

Growth and Aging

ML Wahlqvist, A Kouris-Blazos, KA Ross, TL Setter and P Tienboon

Key messages

- Growth provides an indication of nutritional status in preadult years.
- Changes in body composition and anthropometry reflect changes in growth and thus nutritional status.
- Nutritional needs change in accordance with the demands of growth throughout the different stages of life.
- The interplay of genetic and environmental factors determines growth outcomes and disease risk.
- Undernutrition during the early years of life can drastically impair growth, and can affect stature and health outcomes in later life.
- Catch-up growth is a phenomenon that compensates for deviations in growth from the genetic trajectory.
- Maximal height may not be equivalent to 'optimal' height with respect to positive health outcomes.
- Obesity and overweight in early life, and especially during adolescence, increase the likelihood of obesity and associated risk factors in adult life.
- Chronological and biological age do not necessarily correlate.
- Energy requirements generally decrease with age; however, nutrient needs remain relatively high; animal studies suggest that energy restriction promotes longevity, but human studies suggest that 'eating better not less' is desirable.
- Physical activity can improve health and well-being, and reduce morbidity risk at any stage of the lifespan.
- Many health problems commonly associated with older age are not necessarily products of 'aging'; instead they can be prevented or delayed by consuming a nutritionally adequate diet and engaging in regular physical activity.

7.1 Introduction

Nutrition plays an important role in human growth and development throughout life. Infancy and childhood are important times for nutrition and growth as they strongly predict health outcomes later in life. Nutrition once again plays an important role in later life, when prevention of chronic disease and system degeneration becomes a major priority. All people require the same nutrients to maintain health and well-being, but these are required in differing amounts according to their stage of life. Optimal growth and healthy aging will occur if nutritional requirements are met and environmental influences are conducive to health throughout life.

7.2 Growth and development

'Growth' may be defined as the acquisition of tissue with a concomitant increase in body size. 'Development' refers to changes in the body's capacity to function both physically and intellectually through increased tissue and organ complexity. Different individuals experience these processes at different rates.

There are five stages under which major growth and developmental changes occur in humans:

- infancy
- childhood
- adolescence
- adulthood
- late adulthood.

These stages can be distinguished by changes in growth velocity and distinct biological and behavioral characteristics. Nutritional needs change in response to the demands that these stages of growth place on the body. If nutritional needs are met and adverse social circumstances or disease are not encountered, optimal growth will occur.

Cellular aspects of growth and death

Cell division

Cells are subject to wear and tear as well as to accidents and death. Therefore, we must create new cells at a rate as fast as that at which our cells die. As a result, cell division is central to the life of all organisms. Cell division or the M phase (M = mitotic) consists of two sequential processes: nuclear division called mitosis and cytoplasmic division called cytokinesis. Before a cell can divide it must double its mass and duplicate all of its contents to ensure that the new cell contains all the components required to begin its own cycle of cell growth followed by division. Preparation for division goes on invisibly during the growth phase of the cell cycle, denoted as interphase. Cells spend 90% of their lifetime in interphase, during which cell components are continuously being made. Interphase can last for up to 16–24 h, whereas the M phase lasts for only 1–2 h. Interphase starts with the G₁ phase (G = gap) in which the cells, whose biosynthetic activities have been slowed during the M phase, resume a high rate of biosynthesis. The S phase begins when DNA synthesis starts, and ends when the DNA content of the nucleus has doubled and the chromosomes have replicated. When DNA synthesis is complete the cell enters the G₂ phase, which ends when mitosis starts. Terminally differentiated and other non-replicating cells represent a quiescent stage, often referred to as G₀ phase (Figure 7.1).

In multicellular animals, like humans, the survival of the organism is paramount, not the survival of any of its individual cells. As a result, the 10^{13} cells of the human body divide at very different rates depending on their location and are programmed and coordinated with their neighbors. Something in the order of 10^{16} cell divisions take place in a human body in the course of a lifetime. Different cell types have given up their potential for rapid division so that their numbers can be kept at a level that is optimal for the organism as whole. Some cells, such as red blood cells, do not divide again once they are mature. Other cells, such as epithelial cells, divide continuously. The observed cell-cycle

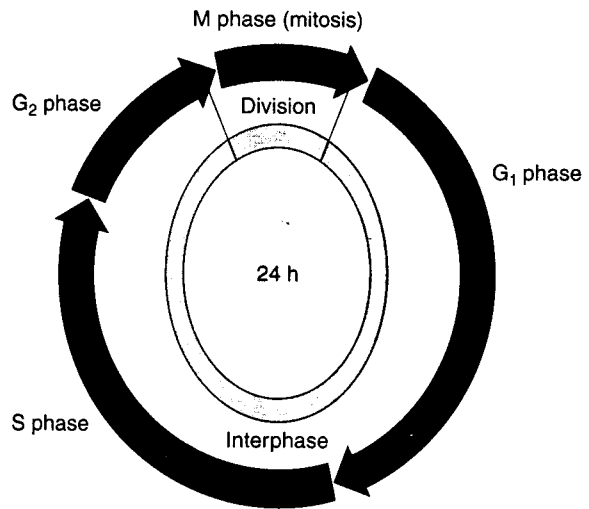


Figure 7.1 Cell growth and division: the four successive phases of a typical cell cycle.

times, also called generation times, range from 8 h to 100 days or more. Cells that are not actively proliferating have a reduced rate of protein synthesis and are arrested in G₁ phase. Once a cell has become committed to divide by passing a special restriction point (R) in its cycle in G₁, it will make DNA in the S phase and proceed through to the next stages. Cells arrested at R stop growing but do not stop biosynthesis. This growth-control mechanism involving a specific restriction point may have evolved partly because of the need for a safe resting state (at R) for cells whose growth conditions or interactions with other cells demand that they stop dividing. Cells that have been arrested at this stable resting state are said to have entered a G₀ phase of the cell cycle. Whether or not a cell will grow and divide is determined by a variety of feedback control mechanisms. These include the availability of space in which a cell can flatten (contact inhibition of cell division) and the secretion of specific stimulatory and inhibitory factors (peptides, steroids, hormones, short-range local chemical mediators and others still to be identified) by cells in the immediate environment.

Cancer cells have escaped from or respond abnormally to many of these control mechanisms that regulate cell division. Cancer cells require fewer protein growth factors than do normal cells in order to survive and divide in culture; in some cases this may be because they produce their own growth factors. A second fundamental difference between normal and cancer cells

is that the latter can go on dividing indefinitely. For example, cells taken from older animals will divide fewer times in culture than the same cells taken from young animals, suggesting that older cells have used up many of their allotted divisions while in the animal. As cells differentiate they become programmed to die after a certain number of divisions. This programmed cell death is an additional safeguard against the unbridled growth of one particular cell. However, cancer involves something more than just abnormalities of proliferation and programmed senescence; it requires the coincidental occurrence of several specific mutations in a single cell, enabling it to proliferate in disregard of the usual constraints and to invade regions of the body from which it would normally be excluded.

Cell death

Apoptosis is an active process in which cells undergo genetically programmed death. Apoptosis occurs when a calcium-dependent enzyme (endonuclease) fragments the genome of the cell into approximately 180 base pairs. The dead cells are then removed by phagocytosis. It is still unclear how many mechanisms are involved in causing cell death, but it is known that alterations in intracellular calcium levels can trigger apoptosis. Cell death appears to be activated by special genes in dying cells. For cell death to occur, genes identified as *ced-3* and *ced-4* must be expressed in a dying cell. A third gene, *ced-9*, is a major control factor, which negatively regulates the *ced-3* and *ced-4* genes. Animal studies have shown that mutations to *ced-9* inactivate the gene, causing the death of cells that were otherwise intended to survive, and thereby killing the animal under study. Cell senescence occurs when the cell does not divide or proliferate and DNA synthesis is blocked. Cells are programmed to carry out a finite number of divisions, which is called the Hayflick limit. If cells do not reach senescence they continue to divide, in effect becoming immortal. Immortal cells eventually form tumors, so this is an area where aging research and cancer research intersect. It is thought that senescence may have evolved because it protected against cancer. Cells exhibiting evidence of DNA damage and oxidative stress may recover from those stresses, provided they are equipped with an adequate DNA repair system and an adequate level of stress-response proteins, and/or antioxidant defenses. Most theories that aim to explain the aging process suggest that senescence results from the accumulation of unrepaired

damage. Telomeres (the sections of DNA at the end of chromosomes) shorten with each division of the cell, resulting in a set lifetime for each normal cell. Inhibition of telomerase is thought to reduce cellular aging. Although genes play an important role and can be an indicator of disease risk in later life, environmental factors also strongly influence the development of disease. The general degenerative properties of aging are also a factor in disease development. Greater understanding of the molecular events regulating cell progression and apoptosis is rapidly emerging. The non-nutritional and nutritional factors affecting cell division and growth are outlined below.

Non-nutritional factors affecting growth

Growth is influenced by a number of important factors including genetics and endocrine control, and also by the surrounding environment, such as nutritional factors, social factors (socioeconomic status and cultural practice) and psychological factors. For example, physical growth of school children aged 6–9 years is the result of both environmental and genetic factors and the interaction between these two factors. It is too simplistic to think that these factors affect growth in isolation, as ultimately, it is the interaction of all of these factors that will affect growth. It is often difficult to isolate the effects on growth of these individual factors as they are often closely linked.

Genetic factors

Genetic factors play arguably the most important role in determining growth and development outcomes. It is clear that the genetic make-up of the individual and what is inherited from previous generations greatly influence how they advance through life. Studies of twins have shown that patterns of growth, age of menarche, body shape, composition and size, and deposition of fat are all closely linked to genetic factors. Many studies support a genetic component in obesity-related traits. Height is influenced strongly by genetic predisposition, whereas weight appears to be mostly influenced by environment. The effects of genetic control are often quite explicit and limited in terms of their action. Dental maturation, for example, appears to be independent of skeletal maturation, and some evidence suggests that genes regulating the growth of different sections of limbs are also independent. Evolution and genetic factors largely explain racial differences, although the role of socioeconomic and educational advantage and

disadvantage, and of living conditions, including food, is likely to be underestimated. For example, skeletal maturation and growth rates differ between white American children and black African-Americans in the first few years of life. While this is largely attributed to genetic factors, increasing evidence for intragenerational and intergenerational effects of intrauterine nutrition and early childhood rearing are important qualifiers.

Gender differences are predominantly explained by genetic factors, such as differences in both the timing of the adolescent growth spurt and the sex-specific changes that occur during puberty. At the age of 10 years, males and females are of a similar height, weight and body fatness. However, after puberty major differences are evident. Although the timing of the growth spurt is largely genetically controlled, hormonal factors are thought to control its intensity and duration, while environmental factors also play an important role. Females typically achieve skeletal maturation at an earlier age than males. Genes on the Y chromosome, which are found only in males, are thought to retard skeletal maturation relative to females. A disturbance in the expression of a single gene or group of genes can have widespread and drastic effects leading to compromised growth and development. Insults to gene expression during intrauterine life, in particular, can have important consequences in later life in terms of growth and development, and even in the formation of degenerative diseases. Inborn errors of metabolism such as phenylketonuria, galactosemia and hereditary fructose intolerance are all genetic conditions that require vigilant dietary management to avoid deficiencies of essential nutrients.

