7	14.9
1–3 years	12.5	14.5
4–6 years	17.8	19.7
7–10 years	28.3	28.3
♂		
11–14 years	43.0	42.1
15–18 years	64.5	55.2
19–50 years	74.0	55.5
50+ years	71.0	53.3
♀		
11–14 years	43.8	41.2
15–18 years	55.5	45.4
19–50 years	60.0	45.0
50+ years	62.0	46.5
Pregnancy		
Lactation		+6.0
0–4 months		+11.0
4+ months		+8.0

*Department of Health (1991). *Dietary reference values for food and nutrients for the United Kingdom*. HMSO, London.

Table A6.4 RNIs for vitamins*

Age	Thiamin (mg/day)	Riboflavin (mg/d)	Niacin (mg/day)†	Vitamin B_6 (mg/day)‡	Vitamin B_{12} (µg/day)	Folate (µg/day)	Vitamin C (mg/day)	Vitamin A (µg/day)	Vitamin D
0–3 months	0.2	0.4	3	0.2	0.3	50	25	350	8.5
4–6 months	0.2	0.4	3	0.2	0.3	50	25	350	8.5
7–9 months	0.2	0.4	4	0.3	0.4	50	25	350	7
10–12 months	0.3	0.4	5	0.4	0.4	50	25	350	7
1–3 years	0.5	0.6	8	0.7	0.5	70	30	400	7
4–6 years	0.7	0,8	11	0.9	0.8	100	30	500	—
7–10 years	0.7	1.0	12	1.0	1.0	150	30	500	—
♂									
11–14 years	0.9	1.2	15	1.2	1.2	200	35	600	—
15–18 years	1.1	1.3	18	1.5	1.5	200	40	700	—
19–50 years	1.0	1.3	17	1.4	1.5	200	40	700	—
50+ years	0.9	1.3	16	1.4	1.5	200	40	700	10

(continued)

Table A6.4 *(contd.)*[*]

Age	Thiamin (mg/day)	Riboflavin (mg/d)	Niacin (mg/day)[†]	Vitamin B_6 (mg/day)[‡]	Vitamin B_{12} (µg/day)	Folate (µg/day)	Vitamin C (mg/day)	Vitamin A (µg/day)	Vitamin D
♀									
11–14 years	0.7	1.1	12	1.0	1.2	200	35	600	—
15–18 years	0.8	1.1	14	1.2	1.5	200	40	600	—
19–50 years	0.8	1.1	13	1.2	1.5	200	40	600	—
50+ years	0.8	1.1	12	1.2	1.5	200	40	600	10
Pregnancy	+0.1[§]	+0.3	[¶]	[¶]	[¶]	+100	+10	+100	10
Lactation									
0–4 months	+0.2	+0.5	+2	[¶]	+0.5	+60	~+30	+350	10
4+ months	+0.2	+0.5	+2	[¶]	+0.5	+60	+30	+350	10

[†]Nicotinic acid equivalent.
[‡]Based on protein providing 14.7% of the EAR for energy.
[§]Last semester only.
[¶]No increment.
*Data from Department of Health (1991). *Dietary reference values for food and nutrients from the United Kingdom*. HMSO, London. Table from appendix 6.2, pp. 716–17 of Thomas, B. (2001). *Manual of dietetic practice*, 3rd edn. Blackwell Science, Oxford.

Table A6.5 RNIs for minerals*

Age	Ca (mg/day)	P (mg/d)	Mg (mg/day)	Na (mg/day)	K (mg/day)	Cl (mg/day)	Fe (mg/day)	Zn (mg/day)	Cu (mg/day)	Se (μg/day)	I (μg/day)
0–3 months	525	400	55	210	800	320	1.7	4.0	0.2	10	50
4–6 months	525	400	60	280	850	400	4.3	4.0	0.3	13	60
7–9 months	525	400	75	320	700	500	7.8	5.0	0.3	10	60
10–12 months	525	400	80	350	700	500	7.8	5.0	0.3	10	60
1–3 years	350	270	85	500	800	800	6.9	5.0	0.4	15	70
4–6 years	450	350	120	700	1100	1100	6.1	6.5	0.6	20	100
7–10 years	550	450	200	1200	2000	1800	8.7	7.0	0.7	30	110
♂											
11–14 years	1000	775	280	1600	3100	2500	11.3	9.0	0.8	45	130
15–18 years	1000	775	300	1600	3500	2500	11.3	9.5	1.0	70	140
19–50 years	700	550	300	1600	3500	2500	8.7	9.5	1.2	75	140
50+ years	700	550	300	1600	3500	2500	8.7	9.5	1.2	75	140