Hormones and growth factors

Hormones are responsible for coordinating much of the appropriate timing and rates of growth. There are many hormones that have recognized effects on growth. There are generally three distinct endocrine phases of linear growth: infancy (including fetal growth), childhood and puberty. Each of these phases is regulated by different endocrine growth-promoting systems. Thyroid and parathyroid hormones, through thyroxine and triiodothyronine, stimulate general metabolism. Other hormones and growth factors include epidermal growth factor (important in actions in the epidermis), platelet-derived growth factor (involved in blood clots and possibly cell division) and melatonin (has a role in regulating puberty and possibly growth

velocity). Hormones are largely responsible for many of the changes in body composition and the development of secondary sexual characteristics that occur as individuals age. The effects of hormones are often influenced and regulated by other hormones and growth factors.

One of the most important hormones that regulates growth is human growth hormone (GH). GH exerts a powerful effect on growth, at least in part, through somatomedins or insulin-like growth factors (IGF-I, IGF-2). IGF acts by stimulating muscle cell differentiation and is therefore important in fetal and postnatal growth and development. GH is important in regulating protein synthesis and cellular division, and is thus thought to be the major regulator of the rate of human growth and development from the latter few months of the first year of life. Synthetic forms of GH are regularly used in a clinical setting to encourage growth in children of a small stature. It is thought that sex steroids, which are active around puberty, may trigger significant secretions of GH, and thus IGF. Hormones such as thyroxine and cortisol may also influence IGF plasma levels. Disturbances to genetic programming *in utero* during critical phases of fetal development may result in impairment of the endocrine factors responsible for growth in later life. Studies have reported associations between growth retardation and abnormalities in levels of hormones such as GH and hormone-mediated factors such as IGF.

Neural control

It has been proposed that within the brain there is a central 'growth centre' that is responsible for regulating growth. The hypothalamus has been implicated as a possible growth centre owing to its close association with the anterior pituitary, a hormonal gland closely involved in growth. By this action, any deviation of growth from its predetermined genetic pathway will be identified by the hypothalamus, and compensated for the subsequent release of growth specific hormones from the anterior pituitary gland to compensate for any growth or developmental impairment. The peripheral nervous system may also play an important role in growth. This effect is thought to be through the secretion of neural chemicals that can then influence growth.

Local biochemical control

Areas of localized growth rely on complex regional influences, whether they are mechanical or chemical

stimuli, for optimal growth. These can be paracrine factors, such as neighboring proteins acting as local signaling and growth factors, or autocrine factors, such as hormones or growth factors, that influence the growth of quite localized areas despite being secreted from a more distant position. These mechanisms appear to be responsible for the growth of specific tissue. Furthermore, the age of groups of cells or a specific tissue can determine the amount of cell replication and division that is possible. This is the case where cells undergo a finite number of mitotic divisions.

Social and cultural factors

The major social factors affecting growth are culture, age, family, gender and socioeconomic status. Emotional disorders have also been related to growth abnormalities. Different countries or regions may exhibit their own cultures and cuisines, which reflect a mixture of geographical, agricultural, historical, religious and economic factors, among others. These factors may either directly or indirectly affect growth through their influence on a number of factors such as health, nutrition, food selection and cooking methods. Infants and children who emigrate to other areas may often experience changes to growth patterns. Generational differences in stature, for example, often reflect changing environments. Many adolescents eagerly seek independence, and factors associated with this have an effect on nutritional status. Physical activity levels, peer pressure, self-esteem and distorted body image, chronic dieting and disordered eating, and substance abuse are all factors that can affect nutritional status and thus growth. Chronic dieting, in particular, can also place an individual at risk of many micronutrient deficiencies, compromised immune function, infertility, long-term degenerative diseases such as osteoporosis and a compromised immune system, which can negatively affect growth and health outcomes.

Family factors that have been associated with compromised growth and development include single-parent families, family conflict, and disturbance to the family unit such as divorce and separation. Growth retardation in conflict situations may partly be a result of stress, which is thought to affect GH levels. The amount of care and attention that an infant receives will also have an important impact on growth. This can be related to the number children and birth position in the family. Various cultures may practice a gender bias, and favor one gender over the other. This can be

seen to a larger degree in developing countries where boys may be more favored and thus receive more care and nourishment. In both developing and industrialized countries, socioeconomic status is associated with many behavioral, nutritional and health outcomes which can influence growth. Usually children of a higher socioeconomic class are taller, display faster growth rates and become taller adults. Children of lower classes are typically smaller at birth, shorter and, particularly in industrialized countries, have higher levels of body fat. Socioeconomic status is also a strong predictor of certain micronutrient deficiencies, such as vitamin A and iron deficiencies.

Independently, educational attainment and income have been positively associated with growth and development, mainly through the alleviation of poverty. Females, in particular, benefit from schooling, as they are able to exert greater control over their environment and show improved growth and pregnancy outcomes. Typically, as income increases a larger amount of money will be spent on animal products such as meat owing to the prestige factor associated with such foods. The inclusion of greater amounts of animal protein in the diet can influence growth and development. Infant feeding practices, such as breast-feeding and weaning, may also follow culturally or socially acceptable patterns of a region. Women of higher socioeconomic status are known to breast-feed for longer periods than women of lower classes. This can have important growth and development outcomes on the infant. Dental caries are common in both developing and industrialized countries where dental hygiene is inadequate and in areas where water fluoridation is absent. Dietary factors associated with increased dental caries under such conditions include the regular consumption of foods high in sucrose, particularly sticky sweet forms.

7.3 Nutritional factors affecting growth

In order for growth to proceed at its predetermined genetic rate, adequate nutrition is essential. Food supplies the individual with the required energy, nutrients and food components to influence growth. The strong link between food intake and growth was supported from studies of food intake patterns during famines encountered at the time of the two world wars. Children of Dutch and German families living in these areas exhibited impaired growth, while in Japan, a

reduction in the mean height of adults was observed between 1945 and 1949. Nutrition affects all body systems and factors influencing growth. For example, a compromised nutrient intake can influence gene replication and expression, hormonal control, neural control and other environmental factors that are important for growth.

Inadequate nutrition is the predominant factor leading to malnutrition, which can be expressed as either undernutrition or overnutrition. Undernutrition occurs when there is not only inadequate energy but also a lack or imbalance of specific food components and nutrients. Chronic energy deficiency, commonly referred to as protein–energy malnutrition (PEM), occurs in both developing and industrialized countries; however, it is more prevalent in the former. Characteristic features of PEM include stunted growth, delayed maturation, reduced muscle mass and decreased physical working capacity.

In addition to sufficient energy, adequate supplies of macronutrients and micronutrients are required to promote optimum growth. The proportions and amounts of these nutrients may change according to the various stages of growth. For example, protein is a prerequisite for optimal growth at all life stages, while fat may arguably have its most important role during infancy and childhood as a major supplier of energy and long-chain polyunsaturated (n-3) fatty acids, which are important in neural development. Components of certain foods, called growth factors, may have powerful effects on growth. For example, some of the proteins present in milk are thought to promote growth. Population groups that consume large amounts of milk also typically exhibit taller statures.

Nutritional status is found to affect greatly hormonal status. GH, for example, will not stimulate linear growth unless there is adequate nutrition. IGF plasma levels appear to respond closely to acute directional changes in the body's nitrogen balance. This suggests that the ingestion of several dietary components, such as essential amino acids, adequate energy and optimal nitrogen balance, may be critical for optimal hormonal control. When this is not the case, it may have negative effects on growth and development. Food and its components, such as metabolites of vitamins A and D, fatty acids, some sterols and zinc, can directly influence gene expression and thus growth. Components of dietary fiber are thought to influence gene expression indirectly by a number of pathways,

Table 7.1 Micronutrients affecting growth

Children	Adolescents
Iodide	Calcium
Zinc	Folate
Iron	
Vitamin A	
Riboflavin	
Vitamin C	
Vitamin D	

including altering hormonal signaling, mechanical stimulation and through metabolites produced by flora of the intestine. The micronutrients that are frequently held responsible for much of the functional impairment and growth retardation experienced globally are shown in Table 7.1.

Iodide

Iodide deficiency at different life stages produces differing health outcomes. Its intake is of most critical importance *in utero* and during the first 2 years of life, when neural cells in the brain undergo major cellular division. Adequate iodide intakes during these times are therefore essential for mental and cognitive growth and development. The extent of mental dysfunction may be lessened if sufficient dietary iodide levels are administered in the early years of life. Complications associated with iodide deficiency in childhood and adolescence may appear as goiter, hypothyroidism, mental dysfunction, retarded mental and physical growth and reduced school performance.

Vitamin A

Severe deficiency in vitamin A is commonly associated with impaired vision, retarded growth and development, poor bone health, compromised immune functioning, and complications with reproductive health and outcomes. In industrialized countries, vitamin A deficiency is rare. Too much vitamin A in the diet can also slow growth. Subclinical vitamin A deficiency greatly increases the risk of morbidity and mortality in vulnerable population groups. Reductions in mortality rates of around 20–25% can be achieved by improving the vitamin A status in young children in populations where deficiency has been identified.

Zinc

Zinc is of crucial importance in over 200 enzyme reactions. It is of structural and functional importance in

biomembranes, DNA, RNA and ribosomal structures. Zinc deficiency has been linked with disturbed gene expression, protein synthesis, immunity, skeletal growth and maturation, gonad development, pregnancy outcomes, behavior, skin integrity, eyesight, appetite and taste perception. Zinc deficiency can cause major intrauterine growth retardation (IUGR) if the maternal diet provides inadequate sources of zinc. It is therefore of great importance for linear growth as well as the development of lean body mass.

Iron

In industrialized countries, iron represents the major micronutrient deficiency; however, in developing countries iron deficiency occurs on a much larger scale. Although iron is important at all life stages, iron deficiency commonly affects preschool and school-aged children who as a consequence face compromised growth if dietary intake is inadequate. Iron is very important in pregnant women, as low intakes can have wide implications for the newborn infant born with limited iron stores. The effects of iron deficiency are varied; however, a major effect is its impairment of cognitive development in children. Other consequences of iron deficiency include a reduced work capacity and a decreased resistance to fatigue.

Other nutrients

Other nutrients of importance to growth include vitamin B₂, which affects general growth, vitamin C, which is important in bone structure, and vitamin D, which is involved in calcium absorption from the intestines. Chronically low dietary intakes of these vitamins can greatly impair growth and bone health. Calcium and folate appear to be micronutrients of importance for growth during adolescence.

Phytochemicals

There is emerging evidence that certain phytochemicals, such as the isoflavones genistein and daidzein (found in legumes, especially soy), may help to inhibit tumor formation by regulating cell-cycle progression, by promoting cell differentiation and apoptosis (cell death). Formation of new vasculature is required for a cancer to grow and metastasize; isoflavones have also been identified as antiangiogenic agents that inhibit the formation of new vasculature and thus the development and dissemination of tumors.

7.4 Nutrition and the life cycle

Energy and nutrient needs differ according to the different life stages and it is important for food intake to reflect these changing demands. Inadequate nutrition exerts its most detrimental impact on prepubertal growth. Supplementation programs and interventions that are provided before this time will have the most beneficial growth and development outcomes. Nutrient needs during infancy are influenced by length of gestation, the newborn's nutrient reserves, body composition, growth rate, activity levels, and the length and duration of breast-feeding. An infant is wholly dependent on a carer for some or all nourishment, ideally via breast milk.

The essential long-chain fatty acids (LCPUFA), such as the n-3 fatty acids docosahexaenoic acid (DHA) and arachidonic acid (AA), are important structurally in cell membranes, particularly in the central nervous system. Most infant formulae contain only the precursor essential fatty acids, α -linolenic acid (ALA, the n-3 precursor) and linoleic acid (LA, the n-6 precursor), from which infants must assemble their own DHA and AA, respectively. Studies have suggested that such formulae may not be effective in meeting the full essential fatty acid requirements that are needed by most infants. Reduced cognitive, motor and visual acuity outcomes have been reported in formula-fed infants compared with their breast-fed counterparts; however, not all studies have reported such findings. From 6 to 24 months of infant life, breast-feeding alone cannot provide all of the nutrients and energy needed to promote and sustain adequate growth. Therefore, complementary feeding is necessary. If complementary feeding is introduced too late there is a risk of impaired growth, macronutrient deficiency, impaired cognitive and physical development, and stunting as a result of PEM.

Early weaning (4 months or earlier) has been associated with negative health outcomes, such as the formation of allergies, diarrhea and even death. Chronic or episodic diarrhea may affect the absorption of nutrients which, if not addressed, may lead to growth impairment. Common reasons for introducing complementary feeding with formula milk or solids include a perceived inferior quality of milk, poor weight gain, difficulties or pain with feeding, mother's employment, refusal by the infant to feed and lack of mother's confidence. Many people perceive formula to be of higher quality than breast milk, particularly as a result of

aggressive marketing strategies from infant formula manufacturers. This may cause the mother to abandon exclusive breast-feeding from an early age. In 2001 the World Health Organization (WHO) released a systematic review on the optimal duration of exclusive breast-feeding. These results indicate that breast-feeding should be exclusive for the first 6 months of life (<http://www.who.int/inf-pr-2001/en/note2001-07.html>).

Some individuals experience erratic growth during childhood, largely reflecting changes in appetite and food intake, or even an underlying illness. Nutrient needs increase throughout childhood, reflecting the continuing growth of all body systems. Children can exhibit good growth and thrive on most lacto-ovo vegetarian and vegan diets provided they are well planned and supplemented. Growth delays have occasionally been reported in children fed severely restricted diets (primarily macrobiotic, Rastafarian and fruitarian forms). However, by school age, the growth of vegetarians and non-vegetarians becomes more alike. Few differences have been found in the timing of puberty or completed adult growth. Little effect is evident on intelligence quotient (IQ), assuming a reasonably adequate vegetarian diet. Typically, girls tend to consume less than boys at all ages. However, it is during adolescence that males begin to increase their intake to levels well above that of most females. Adolescence is a time that requires the greatest total energy intake of all of the life stages, as a result of the body being in a highly metabolically active state. Inadequate intakes of nutrients and energy during this time can potentially impede growth and delay sexual maturation. Pregnancy during adolescence has many increased risks for both the mother and the child, as the fetus and the mother must compete for nutrients to maintain and promote their respective growth. This is of even greater concern if the adolescent is malnourished. Calcium is of particular concern, as it is needed for continuing bone development in the mother while also being required in large quantities by the developing fetus. Birth and maternal complications during adolescent pregnancy are greater than those for older women of similar nutritional status. For example, adolescent mothers face increased risks of infant and maternal mortality, preterm delivery and giving birth to babies prematurely and of a low birth weight. The Australian dietary guidelines for children and adolescents and the American dietary guidelines for healthy people over 2 years old are shown in Tables 7.2 and 7.3.

Table 7.2 Australian dietary guidelines for children and adolescents, 1995

1. Encourage and support breastfeeding
2. Children need appropriate food and physical activity to grow and develop normally. Growth should be checked regularly
3. Enjoy a wide variety of nutritious foods
4. Eat plenty of breads, cereals, vegetables (including legumes) and fruits
5. Low-fat diets are not suitable for young children. For older children, a diet low in fat and in particular, low in saturated fat, is appropriate
6. Encourage water as a drink. Alcohol is not recommended for children
7. Eat only a moderate amount of sugars and foods containing added sugars
8. Choose low-salt foods

Guidelines on specific nutrients

1. Eat foods containing calcium
2. Eat foods containing iron

Source: National Health and Medical Research Council (1995).
© Commonwealth of Australia. Reproduced with permission.

Table 7.3 American dietary guidelines for healthy people over 2 years old, 2000

Aim for fitness

Aim for a healthy weight

Be physically active each day

Build a healthy base

Let the pyramid guide your food choices

Choose a variety of grains daily, especially whole grains

Choose a variety of fruits and vegetables daily

Keep food safe to eat

Choose sensibly

Choose a diet that is low in saturated fat and cholesterol and moderate in total fat

Choose beverages and foods to moderate your intake of sugars

Choose and prepare foods with less salt

If you drink alcoholic beverages, do so in moderation

Source: USDA (2000).

The guidelines are listed in descending order of priority.

7.5 Effects of undernutrition

Identifying the cause of undernourishment is not always an easy task. Inadequate dietary intake may not be the sole cause. Social, cultural, genetic, hormonal, economic and political factors may also be important. Underlying health problems and inadequate care and hygiene may also be contributing factors to undernourishment.

The outcomes of undernutrition are largely determined by its severity and duration. Consequences of undernutrition include death, disability, and stunted mental and physical growth. Poor nutrition often commences *in utero* and in many cases extends into adolescence and adult life. Females in particular are affected by lifelong poor nutrition. Evidence from epidemiological studies from both developing and industrialized countries now suggests a causal relationship between fetal undernutrition and increased risks of impaired growth and various adult chronic diseases. This is the basis of the fetal origins of disease hypothesis. Wasting is often one of the earliest signs of acute undernutrition. Wasting can be detected by reduced measures of weight-for-age and skinfold thickness, reflecting a loss of weight or a failure to gain weight. In severe cases of undernourishment, individuals may exhibit other clinical symptoms such as hair loss, skin discoloration or pigmentation (in marasmus) and edema (in kwashiorkor), and evidence of deficiencies that are characteristic of specific nutrients. Stunting reflects chronic undernutrition and is detected as impaired linear growth. However, where stunting and wasting are both present, as in chronic cases of undernourishment, growth charts may not detect abnormal weights (or heights)-for-length owing to proportional growth retardation of both weight and height (or length). A stunted infant is likely to remain stunted throughout childhood and adolescence and is likely to become a stunted adult, particularly if the individual continues to live in the same environment that instigated the stunting. Adult stunting and underweight have direct effects not only on a woman's health and productivity, but also by increasing the risks of pregnancy complications such as gestational diabetes and the likelihood that her offspring will be born of a low birth weight (LBW); thus, stunting commonly spans generations.

Most growth impairment, of which underweight and stunting are outcomes, occurs within a relatively short period, from before birth until about 2 years of age. Severe undernutrition during infancy can be particularly damaging to the growth of the brain. This can result in major retardation of cognitive growth and functioning. Delayed intellectual development is a risk factor for absenteeism from school and poor school performance. Infants born of LBW who have suffered IUGR are born undernourished and face a greatly increased risk of mortality in the neonatal period or later infancy. Suboptimal intakes of energy, protein,

vitamin A, zinc and iron during the early years of life may exacerbate the effects of fetal growth retardation. There is a cumulative negative impact on the growth and development of an LBW infant if undernutrition continues during childhood, adolescence and pregnancy. The LBW infant is thus more likely to be underweight or stunted in early life. Undernourished girls tend to have a delayed menarche and grow at lower velocities but for longer periods compared with their better nourished counterparts. This means that they may attain similar heights to better nourished girls, if undernutrition is limited to adolescence. However, if childhood stunting was also experienced, undernourished adolescent females are unlikely to reach similar heights to well-nourished girls. Optimal development during adolescence is reliant on both the present and past nutritional intake. Malnourishment and impaired growth during infancy and early childhood can greatly affect an individual's attainment of height.

Catch-up growth

Catch-up or catch-down growth is a phenomenon that appears to compensate for retarded or accelerated intrauterine growth, whereby children return to their genetic trajectory. Many studies have suggested that the potential for catch-up growth and reversal of cognitive impairment among children who have suffered growth retardation during infancy and/or early childhood is thought to be limited after the age of 2 years, particularly when children remain in poor environments. However, other studies have shown that undernourished children in poor environments can display spontaneous catch-up even without environmental change. As adolescence is a time of rapid growth, this provides an opportunity for further catch-up growth. It is thought, however, that the potential for significant catch-up during this time is limited. Reversed stunting in women may reduce the risk of pregnancy outcomes that are commonly associated with women of small stature. However, in most cases, regardless of whether growth catch-up has occurred, problems associated with reduced cognitive function remain. Females appear to display greater catch-up than males. Zinc deficiency, of which growth retardation is an outcome, is a commonly reported reason why males may be less able to catch up. At all ages, zinc requirements are very much higher for males than females and therefore a zinc limitation may explain restricted growth in males to a much greater extent than females, and explain why

males are unable to catch up to the same extent as females. Catch-up growth has been associated with a number of adverse outcomes in later life. It is not known why catch-up growth is detrimental; however, one theory involves IUGR restricting cell numbers; therefore, ensuing catch-up growth is achieved by the overgrowth of a restricted cell mass.

Short stature and plant food environments

Many populations in developing countries exist on diets that are predominantly plant based. Such diets are commonly associated with micronutrient deficiencies, chronic energy deficiency and poor growth outcomes. Thus, populations who consume a predominantly plant-based diet are often seen to exhibit short stature. In addition to containing fewer kilojoules, plant-based diets are thought to contain large amounts of protective phytochemicals that may act to limit the amount of 'metabolic dysregulation' that can lead to the development of degenerative diseases, and the antinutrients may also act to limit growth. It has been suggested that short stature and micronutrient deficiencies may represent adaptations for group survival in adverse environmental conditions. For example, slow growth rates associated with zinc deficiency may represent an adaptation to situations where this is a survival advantage.

Maximal versus optimal height

As undernutrition and stunting during infancy and early childhood have been consistently found to affect detrimentally both the short- and long-term health of an individual, much of the public health focus has been on encouraging secular growth or the trend towards more rapid growth and larger attained size. Secular growth is apparent in many cultures. There is evidence, however, that secular growth in many developed countries, such as North America, Western Europe and Australia, is slowing and reaching a plateau. Current nutrition theory holds that an individual should achieve their maximal height potential in order to achieve the best health outcomes. Recent studies, however, have questioned this theory and have introduced the concept of 'optimal' rather than 'maximal' stature. It has been suggested that smaller stature confers many health benefits and represents an adaptive response to an individual's environment. Although small stature carries with it an increased risk of abdominal obesity and heart disease, tall stature increases the risk of developing cancer and degenerative diseases.

7.6 Effects of overnutrition

Obesity is of global epidemic proportions, affecting children, adolescents and adults in growing numbers. In some countries over half of the adult population is affected, thus leading to increasing death rates from heart disease, hypertension, stroke and diabetes. It is the growing prevalence of obesity in younger age groups that has raised alarm. Obesity is multifactorial and is a problem of both nutrient imbalance and insufficient physical activity levels. Declining physical activity levels have been associated with television viewing and other modern technological advances. Children who watch large amounts of television are particularly at risk of becoming overweight or obese.