(continued)

Table A6.5 (*Contd.*)*

Age	Ca (mg/day)	P (mg/d)	Mg (mg/day)	Na (mg/day)	K (mg/day)	Cl (mμg/day)	Fe (mg/day)	Zn (mg/day)	Cu (mg/day)	Se (μg/day)	I (μg/day)
♀											
11–14 years	800	625	280	1600	3100	2500	14.8‡	9.0	0.8	45	130
15–18 years	800	625	300	1600	3500	2500	14.8‡	7.0	1.0	60	140
19–50 years	700	550	270	1600	3500	2500	14.8‡	7.0	1.2	60	140
50+ years	700	550	270	1600	3500	2500	8.7	7.0	1.2	60	140
Pregnancy	‡	‡	‡	‡	‡	‡	‡	‡	‡	‡	‡
Lactation											
0–4 months	+550	+440	+50	‡	‡	‡	‡	+6.0	+0.3	+15	‡
4+ months	+550	+440	+50								

‡No increment.

‡Supplements required if menstrual losses are high.

*Data from Department of Health (1991). *Dietary reference Values for food and nutrients from the United Kingdom.* HMSO, London. Table from appendix. 6.2, pp. 716–17 of Thomas, B. (2001). *Manual of dietetic practice*, 3rd edn. Blackwell Science, Oxford.

Appendix 7

Nutritional composition of common foods

Protein exchanges

Table A7.1 Foods containing approximately 6 g protein

	Weight (g)	Description
Meat—cooked, e.g. lean beef, lamb, pork	25	1 small slice
Sausage—cooked	40	1 large sausage
Poultry—cooked	25	1 small slice
Fish—cooked or tinned	40	1 tablespoon
Fish fingers—cooked	45	2 fingers
Egg	50	1 average hen's egg
Cheese e.g. Cheddar	25	Matchbox-sized piece
Milk—full fat, semi-or skimmed	200	Average glass ($^1/_3$ pt)
Milk powder—skimmed	15	4 teaspoons
Yogurt	125	Small pot
Pulses—cooked e.g. lentils	100	3 tablespoons
Nuts, e.g almonds, peanuts	25	15–25 nuts
Hummus	100	3 tablespoons

Table A7.2 Foods containing approximately 2 g protein

	Weight (g)	Description
Bread—white/wholegrain	25	1 large thin slice
Potato—boiled	125	2 × size of hen's egg
Potato—mashed	110	2 scoops
Chips	50	8 large
Rice—boiled	75	1½ tablespoons
Pasta—boiled	50	1 tablespoon
Breakfast cereal—cornflake type	25	small average portion
Breakfast cereal—wheat biscuit type	20	1 biscuit
Biscuits, e.g. plain digestive	30	2 biscuits
Cream crackers	20	4 crackers
Cake—plain sponge	25	½ small average slice
Ice cream—plain	50	1 small scoop
Baked beans	40	1 tablespoon

Table A7.3 Foods containing little protein per typical portion

	Protein content*		
Butter	0.6	Apples, pineapple	0.4
Margarine	Trace	Pear	0.3
Cooking oil	Trace	Melon	0.6
Sugar	0.5	Apple juice	0.1
Golden syrup	0.3	Cranberry juice	Trace
Jam, honey	0.5	Boiled sweets	Trace
Marmalade	0.1	Peppermints	0.5
Carrots, boiled	0.6	Cola, lemonade	Trace
Celery, cucumber, lettuce	0.5–0.8	Tea, infusion	0.1
Swede, boiled	0.3	Coffee, infusion	0.2

*g/100 g food.