Effects of overweight and obesity

Being overweight or obese as a child and adolescent has many associated health, social and psychological implications. Overweight and obese children may suffer from impaired social interaction and self-esteem. They are often taller than their non-overweight peers and are often viewed as more mature. This is an inappropriate expectation that may result in adverse effects on their socialization. Obese individuals at all ages often do less well academically, leading to higher rates of poverty, and have poorer job prospects in later life. Being overweight or obese can negatively affect mobility and physical fitness. Serious physical complications associated with high weights in children are rare, but include cardiomyopathy, pancreatitis, orthopedic disorders, and respiratory disorders such as upper airway obstruction and chest wall restriction. These are largely restricted to the severely obese and are of low prevalence. In adolescents, obesity confers significant cardiovascular risks, abnormal glucose tolerance, hypertension and lipid profile abnormalities. Furthermore, a greater percentage of abdominal fat in children of both genders is associated with early maturity. Few studies have investigated the long-term effects of childhood obesity on adult health outcomes, but obesity experienced during childhood or adolescence seems to increase the risk of adult morbidity and mortality.

Age of onset and obesity in later life

Differences exist in the prevalence of obesity between boys and girls, between men and women, and between social classes. Social factors were a predominant

determinant of body fatness and thus obesity. Obesity is, however, also a familial condition. It has been reported that less than 10% of obese children have both parents of a normal weight, with 50 and 80% of obese children having one and two obese parents, respectively. Obese children are more likely to remain obese as adolescents and as adults. The age of the onset of obesity strongly influences this risk. The older the obese child, the more probable it is that he or she will become an obese adult. The correlation between adult and childhood obesity rises with age and with the severity of childhood obesity, while the rate of spontaneous weight reduction decreases with age. The proportion of overweight or obese in adults who had been overweight or obese in adolescence ranges from 20 to 45%, while from 25 to 50% of overweight or obese adolescents had been overweight or obese in early childhood. Obese adolescents have significantly more abdominal fat than the non-obese. Obesity that begins during or close to adolescence is often characterized by the abdominal type of fat distribution. In the 1960s it was reported that both hypermasculine and hyperfeminine types of body fat distribution in women often began prior to adulthood, with the hyperfeminine variety tending to originate prepubertally and the hypermasculine tending to originate during adolescence. Adolescence may thus be a sensitive period for the development of android or abdominal obesity in both males and females.

Predictors of body size and fatness in adolescence

The interaction of both genetic and environmental factors appears to determine human body fatness. Measures that predict body size and fatness in adolescence have been identified. Predictors in early life of body size and fatness in adolescence include birth weight, anthropometric measures at 12, 50 and 80 months, weight and height velocities during the first year, the presence of a major illness, and parental socioeconomic status in early life. Tienboon *et al.* (1992) found that the consumption of fish oil [high in eicosapentaenoic acid (EPA) and DHA] in early life lowered the risk of developing obesity, particularly abdominal obesity. Self-reported measures of exercise, appetite and consumption of some specific food items correlated more closely with contemporary predictors of adolescent body size and fatness when measured at adolescence.

Factors affecting weight status in children and adolescence

Many factors, including season, geographical region, population density, ethnicity, socioeconomic status, family size, gender, parental education, physical activity levels, maternal age and maternal preference for a chubby baby, have been reported to affect the development of obesity in children and adolescents. Weight status may differ between children and adolescents living in rural and urban areas. Adolescents from urban areas may be significantly taller and heavier, and have more superficial fat and longer legs than adolescents from rural areas.

Rate of weight gain and mode of feeding in early life

Early feeding experience is related to development of excess weight in infancy. Both breast-feeding and the delayed introduction of solid foods appear to exert a protective effect against adiposity up to 2 years of age and probably in later life. However, not all studies have shown this effect. The ages where most obesity arises before adulthood are between 0 and 4 years, 7 and 11 years and during adolescence. During infancy, rapid increases in weight have been associated with obesity at adolescence in boys. Early catch-up growth, between the ages of 0 and 2 years, has been frequently reported as a predictor of childhood obesity, particularly for central or abdominal obesity.

Familial aggregation of weight status

In the 1920s one of the first and more detailed of the early genetic studies was conducted and it was found that alleles for obesity tended to be dominant, and thus more likely to be expressed compared with non-obese alleles. Slender individuals tended to be homozygous, whereas obese persons were often heterozygous for these alleles. Overweight or obese individuals generally have at least one overweight or obese parent. Between 49 and 69% of overweight or obese children have at least one obese parent, with a fat mother (26–43%) being more common than a fat father (12–16%). When all other family members are obese, the likelihood of children being obese is very high (24–28%).

Many studies have investigated the relationship between body fatness and fat distribution among families. The effects of environmental factors with respect to weight status have been demonstrated in studies of

twins and siblings. Both genetic and environmental factors appear to play a role in total body fat levels and distribution patterns. Measures of body mass index (BMI) for immediate family members are often highly correlated. In addition, it appears that people living together can have similar degrees of fatness. Under shared environmental conditions, both genetically related and unrelated household members show similar amounts of total body fatness. In this sense genetically unrelated subjects are about as similar, fat-wise, as genetically related individuals. However, studies have found that genetic rather than environmental factors may be more influential in determining the fat distribution pattern. Spouses who do not share genetic make-up often show little relationship in fat distribution, even after 20 years of cohabitation. However, family members of shared genetic origin show similarities in fat distribution patterns, and the degree of correlation is gender specific.

7.7 Growth during childhood and adolescence

The most rapid periods of growth take place during the first few months of life and during adolescence. Growth velocity slows significantly after the first year of life. The growth rate accelerates again as an individual enters puberty in adolescence over a period of 1–3 years. After the peak growth velocity of puberty has been attained, the growth rate slows considerably

until growth in height ceases at around 16 years of age in girls and 18 years in boys (Figure 7.2).

During the first year of life, a well-nourished infant will ideally increase in length by 50% and in weight by 300%. Rapid and essentially linear growth occurs in well-nourished infants, with rates of weight and height keeping pace with each other. Infants have a high surface area to body weight, which means that their energy and nutritional needs are much higher than adults on a per kilogram of body weight basis. Infants are prone to heat stress and dehydration because of this; however, the ratio of surface area to body weight decreases with age. The period of infancy ends when the child is weaned from the breast (or bottle), which in pre-industrialized societies occurs at a median age of 36 months, and less in more developed countries. During early childhood, height and weight increase in an essentially linear fashion. Steady growth necessitates a gradual increase in the intakes of most nutrients to support growth and development. It is thought that the age of 6–7 years is a critical period for determining future weight and height status. During childhood, an individual is still largely dependent on the caregiver for providing nourishment, but this begins to change during late childhood when a child begins to develop increased control over his or her food intake and relies less on caregivers.

During adolescence the body undergoes a large number of changes as a result of puberty. Once puberty is reached, an individual is capable of sexual reproduction.

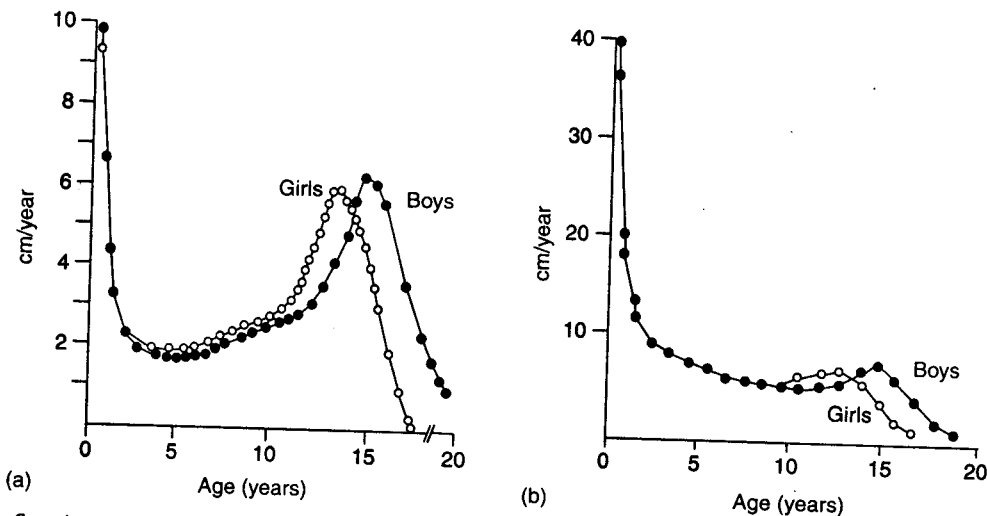


Figure 7.2 Growth velocities through childhood and adolescence: (a) 50th centile weight velocities; (b) 50th centile height velocities. (Reproduced with permission from Gracey *et al.*, 1989.)

The onset of puberty is characteristically earlier in females than in males (10.5–11 years and 12.5–13 years, respectively). As males reach puberty later than females, they experience on average 2 more years of prepubertal growth than females, resulting in typically higher statures going into puberty than females.

Before puberty, there are no significant height differences between girls and boys. It is during the adolescent growth spurt that major skeletal differences between males and females become apparent. In males, there is a widening of the shoulders with respect to the pelvis, while the opposite is seen in females. Typically, height gains of approximately 20 and 15 cm are realized in males and females, respectively.

In both genders, a sequential pattern of growth is observed: the feet and hands, the calves and forearms, the hips and chest, and the shoulders, followed by the trunk. Skeletal growth ceases once the epiphyses, the active area of bone at the end of long bones, have closed. Once this occurs bones are unable to achieve further length and are largely unresponsive to exogenous GH administration. In males, puberty is marked by growth of the sexual organs, followed by changes in the larynx, skin and hair distribution. The growth of the ovaries signifies the beginning of puberty in females. Fat is deposited in the breasts and around the hips, dramatically changing the female body shape. Menarche coincides with the peak adolescent growth spurt, after which growth decelerates.

Body composition changes during childhood and adolescence

Growth and development in children and adolescents are associated with changes in body composition that affect body fatness and leanness. Body composition is used as one of the measures of growth. It also provides an indication of both nutritional status and physical fitness.

Changes in fat-free mass and body water content

Lean body mass (LBM) includes all non-lipid body constituents as well as essential fats and phospholipids. In adolescence, LBM increases to a much greater extent in males than in females, with muscle and bone representing the largest gains in growth. Fat-free mass (FFM) is similar to LBM, but it excludes all essential fats and phospholipids. FFM density (effectively lean mass) increases from the first year of life through to 10 years of age, and then again dramatically during

puberty. Both males and females show a linear increase in FFM mineral content from the age of 8–15 years. The body's water content, measured as FFM–water content, decreases from birth to adulthood, from 81 to 72%. This drop in water content coincides with the abrupt rise in the mean FFM density seen between these ages.

Changes in body fat

Full-term infants have 10–15% body fat. In infancy, the amount of fat is correlated with body weight. Body fat shows a remarkable increase during the first year of life. By the end of this year it is estimated that body fat represents 20–25% of total body weight. Thereafter, there is a decline in the percentage of body fat, to its lowest level in the mid-childhood years, and then an increase in adolescence. Girls experience a much larger increase in body fat than boys during adolescence. Adolescent boys typically have significantly less superficial fat, less total body fat and less percentage body fat than adolescent girls. Further, adolescent boys have a significantly higher abdominal or central fat distribution than girls, as shown by waist–hip ratio and subscapular–triceps skinfold thicknesses ratio.

Fat patterning in children and adolescents

The waist-to-hip circumference ratio (WHR) measures the predominance of fat storage in the abdominal region relative to the gluteal region. A high WHR is indicative of excess abdominal fat, that is, a central fat distribution. In adults, the WHR has been related to a number of metabolic diseases and is a strong predictor of mortality; however, the implications of a high WHR in children are not clear. WHR is significantly influenced by age and gender. Boys generally have higher measures of WHR than girls. The WHR decreases with age from about 1.1 in the youngest children to about 0.8 in pubertal children. From puberty onwards, the WHR approaches the values reported for adults. In children, WHR is more or less independent of the total body fatness, whereas in adults the WHR is positively correlated with body fatness, as measured by BMI. In general, subcutaneous adipose tissue is distributed peripherally for most children up until puberty. For most boys but only a few girls, fat begins to be stored more centrally. The rate of change towards a more central fat distribution decreases in girls after about 13–14 years of age, but continues in boys. In adults, a central fat pattern is common in men, but not in women.

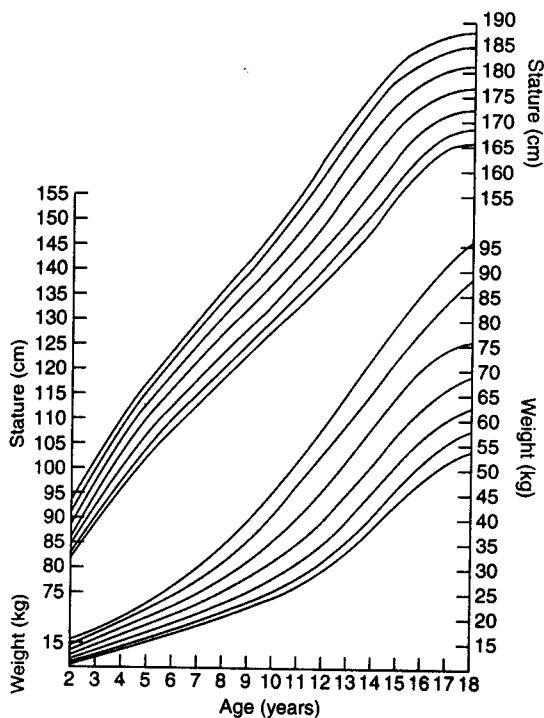


Figure 7.3 Growth chart (attained): boys 2–18 years. (Source: Lee and Nieman, 1996.)

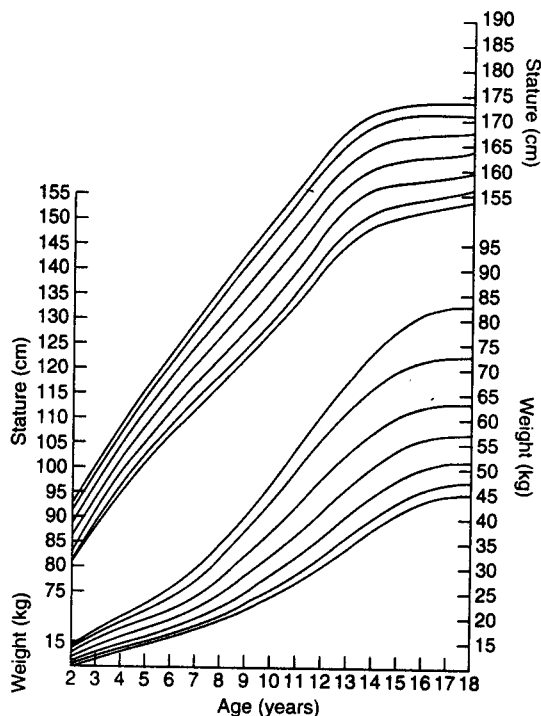


Figure 7.4 Growth chart (attained): girls 2–18 years. (Source: Lee and Nieman, 1996.)

However, a central fat pattern is more prevalent in elderly women than in younger adult women.

Assessment of growth

Growth charts are routinely used to assess growth and are used in diagnosis and management of diseases and in monitoring the efficacy of therapy. Chronic undernutrition or overnutrition is typically reflected in growth rates and therefore the monitoring of growth is used as an integral part of nutritional assessment. Common growth charts include weight-for-age, length-for-age (for children below 2 years of age) and height-for-age (for children 2 years and over) (Figures 7.3 and 7.4).

Although growth charts that show attained growth may show a steady progressive increase in anthropometric body measures with age, growth velocity charts reveal the changing rates of growth more visibly (see Figure 7.2). The growth charts may suggest that an infant or child who is above the 97th centile or below the third centile is unusually large or small; however, such a conclusion is not always appropriate. The growth of a well-nourished individual will typically follow a

growth curve and therefore an individual who is considered small or large from a growth chart may simply reflect that the infant or child's growth is a result of individual variation. A greater cause for alarm is when an individual crosses centiles, particularly if this happens over a short period. This may indicate inappropriate weight gain or acute growth failure or retardation, such as wasting or stunting, as a result of chronic undernutrition due to either chronic malnourishment or an underlying disease condition. Wasting is detectable on weight-for-age growth charts and by reduced skinfold measures, while stunting is seen as impairment of linear growth as detected by length- or height-for-age growth charts. It should be noted, however, that there are large variations between individuals in rates and patterns of growth, and these variations must be taken into consideration when determining whether a child's growth is abnormal.

Appropriate measures must be used for specific population groups. Growth charts based on Caucasian bottle-fed babies are unlikely to be appropriate for use in certain developing countries, as using such charts may misclassify an individual or population group as

Table 7.4 International cut-off points for body mass index (BMI) for overweight and obesity by gender between the ages of 2 and 18 years

Age (years)	Overweight		Obese	
	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	19.23
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	20.08
7	17.92	17.75	20.63	20.51
7.5	18.16	18.03	21.09	21.01
8	18.44	18.35	21.60	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	25.42
11.5	20.89	21.20	25.58	26.05
12	21.22	21.68	26.02	26.67
12.5	21.56	22.14	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
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

Reproduced with permission of BMJ Publishing Group from Cole *et al.* (2000) *British Medical Journal*; 330: 1240.

underweight. New international growth references are being developed based on pooled data from seven countries, combining measurements from over 13 000 breast-fed healthy infants and children. This will provide a more scientifically reliable tool for use in all countries to monitor growth and nutritional status. Other anthropometric measures that are routinely used to monitor growth and nutritional status include head circumference, which is used in young infants, mid upper arm circumference, which is widely used in developing countries as a measure of muscle and fat in both children and adults, and skinfold thicknesses, which measure subcutaneous fat. BMI centiles (Table 7.4),

using Quetelet's ratio (weight in kilograms divided by the square of height in meters) have recently been developed to detect overweight and obesity in children.

Both GH and IGFs can be measured in the plasma and their levels are thought to reflect changes in growth and cellular activity. IGF plasma levels are considerably more stable and do not display as large fluctuations as GH exhibits. Because of this greater stability, plasma levels of IGF can provide a sensitive measure of growth. Growth is a process that is multifactorial. The relative contributions of genetic and environmental factors to growth and development outcomes of an individual are still under investigation. Both overnutrition and undernutrition exert negative and often irreversible impacts on growth at all life stages, and unless addressed in the early years life, long deficits may result.

7.8 Aging

Aging is not a disease. Nor are the so-called diseases of aging – cancer, heart disease, arthritis and senility – the inevitable consequences of advancing years. If we live long enough, changes in body composition, physical function and performance will occur in all of us. Many of these changes, as well as health problems which become more common in old age, have long been attributed to the 'normal aging process'. This section will highlight that these health problems can be delayed to the last few years of life (i.e. compression of morbidity).

Sociodemography

Humans are living longer than ever before with several population life expectancies at birth now exceeding 80 years. Since the early 1970s, life expectancies have increased globally by about 1 year every 3 years. The elderly today are living almost 20 years longer than their ancestors at the beginning of the twentieth century. At present, the proportion of centenarians is also increasing (upwards of 1 in 1000 of the population in economically advantaged countries); however, individuals do not appear to exceed a maximal lifespan of about 120 years. Maximal lifespan may yet increase as biotechnology, lifestyle and health care develop in favor of greater longevity.

Adults are reaching older age in better health and the majority will live independently. Life expectancy is increasing for men and women alike. Between 1981 and 2001 the number of older people in the population increased by 50%, with an even greater increase

in those aged over 70. Although maximum life expectancy has not increased over the past century, average life expectancy has changed substantially. Men born in 2020 can expect to live to 79, and women to 87. Our ability to live longer is, in part, attributable to better nutrition and to other lifestyle changes (e.g. reduced substance abuse, greater recreational opportunities), to improved health care (e.g. reduced infant and maternal mortality, earlier diagnosis and management of cancers and heart disease), to educational and economic improvements, and to better housing (especially less crowding) and social support systems. But as we live longer, our nutritional needs may change, either with 'healthy' aging or because of the advent of disease. Keeping an elderly population well is of great importance for the individuals themselves, for the well-being of society in general (the transfer of knowledge and skills to younger people, especially descendants and a reduced burden on others), and for reasons of available resources to care for the aged. Remarkably, the numbers of elderly people in developing countries now approach and will exceed those in developed countries (Table 7.5), so that the problem is global.

Biological and chronological age and compression of morbidity

Biological and chronological age

Aging may be defined as chronological age (a person's age in years since birth) or biological age (the decline in function that occurs in every human being with time). Some elderly people look and function as though they were older and others as though they were younger at the same 'chronological age'. Prospective studies, where some assessment of biological age has been made during the twentieth century in Sweden, indicate that people are less biologically old at the same chronological age than they used to be, and that this difference may be as much as 10 years of biological age. This is a rather remarkable change and some of it is likely to be attributable to improved lifelong nutrition. It may well be that much of what we currently regard as aging is preventable by nutritional means. In other words, even though genes have a strong influence on biological age, it is now believed that lifestyle factors also have a strong influence. You may be able to remain biologically younger if you look after yourself in your younger adult years. The question is, what aspects of aging are biologically inevitable, having to do, for example, with the programmed death of cells (apoptosis), and how

Table 7.5 Proportion of the population aged 65 years and over, selected countries 1985 and 2005

	% of population aged 65+	
	1985	2005
Europe		
France	12.4	14.8
Germany (FRG)	14.5	18.9
Greece	13.1	16.9
Hungary	12.5	15.0
Italy	13.0	16.9
Poland	9.4	12.3
Sweden	16.9	17.2
UK	15.1	15.3
North America		
Canada	10.4	12.5
USA	12.0	13.1
Other developed countries		
Australia	10.1	11.4
Japan	10.0	16.5
Less developed countries		
Brazil	4.3	5.8
China	5.1	7.4
India	4.3	6.1
Kenya	2.1 ^a	2.1
Mexico	3.5	4.6

Reprinted from Grundy (1992) with permission from Elsevier Science.

^a1988.

much is it age related? While the clock cannot be turned back in terms of chronological age, the search for prolonging youth continues to invoke much interest and research. The older people are, the more dissimilar they become from others of the same chronological age. Some of this variability may reflect heterogeneity in true rates of aging, but other factors that accompany aging also seem to be of major importance. These include lifestyle factors such as poor eating habits, a sedentary lifestyle and smoking, and the development of disease. Each of these factors can contribute to deterioration in cardiovascular, lung or endocrine functions, thereby accelerating one's apparent rate of aging. For example, declining cardiovascular function was observed in the Baltimore Longitudinal Study of Aging. However, after careful exclusion of those with heart disease, no consistent declines in function with age remained. Thus, the apparent declines in the study group members as they aged were due to inclusion of people with defined disease rather than to the aging process per se. As discussed later in this chapter, the

accumulating effects of years of poor eating habits can increase the risk of many health conditions as one grows older; yet, it is never too late to change!