Carbohydrate exchanges

Table A7.4 Foods containing approximately 10 g carbohydrate

	Weight (g)	Description
Wholemeal bread	25	1 thin slice/large loaf
White bread	20	1 thin slice/small loaf
Potatoes—boiled	60	1 size of hen's egg
Potatoes—mashed	60	1 scoop
Potatoes—roast	40	1 very small
Sweet potato—boiled	50	1 size of hen's egg
Rice—boiled, brown, white	30	¾ tablespoon
Pasta—boiled, e.g. spaghetti, macaroni	50	1 tablespoon
Pulses, e.g. lentils	60	2 tablespoons
Peas—frozen	100	3 tablespoons
Parsnip—boiled	80	1 medium
Sweetcorn—boiled	50	2 tablespoons
Thick soup, e.g. tinned vegetable	100	1 small tin
Thin soup, e.g. minestrone	250	1 standard mug
Sausages	100	2 large sausages
Beefburger, economy	100	1 economy burger
Beefburger, 100% meat = no CHO	—	—
Fish fingers	60	2 fish fingers
Breakfast cereals, e.g. branflakes	15	2 tablespoons
Breakfast cereals, e.g. wheat biscuit type	20	1 biscuit
Muesli, no added sugar	15	¾ tablespoon
Porridge—made with water	125	small average portion
Biscuits—plain digestive	15	1 digestive
Apple, pear	100	1 medium
Orange	120	1 small
Banana	45	½ small banana
Melon—galia, honeydew	200	1 medium slice
Pineapple, fresh	100	1 large slice
Grapes	70	15 large grapes
Orange juice—no added sugar	110	½ average glass
Apple juice—no added sugar	100	½ average glass
Cranberry juice	70	1/3 average glass
Milk—full fat, semi or skimmed	200	1 average glass

Table A7.4 (*Contd.*)

	Weight (g)	Description
Yogurt—low fat, fruit	70	½ small pot
Yogurt—low fat, plain	135	1 small pot
Ice cream—plain dairy, vanilla	50	1 small scoop
Lemonade	170	1 small glass
Lucozade®	60	1/3 average glass
Cola	90	½ average glass
Beer—best bitter	450	¾ pint glass
Lager—premium	400	¾ pint glass
Wine—medium white	330	2½ small wine glasses
Wine—red contains 0.2 g CHO/100 ml	—	—
Crisps	20	¾ small packet
Peanuts—dry roasted	100	1 large packet

Average portion sizes

Table A7.5 Examples of household measures of foods used commonly in the UK*

Food group	Household measure	Quantity	kcal/portion
Cereals and starchy foods	1 med. bowl breakfast cereal-sweet	40 g	149
	1 med. bowl breakfast cereal	40 g	132
	1 medium bowl porridge (whole)	200 g	232
	1 biscuit Weetabix	20 g	70
	1 medium slice of bread	35 g	76
	1 bread roll	50 g	134
	4 tablespoons of cooked pasta	60 g	52
	4 tablespoons of cooked white rice	60g	83
	2 egg-sized boiled potatoes	60 g	45
	1 medium plate of chips	100 g	239
	1 average jacket potato with skin	180 g	245
	1 croissant/brioche	50 g	180
	1 chapatti	55 g	180
	1 crumpet	40 g	79
	1 poppadum grilled	10 g	37
	1 naan bread	160 g	537
Fruit	1 medium apple (without core)	100 g	48
	1 medium banana (no skin)	100 g	95
	½ avocado (flesh only)	75 g	143
	1 cherry (no stone)	10 g	5
	1 medium clementine/mandarin	60 g	22
	1 apricot (without stone)	65 g	20
	1 slice melon (without skin)	180 g	34
	1 medium pear	170 g	68
	1 medium nectarine (no stone)	110 g	44
	1 medium kiwi (without skin)	60 g	29
	1 grape	5 g	3
	½ grapefruit (flesh only)	80 g	24
	1 medium plum (without stone)	55 g	20
	1 glass of fruit juice	200 ml	72

Table A7.5 (*Contd.*)

Food group	Household measure	Quantity	kcal/portion
Vegetables	1 average portion of vegetables, e.g. cauliflower, Brussels sprouts, carrots	90 g	38
	medium boiled carrot	45 g	16
	1 slice cucumber	6 g	1
	1 spring onion	20 g	5
	1 average onion	90 g	32
	1 average portion of peas	65 g	45
	1 average tomato	85 g	15
	1 tablespoon sweet corn	30 g	37
	1 broccoli spear	45 g	10
Dairy products	1 pint of whole milk	568 ml	375
	1 pint of semi-skimmed milk	568 ml	261
	1 pint of skimmed milk	568 ml	187
	1 pot of yogurt- low fat	125 g	113
	Hard cheese (small matchbox size)	30 g	122
	Cottage cheese—small pot	112 g	110
Protein sources	2–3 thin slices of beef-lean	90 g	193
	2–3 thin slices of pork-lean	90g	371
	2–3 thin slices of lamb-lean	90g	257
	1 medium burger	105 g	270
	1 rasher bacon-back	25 g	51
	1 medium chicken portion	150 g	223
	1 rump or fillet steak (5 oz)	115 g	326
	1 medium slice of ham	50 g	60
	2 hot dog sausages	70 g	192
	1 medium piece of fish	120 g	115
	2 sardines (tinned in tom. sauce)	40 g	71
	2 fish fingers	60 g	128
	1 average serv. tuna in sandwich	45 g	45
	1 hen's egg	50 g	73
	1 omlette/1 serving scrambled egg	120 g	228
	Average portion of beans/lentils	120 g	105
	I small tin of baked beans	170 g	143
	1 handful of nuts	40 g	243

(continued)

Table A7.5 (*Contd.*)

Food group	Household measure	Quantity	kcal/portion
Fats and fat-rich	1 teaspoon of butter/margarine	5 g	37
	1 tablespoon of oil	11 ml	90
	1 packet of crisps	28 g	153
Sweet foods	1 sugar cube	5 g	20
	1 sachet of sugar	10 g	40
	2 squares of chocolate	5 g	26
	1 dessertspoon of jam	30 g	78
	1 sweet biscuit	20 g	91
	1 vanilla slice	110 g	305
	1 slice of chocolate/sponge cake	65 g	298
	1 slice of apple pie	115 g	214
	1 jam doughnut	75 g	252
	1 slice fruit cake	70 g	248
	1 jam tart	24 g	91
	1 fruit scone	48 g	156
Drinks	1 small glass	150 ml	
	1 medium glass	200 ml	
	1 mug	250 ml	
	1 can of fizzy sweet drink	330 ml	129
	1 average glass of wine	125 ml	88
	1 average bottle of wine	750 ml	525
	1 measure of spirits	23 ml	51
Composite meals	1 medium pizza	300 g	750
	1 slice of quiche/flan	120 g	377
	1 average portion of stew	330 g	396
	1 average portion of curry	330 g	677
	1 average portion of lasagne	450 g	460
	1 individual steak and kidney pie	200 g	646
	1 average portion of shepherds pie	300 g	354
Spoon sizes	1 teaspoonful	~5 ml	
	1 dessertspoonful	~10 ml	
	1 tablespoonful	~15 ml	

* Sources: Crawley, H. (1990). *Food portion sizes*, MAFF publication. HMSO, London; and Krebs, J. (2002). *McCance & Widdowson's the Composition of Foods*, 6th edn. FSA, London.

Appendix 8

Useful contacts

Manufacturers' contact details

The manufacturers of nutritional products referred to in this book can be contacted via details below. As both products and manufacturers are subject to change, readers are advised to check the latest issue of the *British National Formulary* for the most current information.