Compression of morbidity

The accumulating effects of years of poor eating habits can increase the risk of many health conditions as one grows older. The good news is, however, that food habits may be amenable to modification. In other words, we can adopt lifestyle habits such as regular exercise and healthy eating that will slow functional decline and compositional changes within the limits set by genetics. It is possible to compress morbidity into the last few years of life (i.e. increase health span potential) if we take care of lifestyle and environmental factors throughout life, even once we reach old age. For example, an exercise intervention study in mid-life has been shown to compress morbidity (measured with disability score) towards the end of life. Several of the health problems and bodily changes experienced by older adults that have been attributed to the normal aging process are increasingly being recognized as linked to lifestyle or environmental factors. For example, decline in LBM and increases in body fat which tend to occur as people grow older cannot be entirely attributed to the aging process per se. A major contributor to these changes is the increasingly sedentary nature of people's lifestyles as they grow older in Western countries. Social and physical inactivity and inadequate nutrient and phytochemical intakes are now thought to be instrumental in trying to compress morbidity towards the end of life, and in maintaining or increasing physiological and nutritional reserves.

Physiological reserves, frailty and prevention strategies

Many bodily functions remain relatively unaffected until about 75 years of age when, on average, they start to decrease more noticeably. Nutritionally related health problems are often compounded in later life by reduced physiological reserves of many organs and functions. This applies to both reduced metabolic tissues (e.g. insulin resistance or reduced insulin response to a meal load or a greater glycemic response to the same food) and organ tissues (e.g. reduced cardiac reserve means that an added salt load may tip someone into heart failure, whereas otherwise it would not). While a younger person will be able to consume an inadequate diet with no foreseeable consequences, an elderly person is more likely to experience problems

because of diminished physiological function. Many studies have shown significant reductions in different body functions with age. These may not be inevitable, however. For example, what used to be regarded as a decline in brain function at about the age of 70 may not be seen until much later, raising the possibility that biological age in some body functions may be occurring at a later and later chronological age. Measures of physiological and nutritional reserves may be important indicators of health in older adults. Prevention of associated health problems may be possible if physiological and nutritional reserve levels are known.

Frailty

Avoidance of frailty is one of the major challenges facing older people and their carers. Frailty among older people has been defined as 'a condition or syndrome which results from a multi-system reduction in reserve capacity to the extent that a number of physiological systems are close to, or past, the threshold of symptomatic clinical failure. As a consequence the frail person is at an increased risk of disability and death from minor external stresses' (Campbell and Buchner, 1997). As the number of chronic conditions increases with age they contribute to disability and frailty, which in turn reduce a person's level of independence, sometimes resulting in institutionalization. Falls, incontinence and confusion are regarded as clinical consequences of frailty, and a number of risk factors is associated with each of these conditions. The risk of falling is increased as muscle strength and flexibility decline, and if balance and reaction time are impaired. Urinary incontinence is also a risk factor for falls among elderly people. Dehydration and PEM are two nutritional factors that can contribute to the confusion often experienced by elderly adults. Urinary incontinence often results in elderly people restricting their fluid intake in an effort to control their incontinence or reduce their frequency of urination. Studies are underway in relation to its prevention and management, and there is great interest in the extent to which it is reversible.

Prevention strategies

The major prevention strategies that elderly individuals can take to increase their physiological and nutritional reserves include:

- consuming a wide variety of foods
- engaging in physical activity, as this maintains lean muscle and bone mass, thus increasing nutritional

and physiological reserves to prevent major health problems

- engaging in social activity
- avoiding substance abuse (including alcohol, tobacco, excessive caffeine intake and unnecessary intake of medications).

By focussing on the complete lifestyle rather than on just one component such as nutrition, elderly people can enjoy life without experiencing major consequences of nutritional error.

Food variety

Research has shown that food variety has an important role to play in the prevention of onset of diseases such as diabetes, cancer and cardiovascular disease. A varied diet, ideally containing 20–30 biologically distinct foods a week, is seen to be beneficial in the prevention of certain disease states. Specifically, an association between increased food variety and lower glycemic response in both insulin-dependent and non-insulin-dependent diabetes mellitus (IDDM and NIDDM) has been found. Greater dietary diversity has also been found to be predictive of less morbidity and greater longevity in people aged over 70 years. Mortality follow-up studies of elderly people aged 70 and over in Australia, Greece, Spain and Denmark have found that more varied food patterns, even as late as 70 years and onwards, could reduce the risk of death by more than 50%. To obtain this mortality advantage, the elderly in these studies had to have food patterns consistent with the following food groups, giving a score ranging from 0 to 8: (1) high in vegetables (>300 g/day); (2) high in legumes (>50 g/day); (3) high in fruits (>200 g/day); (4) high in cereals (>250 g/day); (5) moderate in dairy products (<300 g of milk/day or equivalent in cheese/yogurt); (6) moderate in meat and meat products (<100 g/day); (7) moderate in alcohol (<10 g/day); (8) high in monounsaturated fat (mainly from olive oil) and low in saturated fat (i.e. high monounsaturated:saturated fat ratio). This food pattern is consistent with food patterns prevalent in Greece in the 1960s, when Greeks enjoyed the longest life expectancy in the world. The subjects achieved greater mortality advantage if they followed the entire food pattern (i.e. had high dietary variety scores ≥ 4) as opposed to just achieving the required amount for one or two of the food groups (Figure 7.5).

This suggests that there may be synergy between the food groups and that we need to follow dietary

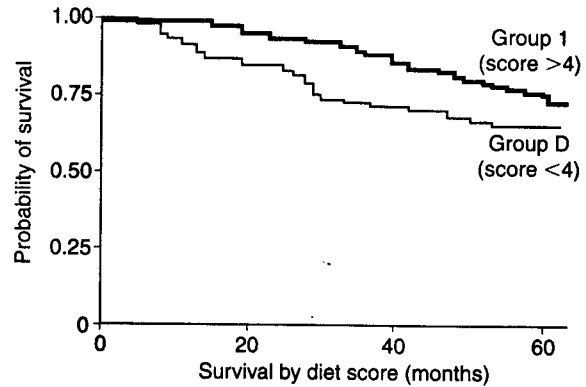


Figure 7.5 Kaplan-Meier survival curves for individual subjects with diet score up to 3 and 4 or more. (Source: Trichopoulos *et al.*, 1995.)

recommendations as a whole rather than focussing on just one food group or nutrient.

Physical activity

Ageing as we know it in modern society is, in many ways, an exercise deficiency syndrome, implying that we may have far more control over the rate and extent of the ageing process than we previously thought (Fiatarone, 1996).

Some of the most dramatic changes that we see with age are changes in body composition. A decline in muscle mass and increases in body fat tend to occur as people grow older. What is often not appreciated, however, is that these too cannot be blamed on the aging process *per se*. A major contributor to these changes is the increasingly sedentary nature of people's lifestyles as they grow older. Reduced physical activity leads to loss of muscle, and as a direct consequence basal metabolic rate (BMR) falls. A lower metabolic rate means that we need to eat less in order to maintain the same body weight. If one does indeed eat less to avoid weight gain, rather than remaining (or becoming) active, it becomes increasingly difficult to meet the needs for essential nutrients. Without doubt, it is preferable to keep physically active, maintain muscle mass and continue to enjoy eating.

Physical activity has been associated with greater energy intakes and subsequently nutrient intakes and quality of life in the aged. This results in a higher plane of energy nutrition. This runs counter to the disturbing advocacy that energy restriction prolongs life. These studies were conducted on rats and have no direct application to humans. To suggest that elderly

people restrict their food intake to prolong life is absurd when this may contribute to frailty and loss of lean mass. The evidence is, however, that any extra energy intake must be from nutrient (and phytochemical)-dense foods, without excessive abdominal fatness. Many studies have shown energy intake declines with age, making a nutritionally adequate diet more difficult to achieve. Older men consume about 800 kcal less than younger men, and older women consume about 400 kcal less than younger women. A reduction in BMR is partly responsible for this decline in energy intake, but physical inactivity appears to be the major cause. Prospective studies show that increased energy intakes in the order of 300–500 kcal/day, which is balanced with increased physical activity to avoid fat gain, confer either decreased cardiovascular or total mortality and improve life expectancy. Physical activity also seems to protect against osteoporosis and fractures, diabetes, and breast and colon cancers, to improve mental health and cognitive function, reduce symptoms of anxiety and depression and enhance feelings of well-being in older people. An exercise intervention study in mid-life has been shown to compress morbidity (measured with disability score) towards the end of life. The subjects who belonged to a 'runner's club' in mid-life had significantly less disability in their eighties compared with control subjects. While the evidence points to the value of early and lifelong regular physical activity, recent evidence underlines just how much survivors can gain from the combination of endurance and strength training well into later life, with studies available on people well into their eighties. In other words, physical activity in old age can defer morbidity and mortality, and compress the morbidity period before death.

Social activity

Social activity is now thought to be one of the most important determinants of longevity. Participation in fewer social activities outside the home and limited social networks have been linked with higher mortality in old age. The impact of social activity on longevity could be through its impact on psychological well-being and nutrition. For example, elderly people who are socially isolated, lonely, institutionalized, recently bereaved and socially inactive have been found to have inadequate food intakes. Glass *et al.* (1999) examined associations between social (e.g. church), productive (e.g. shopping) and physical/fitness activities (e.g. walking)

at baseline and 13 year survival in 3000 older people. Social and productive activities were found to be as effective as fitness activities in lowering the risk of death. Further studies of this kind indicate the importance of social activity to the health and mortality of older people and perhaps younger people as well.

Effects of aging on physiological function

Physiological changes that occur with aging contribute to the body's declining function which, in turn, influences nutritional status just as growth and development do in the earlier stages of the life cycle. Among physiological changes, hormone activity alters body composition, immune system changes raise the risk of infections and some chronic diseases, atrophic gastritis interferes with nutrient digestion and absorption, and tooth loss and depression can adversely influence food choice. Animal studies suggest that energy restriction promotes longevity, but human studies suggest that 'eating better, not less' is desirable.

Theories of aging and energy restriction

There are three main theories of aging which predict the role that genes play in the aging process:

- programmed aging
- error theory
- free radical theory.

The theory of programmed aging suggests that the body has a built-in clock that begins ticking at birth. This theory is supported by the discovery that normal cells have a limited capacity to divide, because telomeres (the sections of DNA at the end of chromosomes) shorten at each division, resulting in a fixed lifespan for each normal cell. Furthermore, the aging process accelerates so rapidly in some individuals that they become biologically 'old' in their teens. The error theory attributes aging to increasing damage of DNA, and the progressive decline in the function of specialized enzymes that repair DNA. It is thought that diseases such as cancer, heart disease, osteoporosis and diabetes may be the result of accumulation of errors. The free radical theory proposes that free radicals (highly reactive oxygen molecules) are produced by oxygen-consuming biological reactions in the body. Free radicals damage cells and have been implicated in the development of cancer and heart disease. There is no evidence that taking antioxidants will improve longevity, but antioxidants consumed from food may have an indirect effect

by reducing the damage produced by free radicals. A free radical is a molecule with an unpaired, highly reactive electron which is often associated with the development of cancer, arteriosclerosis, autoimmune diseases and aging. Antioxidants include phytochemicals (flavonoids) and nutrients (vitamins C and E and β -carotene), as well as enzymes such as superoxide dismutase (SOD), catalase and glutathione peroxidase. These prevent most, but not all, oxidative damage. Bit by bit, the damage mounts and contributes, so the theory goes, to deteriorating tissues and organs. Antioxidants may reduce the risk of cancer by protecting cellular DNA from free radical damage.