Abbott Laboratories Ltd
Abbott House
Norden Road, Maidenhead
Berks SL6 4XE
Telephone 01628 773355

Alembic Products Ltd
River Lane
Saltney, Chester
Cheshire CH4 8RQ
Telephone 01244 680147

Complan Foods
Imperial House
15–19 Kingsway
London
WC2B 6UN
Telephone 0845 6003170

Fresenius Kabi Ltd
Cestrian Court
Eastgate Way
Manor Park
Runcorn
Cheshire
WA7 1NT
Telephone 01928 533533

HJ Heinz Company Ltd
South Building,
Hayes Park
Hayes, UB4 8AL
Telephone 020 85737757

KoRa Healthcare Ltd
Frans Maas House
Swords Business Park, Swords
Co Dublin, Ireland
Telephone 00 353 1890 0406

Mead Johnson Nutritionals
Uxbridge Business Park
Sanderson Road
Uxbridge UB8 1DH
Telephone 00800 88342568

Nestlé Clinical Nutrition
St George's House
Park Lane, Croydon
Surrey CR9 1NR
Telephone 020 86675130

Novartis Consumer Health
Wimblehurst Road
Horsham
West Sussex RH12 5AB
Telephone 01403 210211

Nutricia Clinical Care
White Horse Business Park
Trowbridge
Wilts BA14 0XQ
Telephone 01225 711688

SHS International Ltd
100 Wavertree Boulevard
Wavertree Technology Park
Liverpool L7 9PT
Telephone 0151 2288161

SMA Nutrition
Wyeth Pharmaceuticals
Huntercombe Lane South
Taplow, Maidenhead
Berks SL6 0PH
Telephone 01628 604377

Vitaflo Ltd
South Harrington Building
182 Sefton StreetBrunswick Business Park
Liverpool L3 4BL
Telephone 0151 7099020

Websites

International bodies

American Dietetic Association: www.eatright.org
Arbor Nutrition guide: www.arborcom.com
Centers for Disease Control: www.cdc.gov
EU: Diet, Physical Activity and Health—EU Platform for Action: www.europa.eu.int/comm/health/ph_determinants/life_style/nutrition/platform/platform_en.htm
European Childhood Obesity Group (ECOG): www.ecog-obesity.eu
European Federation of the Association of Dietitians: www.efad.org
European Nutrition for Health Alliance: www.european-nutrition.org
EuroFIR[2]: www.eurofir.net
Food & Agriculture Organization: www.fao.org
The Glycemic Index Foundation: www.glycemicindex.com
Institute of Medicine, USA: www.iom.edu
International Agency for Research on Cancer: www-dep.iarc.fr
International Baby Food Action Network (IBFAN): www.ibfan.org
International Confederation of Dietetic Associations: www.internationaldietetics.org
International Obesity Task Force: www.iotf.org
National Institute of Health (USA) Office of Dietary Supplements: www.ods.od.nih.gov
United Nations Standing Committee on Nutrition: www.unsystem.org/scn
United Nations—harmonized training package for Nutrition in Emergencies: www.oneresponse.org/nutrition
United States Department of Agriculture—MyPyramid: www.mypyramid.gov
United States Department of Health & Human Services, Center for Disease Control: www.cdc.gov
World Cancer Research Fund: www.wcrf-uk.org
World Health Organization Headquarters: www.who.int
European office: www.euro.who.int

UK bodies

Age UK: www.ageuk.org.uk
Association of breastfeeding mothers (ABM): www.abm.me.uk
Association for Nutrition: www.associationfornutrition.org
Association for the Study of Obesity: www.aso.org.uk
Bandolier (independent evidence-based healthcare): www.jr2.ox.ac.uk/bandolier
Better Hospital Food: www.betterhospitalfood.com
Blood Pressure Association: http://www.bpassoc.org.uk
British Association for Parenteral and Enteral Nutrition: www.bapen.org.uk
British Dietetic Association: www.bda.uk.org
BDA Weight Wise Campaign: www.bdaweightwise.com
British Heart Foundation: www.bhf.org.uk
British Hypertension Society: http://www.bhsoc.org
British HIV Association: www.bhiva.org
British Liver Trust: www.britishlivertrust.org.uk

British Medical Association: www.bma.org.uk
British National Formulary www.bnf.org/bnf
British Nutrition Foundation: www.nutrition.org.uk
British Society of Gastroenterologists: www.bsg.org.uk
Cancer Research UK: www.cancerresearchuk.org
Care and Quality Commission: www.cqc.org.uk
Care Standards Inspectorate for Wales: www.cssiw.org.uk
Caroline Walker Trust: www.cwt.org.uk
Chartered Institute of Environmental Health: www.cieh.org.uk
Child Growth Foundation (growth charts): www.childgrowthfoundation.org
Child Poverty Action Group: www.cpag.org.uk
Clinical national guidelines: www.sign.ac.uk
Cochrane reviews: www.york.ac.uk/inst/crd/cochlib.htm
Coeliac UK: www.coeliac.co.uk
Consensus Action of Salt Health: www.actiononsalt.org.uk
Core (formerly Digestive Disorders Foundation): www.digestivedisorders.org.uk
Cystic Fibrosis Trust: www.cftrust.org.uk
Department for Education: www.education.gov.uk
Department for Environment, Food and Rural Affairs: http://www.defra.gov.uk
Department of Health: www.dh.gov.uk
Department of Health, Social Services and Public Safety in Northern Ireland: www.dhsspsni.gov.uk
Department for International Development: www.dfid.gov.uk
Diabetes UK: www.diabetes.org.uk
Dietitians in Sports and Exercise Nutrition: www.disen.org
Dietitians Working in Obesity Management: www.domuk.org
Dose Adjustment for Normal Eating: www.dafne.uk.com
Economic and Social Data Service www.esds.ac.uk
European Dialysis & Transplant Nurses Association & European Renal Care Association: www.edtna-erca.org
The Faculty of Public Health: www.fphm.org.uk
FareShare: www.fareshare.org.uk
Food Climate Research Network: www.fcrn.org.uk
The Food Commission: www.foodcomm.org.uk
Food in Later Life: www.foodinlaterlife.org
Food in schools programme: www.teachernet.gov.uk; www.wiredforhealth.gov.uk
Food Standards Agency: www.food.gov.uk; www.eatwell.gov.uk; www.eatwell.gov.uk
Foundation for Genomics and Population Health: www.phgfoundation.org
FSA Expert Group on Vitamins and Minerals Report: Safe Upper Levels for Vitamins and Minerals: www.food.gov.uk/multimedia/pdfs/vitmin2003.pdf
General Medical Council: www.gmc-uk.org
Health and Social Care Information Centre: www.ic.nhs.uk
Health in Wales: www.wales.nhs.uk
Health Professions Council: www.hpc-uk.org
Healthy Start: www.healthystart.nhs.uk
Heart UK: www.heartuk.org.uk