There has been a popularized view, derived mainly from rodent experiments, which has argued that energy restriction may decrease the cancer risk and increase longevity. Most of these studies are flawed insofar as extrapolation to humans is concerned because either they are conducted from early life with excessive early mortality, or they do not account for energy expenditure, and therefore energy balance, reflected in body fatness and/or its distribution. Where the full energy equation is available, increased energy throughput (e.g. higher energy intakes with no increase in body fatness) has been associated with decreased cancer risk and/or increased life expectancy. Increased energy intake (and possibly its frequency) has in its own right been associated with increased cancer risk at several sites. Again, the quality of extra food intake seems important. The Zutphen prospective study in the Netherlands showed that increased energy intake, which included relatively more plant-derived food and fish, was associated with lower cancer and total mortality over 10 years.

Body composition

Some of the most dramatic changes seen with age are changes in body composition. A decline in muscle and bone mass and increases in body fat tend to occur as people grow older, and subcutaneous fat is redistributed from limbs to the trunk. Some of these changes occur because of the activity of some hormones that regulate metabolism decreases with age (e.g. insulin, GH, androgens), while the activity of others increases (e.g. prolactin): the former contribute to a decrease in lean mass and the latter to an increase in fat mass. However, what is often not appreciated is that these cannot be blamed entirely on the aging process per se. A major contributor to these changes is the increasingly sedentary nature of people's lives as they grow

older. Reduced physical activity leads to loss of muscle, and as a direct consequence BMR falls. A lower metabolic rate means that older people need to eat less energy in order to maintain the same body weight, but should ensure that this does not lead to a decrease in micronutrient intake. The incidence of people becoming underweight has been shown to increase with age. A lower body weight has been more strongly linked with morbidity in the elderly than mild to moderate excess weight, and the problem is often insidious. Survival rates in Finnish elderly (85 years and over) over a 5 year period showed the highest mortality to be in those with a BMI less than 20 kg/m^2 and the lowest mortality to be in the group with a BMI of 30 or more. Other studies have shown that elderly with BMIs below 27 kg/m^2 lived shorter lives than those with higher BMIs. Weight change and especially weight loss are of greater concern in the elderly than over-fatness. In developed countries 30–50% of older adults have been reported to be at high risk of developing health problems as a result of an inadequate food and nutrient intake.

Immune system

Both physical stressors (e.g. alcohol abuse, other drug abuse, smoking, pain, heat, illness) and psychological stressors (e.g. divorces, exams, migration, loss of a loved one) elicit the body's stress response: the classic fight-or-flight response. Stress that is prolonged or severe can drain the body of its reserves and leave it weakened, aged and vulnerable to illness. As people age, their ability to adapt to both external and internal stressors, especially via the immune system, is diminished owing to physiological changes that occur with aging. The immune system can also be compromised by nutrient deficiencies (see below), and so a combination of age and subclinical nutrient deficiencies makes older adults vulnerable to infectious diseases, including chronic diseases where the immune system is involved, such as arthritis and cancer.

Gastrointestinal tract

The intestine loses strength and elasticity with age; this slows motility and increases the risk of developing constipation (which is four to eight times more common in the elderly than in younger adults). Atrophic gastritis is also more common among older adults; about 30% of adults aged over 60 have this condition. It is a condition characterized by chronic inflammation of

the stomach, accompanied by a diminished size and functioning of the mucosa and glands, resulting in less hydrochloric acid being secreted and increased levels of bacteria. These changes in the stomach can impair digestion and absorption of nutrients, especially vitamin B₁₂, biotin, calcium and iron.

Tooth loss

Chewing can be painful or difficult in old age as a result of tooth loss, gum disease and ill-fitting dentures. This can result in a reduced variety of foods consumed and an increased risk of developing nutrient deficiencies.

Sensory loss

Changes in taste and smell are variable and are often associated with lifelong cigarette smoking, poor dental hygiene and disease. Nevertheless, this phenomenon may make eating less enjoyable and may partly explain why older people tend to increase salt intake and use caffeinated beverages (caffeine also increases their appetite). Aging is associated with a decrease in the opioid (dynorphin) feeding drive and an increase in the satiety effect of cholecystokinin. Recent studies suggest that the early satiety in older persons may be caused by a nitric oxide deficiency, which decreases the adaptive relaxation of the fundus of the stomach in response to food.

Psychological changes

Depression is common among older adults, but is not an inevitable component of aging. It is frequently accompanied by loss of appetite and of motivation to cook.

Nutritionally vulnerable older adults

Contrary to the popular 'tea 'n' toast' myth, it appears that many older adults outside institutions eat reasonably well. The dietary patterns of older adults have generally been found to be similar to or healthier than those of their younger counterparts. Nevertheless, their intakes of cereals, fruit, vegetables and milk products are still below the recommended amounts. Some older people may consume a higher calorie diet without adverse health effects, which may be attributed to higher physical activity levels or intake of protective nutrients such as phytochemicals. Aging is often associated with less efficient processing of some essential nutrients, so older people may require higher intakes of particular nutrients, while requiring lower intakes of others.

As a general guide, adult energy requirements decline by an estimated 5% per decade. In developed

countries energy intakes fall with advancing age (from 2800 to 2000 kcal for men and from 1900 to 1500 kcal for women), but average intakes of protein, total fat, polyunsaturated n-6 linoleic acid, vitamin A, thiamin, riboflavin, niacin, vitamin C, iron and phosphorus remain adequate in the 65-plus age group. Saturated fat and refined carbohydrates (high sugar content) continue to be consumed in excess of the recommended levels, and monounsaturated fats, n-3 fatty acids (from plants and fish), unrefined carbohydrates, fiber, folate, vitamin B₆, calcium, magnesium and zinc tend to be below the recommended intakes. These intakes may not result in the appearance of any diagnostic features or symptoms of true deficiency, but may result in subtle or subclinical nutrient deficiencies. In developed countries mild vitamin and mineral deficiencies are very common in older people, particularly those in institutions; 30–50% of older adults have been reported to be at high risk of developing health problems as a result of an inadequate food and nutrient intake, including cognitive impairment, poor wound healing, anemia, bruising, and an increased propensity for developing infections, neurological disorders, stroke and some cancers (e.g. vitamin A deficiency is associated with lung cancer).

Nutritionally vulnerable 'at-risk' groups

Some subgroups within older populations appear more likely to be consuming inadequate diets (e.g. less regular consumption of cooked meals). In Australia in 1998, 50% of older people lived with their partner and 63% lived with at least one relative; 28% lived alone and 6% in cared-for accommodation. Providing nutritious food via a Meals-on-Wheels program may not overcome the associated problem of social isolation, a risk factor for poor nutrition. This may be overcome by encouraging the individual to eat with family or friends, as this has been shown to increase food intake. An elderly person may eat less food for several reasons.

Nutritionally vulnerable 'at-risk' groups within older populations who are more likely to be consuming inadequate diets (e.g. less regular consumption of cooked meals) and to be at risk of protein–energy malnutrition include those who are:

- institutionalized
- older men living alone
- from low socioeconomic status groups

- socially isolated and lonely
- recently bereaved
- depressed or cognitively impaired
- physically and socially inactive

and those with:

- physical handicaps, impaired motor performance and mobility
- presence of chronic diseases (e.g. arthritis, diabetes, hypertension, heart disease, cancer)
- polypharmacy (unnecessary intake of medications, drug–nutrient interactions; some drugs affect appetite/mood and cause nausea)
- sensory impairment: taste/smell (reduction in taste), eyesight (cataracts)
- reduced sense of thirst (hypodipsia)
- problems with chewing (loss of teeth and poorly fitting dentures)
- limited food storage, shopping difficulties, inadequate cooking skills
- erroneous beliefs and food faddism, food preferences.

Medications, depression, dementia, chronic illness, disability, loneliness and diminished senses of smell and taste may decrease the pleasure of eating. Food beliefs in relation to health can be strongly held among elderly people and lead to both food faddism and undesirable food avoidance. There may be a significant association between food beliefs and food habits, as evidenced in studies of various elderly communities around the world. Nutrients at greatest risk of inadequate intake in 'at-risk' elderly groups are:

- energy
- protein
- folate
- vitamin B₆
- vitamin B₁₂
- vitamin D
- zinc
- calcium
- magnesium
- phytonutrients
- water.

Low intakes of these nutrients have important implications for bone health (calcium), wound healing (zinc, protein, energy), impaired immune response (zinc, vitamin B₆, protein, energy) and vascular disease via elevated homocysteine levels (folate, vitamin B₆).

Risky food patterns

When older people are physically active, marginal food patterns are less likely to lead to problems of the aged such as:

- frailty
- protein energy dysnutrition
- micronutrient and phytochemical deficiency, because greater amounts of nutritious food can be eaten without positive energy balance
- chronic metabolic disease (NIDDM, cardiovascular disease, osteoporosis) and certain cancers (breast, colonic, prostate)
- depression (there is growing evidence that n-3 fatty acid deficiency can contribute to depression in some individuals, and that exercise can alleviate it)
- cognitive impairment with the apoE4 genotype; excess dietary saturated fat is likely to increase the risk of Alzheimer's disease; and some antioxidants such as vitamin E and glutathione may reduce the risk.

Specific risky food patterns in later life include:

- large rather than smaller frequent meals or snacks, because of the inability of insulin reserve to match the carbohydrate load in those proven to have impaired glucose tolerance (IGT) or in those with NIDDM; or because, where appetite is impaired, nutritious snacks can help to avoid chronic energy undernutrition
- alcohol excess, no alcohol-free days and/or alcohol without food (since food reduces the impact of alcohol ingestion on blood alcohol concentration and its consequences)
- eating alone most of the time (since social activity encourages interest in food and, usually, healthy food preferences)
- use of salt or salty food rather than intrinsic food flavour (especially as taste and smell tend to decline with age), as excess sodium contributes to hypertension through an increased Na/K molar ratio, and to salt and water retention in cardiac decompensation, and promotes urinary calcium loss.