Hospital Caterers Association: www.hospitalcaterers.org
Infant feeding: www.babyfriendly.org.uk/commun.asp#plan; www.breastfeeding.nhs.uk; www.nctpregnancyandbabycare.com; www.surestart.gov.uk *Feeding pre-term infants:* www.laleche.org.uk; www.bliss.org.uk
Institute of Food Research: www.ifr.ac.uk
Institute of Health Promotion and Education: www.ihpe.org.uk/home/index.htm
International Diabetes Federation: www.idf.org/home
Joint Health Claims Initiative: www.jhci.co.uk
Kidney Patient Guide: www.kidneypatientguide.org.uk
La Leche League (UK): www.laleche.org.uk
Leatherhead Food Research Association: www.lfra.co.uk
Ministry of Defence: www.mod.uk
MRC Human Nutrition Research: www.mrc-hnr.cam.ac.uk
National Advisory Group (INVOLVE) (supporting active public involvement in NHS): www.invo.org.uk
National Audit Office: www.nao.gov.uk
National Childbirth Trust (NCT): www.nct.org.uk
National Heart Forum: www.heartforum.org.uk
National Institute for Clinical Excellence: www.nice.org.uk
National Kidney Foundation: www.kidney.org
National Obesity Observatory: www.noo.org.uk
National Osteoporosis Society: www.nos.org.uk
National Statistics Office: www.statistics.gov.uk
NHS 5aday Programme: www.5aday.nhs.uk
North Atlantic Treaty Organization (NATO): www.ftp.rta.nato.int
Northern Ireland at the Regulation and Quality Improvement Authority (RQIA): www.rqia.org.uk
The Nutrition Society: www.nutritionsociety.org.uk
Office for National Statistics: www.statistics.gov.uk
Office for Standards in Education, Children's Services and Skills: www.ofsted.gov.uk
Oxford University Press: www.oup.com
Overseas Development Institute: www.odi.org.uk
Patient literature: www.publications.dh.gov.uk
Prader–Willi Syndrome Association: www.pwsa.co.uk
Prodigy: www.prodigy.nhs.uk
Public Health Agency, Northern Ireland: www.publichealth.hscni.net
The Refugee Council: www.refugeecouncil.org.uk
The Royal Society for Public Health: www.rsph.org.uk
The Royal Society of Medicine www.roysocmed.ac.uk
The Royal College of Paediatrics and Child Health: www.rcpch.ac.uk
Rowett Research Institute: www.rowett.ac.uk
The Scientific Advisory Committee on Nutrition: www.sacn.gov.uk
School Food Trust: www.schoolfoodtrust.org.uk
Scotland's Health Improvement Agency: www.healthscotland.com
The Scottish Commission for the Regulation of Care: www.carecommission.com
Scottish Intercollegiate Guidelines Network (SIGN): www.sign.ac.uk

Scottish Nutrition and Diet Resources Initiative: www.sndri.gcal.ac.uk
Slimming World®: www.slimming-world.co.uk
Society of Health Education and Health Promotion Specialists: www.hj-web.co.uk/sheps
Sport and Exercise Nutrition Register (SENr): www.senr.org.uk
The Stroke Association: http://www.stroke.org.uk
Sustain: The alliance for better food and farming: www.sustainweb.org
Sustainable Development: www.sustainable-development.gov.uk
Sustrans: www.sustrans.org.uk
UK Food Group: www.ukfg.org.uk
The UK Public Health Association: www.ukpha.org.uk
Vegetarian Society: www.vegsoc.org
Walking the Way to Health Initiative: www.whi.org.uk
Weight Watchers®: www.weightwatchers.co.uk
Weight Wise campaign: www.bdaweightwise.com/bda

The National Statistics Socio-Economic Classification (UK)

From 2001, the National Statistics Socio-economic Classification (NS-SEC) has been used for official statistics and surveys. It was rebased in 2010.[1] It replaces social class based on occupation and socio-economic groups. The information required to create the NS-SEC is occupation (coded to the Standard Occupational Classification 2000) and details of employment status (whether an employer, self-employed, or employee; whether a supervisor; number of employees at the workplace)(see Box A9.1). There are eight classes, the first of which can be subdivided.
A simpler, self-coded version of the NS-SEC has been developed with 5 classes for use in postal surveys or where detailed occupation information is not needed (Box A9.2).

Box A9.1 The National Statistics Socio-economic Classification Analytic Classes*

1 Higher managerial and professional occupations
- 1.1 Large employers and higher managerial occupations
- 1.2 Higher professional occupations

2 Lower managerial, administrative and professional occupations
3 Intermediate occupations
4 Small employers and own account workers
5 Lower supervisory and technical occupations
6 Semi-routine occupations
7 Routine occupations
8 Never worked and long-term unemployed

* For complete coverage, the 3 categories of students, occupations not stated or inadequately described, and not classifiable for other reasons are added as 'Not classified'.

Box A9.2 Simpler National Statistics Socio-economic Classification Analytic Classes

1 Higher managerial and professional occupations
2 Intermediate occupations
3 Small employers and own account workers
4 Lower supervisory and technical occupations
5 Semi-routine and routine occupations

[1] Further information: www.ons.gov.uk/about-statistics/classifications/current/soc2010/soc2010-volume-3-ns-sec--rebased-on-soc2010--user-manual/index.html.

Appendix 10

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