Nutrients at risk of inadequate intake

Protein–energy dysnutrition

It is usual to speak about protein–energy malnutrition (PEM), otherwise known as protein–calorie malnutrition (PCM), but in the aged, the body compositional disorder may be rather more complex. The most

common nutritional scenario in the aged is for there to be a decrease in lean mass (comprising water and protein-dominant tissues such as muscle, organs such as liver, and also bone) and an increase in abdominal fat. This disorder could not be described as PEM, but can be described as protein-energy dysnutrition (PED). Illness or inadequate food intake may result in PED, a condition more common among elderly adults, especially in institutionalized care. It is associated with impaired immune responses, infections, poor wound healing, osteoporosis/hip fracture and decreased muscle strength (frailty), and is a risk factor for falls in the elderly. About 16% of elderly people living in the community consume <1000 kcal/day, an amount that cannot maintain adequate nutrition. Undernutrition also occurs in 3–12% of older outpatients, 17–65% of older people in acute-care hospitals and 26–59% of older people living in long-term care institutions. Studies show that being underweight in middle age and later places a person at greater risk of death than being overweight. Marasmus is a condition of borderline nutritional compensation in which there is marked depletion of muscle mass and fat stores but normal visceral protein and organ function. Because there is a depletion of nutritional reserves, any additional metabolic stress (e.g. surgery, infection, burns) may rapidly lead to kwashiorkor (hypoalbuminemic PEM). Characteristically, elderly people deteriorate to this state more rapidly than younger people, and even relatively minor stress may be the cause. Usually, susceptible elderly people are underweight, but even those who appear to have ample fat and muscle mass are susceptible if they have a recent history of rapid weight loss. The protein requirements of older people seem to be similar to or higher than those of younger people. The current dietary recommendation of protein for adults is 0.75–0.8 g/kg, whereas the recommendation for the elderly is slightly higher at 0.91 g/kg. In elderly people with PEM, edema is usually absent, and serum albumin and hemoglobin levels, total iron-binding capacity and tests of cell-mediated immune function are usually normal. When hypoalbuminemic PEM occurs, the serum albumin level is <3.5 g/dl, and anemia, lymphocytopenia and hypotransferrinemia (evidenced by a total iron-binding capacity <250 µg/dl) are likely. Often, anergy and edema are present. Albumin has a 21 day half-life, and is an excellent measure of protein status, except in people suffering from illness or trauma and after surgery. Normal, able-bodied elderly people

should have serum albumin levels >4 g/dl; only when a person is recumbent do fluid shifts result in a normal albumin level of 3.5 g/dl. Albumin levels <3.2 g/dl in hospitalized older people are highly predictive of subsequent mortality. Cholesterol levels <160 mg/dl in nursing-home residents predict mortality, presumably because such levels reflect malnutrition. Acute illness associated with cytokine release can also lower cholesterol levels. Anergy (failure to respond to common antigens, such as mumps, injected into the skin) can occur in healthy as well as malnourished older people. The combination of anergy and signs of malnutrition correlates more strongly with a poor outcome than either one alone.

Folate

Before mandatory folate fortification of cereals in several developed countries such as America and Australia, many older adults did not consume enough folate. This was compounded by the fact that folate absorption appears to be affected by atrophic gastritis, which is common in older adults. Elevated homocysteine levels have recently been defined as a marker of poor folate status in older people, and elevated levels of the former have been linked with an increased risk of heart disease and strokes. Folate metabolism may also be altered by the ingestion of antacids, anti-inflammatory drugs and diuretics commonly used by older adults. Several governments have legislated for the fortification of grain products with artificial folate since about 1998. The aim of this fortification exercise was to reduce birth defects of the spine, called spina bifida. Pregnant women need enough folate early in pregnancy to prevent most cases of spina bifida. A study in Framingham, Massachusetts, USA, studied folate levels in over 1100 people, before and after fortification was mandated. The folate levels in blood more than doubled, and the percentage of people, including older adults, with low folate levels dropped by more than 90%. This is partly explained by the greater bioavailability of artificial folate in fortified foods. For example, only about half of the folate found naturally in food is available for use in the body; in contrast, the folate found in fortified foods is nearly all absorbed.

Can folate fortification have any adverse effects on elderly people? Vitamin B₁₂ deficiency is quite common in older adults owing to inadequate absorption (see below). Vitamin B₁₂ is needed to convert folate to its active form. Therefore, one of the most obvious

vitamin B₁₂ deficiency symptoms is the anemia of folate deficiency (megaloblastic anemia). Vitamin B₁₂, but not folate, is also needed to maintain the sheath that surrounds and protects nerve fibers. Either B₁₂ or folate will clear up the anemia, but if folate is consumed via fortified foods when a B₁₂ supplement is needed instead, the result is devastating owing to permanent nerve damage and paralysis. In other words, folate 'cures' the blood symptoms of a vitamin B₁₂ deficiency, but allows the nerve symptoms to progress, so that folate can mask a vitamin B₁₂ deficiency. With sufficient folate in the diet, the neurological symptoms of vitamin B₁₂ deficiency can develop in older adults without evidence of anemia. This highlights some of the safety issues surrounding the fortification of the food supply.

Vitamin B₆

During the course of life, plasma vitamin B₆ falls by approximately 3.6 µmol/l per decade. A number of studies suggest that age-related changes occur in both the absorption and metabolism of this vitamin, and as a consequence aged adults may have a higher requirement. Studies also show that vitamin B₆ deficiency results in decreased immune response. Vitamin B₆ deficiencies (as well as vitamin B₁₂ and folate) also result in higher concentrations of homocysteine. Supplementation of vitamin B₆ in healthy elderly has been found to improve immune function and long-term memory.

Vitamin B₁₂

The prevalence of pernicious anemia increases with age, as does atrophic gastritis, and the absorption of vitamin B₁₂ is reduced in individuals with either condition. The prevalence of *Helicobacter pylori* also increases with age and has been shown to be associated with vitamin B₁₂ malabsorption, possibly because it contributes to gastric atrophy. As the likelihood of vitamin B₁₂ deficiency is more common among older adults this not only increases the risk of irreversible neurological damage but is likely also to contribute to megaloblastic anemia and homocysteine concentrations associated with vascular disease (see Folate).

Vitamin D

Older adults are at greater risk of vitamin D deficiency than are younger people, and therefore at greater risk of exacerbated bone health decline (and resulting osteopenia and osteoporosis).

Risk factors for vitamin D deficiency include:

- lack of exposure to sunlight (may be due to less physical activity or sunscreen use)
- decline in renal function
- impaired skin synthesis (may be due to aging skin)
- low fish intake (especially fatty fish)
- low intake of egg yolks, butter, vitamin D-fortified margarine and cheese.

The diet becomes an important source of vitamin D in people who do not receive enough sunlight. Diets of elderly people are often deficient in vitamin D-rich foods such as oily fish, and fat-soluble vitamin absorption may be impaired. This is thought to contribute to the high incidence of vitamin D deficiency in older people. It appears that in the USA and Great Britain, some 30–40% of older patients with hip fractures are vitamin D deficient. However, even during old age, improving vitamin D status can provide profound benefits for bone health. In a Finnish study of outpatients over the age of 85 years and municipal home residents aged 75–84 years, those randomly assigned to receiving an annual vitamin D injection had significantly fewer fractures over a 5 year follow-up period. Probably the most striking and impressive study is one by Chapuy *et al.* (1992) of a nursing-home population of 3270 women, with an average age of 84 years. In a randomized, controlled trial of vitamin D (20 µg/day) and calcium (1200 mg/day), those receiving the supplement experienced 43% fewer hip fractures and 32% fewer non-vertebral fractures over an 18 month period. If blood levels of vitamin D are not reduced, vitamin D resistance may occur. Vitamin D resistance is relatively common because of impaired renal function in later life and the best indicator of this is an elevated parathyroid hormone (PTH) concentration in blood, a phenomenon referred to as secondary hyperparathyroidism. Vitamin D is important not only for bone, but also for immune function and muscle strength, and as a cell differentiator to reduce the risk of neoplastic disease.

Zinc

Plays an important role in wound healing, taste acuity and normal immune function, and may affect albumin status in older adults. It is a crucial element in numerous metalloenzymes, and its intake is dependent on foods such as meat, and limited from plant foods, in which it is bound to phytic acid, oxalate and dietary

fiber. It is more bioavailable in cereals that are leavened because of the presence of phytase in yeast, which breaks down phytic acid. Low zinc intakes are associated with low energy and meat intakes. Older adults may absorb zinc less effectively than younger people, and so a diet including zinc-rich foods is important in later life. Zinc deficiency in older people is likely to be an important contributor to proneness to infection, in particular respiratory infection, such as pneumonia. Some of the symptoms of zinc deficiency are similar to symptoms associated with normal aging such as diminished taste and dermatitis, so it is difficult to determine whether to attribute these symptoms to zinc deficiency or simply to the aging process.

Calcium

Aging is associated with a decrease in calcium absorption, which is probably due to alterations in metabolism of vitamin D. However, calcium is a very important nutrient in older age, as osteoporosis becomes a problem. Postmenopausal women not on hormone replacement therapy (HRT) have higher calcium needs. Many women do not meet the current Australian recommended calcium intake for postmenopausal women (1000 mg/day). Recent studies suggest that postmenopausal women need 1500 mg calcium/day. It is recommended that elderly people who suffer from a milk allergy or are lactose intolerant seek calcium from non-milk sources or supplements to help them to meet their daily requirements.

Phytonutrients

Phytochemicals (from the Greek *phyto*, meaning plant) are unlike vitamins and minerals in that they have no known nutritional value. Phytochemicals are naturally occurring plant secondary metabolites that plants produce to protect themselves against bacteria, viruses and fungi. Many phytochemicals function as antioxidants, which protect cells from the effects of oxidation and free radicals within the body. They have been recognized only recently as potentially powerful agents that may offer protection from diseases and conditions such as heart disease, diabetes, some cancers, arthritis, osteoporosis and aging. They are present in a number of frequently consumed foods, especially fruits, vegetables, grains, legumes and seeds, and in a number of less frequently consumed foods such as licorice, soy and green tea. Phytonutrients may play a protective role in cardiovascular

disease, certain cancers and menopausal symptoms. Phytonutrients are likely to contribute to protection against many of the diseases associated with aging. A diet rich in phytoestrogens (isoflavones, lignans) may lessen the symptoms and impact of the menopause by improving vaginal health, reducing the incidence of hot flushes and improving bone mineral content (BMC). Food sources of phytoestrogens include soy, chickpeas, sesame seeds, flax seed (linseed) and olives.

One study in particular has shown that ingestion of soy may be more effective than HRT in improving BMC and therefore reducing the risks of osteopenia and osteoporosis.

In studies looking at the effect of HRT on BMC, it took 36 months to achieve an increase of just under 4%. In another study, however, an increase of 5.2% in BMC was detected after just 12 weeks of soy consumption.

Water

Total body water declines with age. As a result, an adequate intake of fluids, especially water, becomes increasingly important in later life, as thirst regulation is impaired and renal function declines. Dehydration is a particular risk for those who may not notice or pay attention to thirst, or who may find it hard to get up to make a drink or reach the bathroom. Older people who have decreased bladder control may also be at risk because they may be afraid to drink too much water.

Dehydrated elderly people appear to be more susceptible to urinary tract infections, pressure ulcers, pneumonia and confusion. Recommended intakes for the elderly are approximately 6–8 glasses of fluid a day, preferably water.

Nutrition-related health problems in the aged

There is growing awareness that the major health problems, and even mortality, in the aged have nutritional contributors and can be (in part) prevented by food intake. These health problems do not necessarily need to occur with aging, and death can be delayed. As the number of chronic conditions increases with age they contribute to disability and frailty, which in turn reduces a person's level of independence, sometimes resulting in institutionalization. The primary nutritional problems affecting the elderly are:

- protein–energy dysnutrition
- subclinical/mild vitamin deficiencies and trace mineral deficiencies
- obesity

all of which can contribute to the development of chronic conditions seen with aging. Some common nutrition-related problems in the aged are outlined below.

Sarcopenia

The condition or state of sarcopenia is specifically involuntary loss of flesh or muscle that occurs with age, and is more marked in women. It has been demonstrated that reduced muscle mass and body cell mass is associated with a loss of muscle strength, and impaired immune and pulmonary function. Furthermore, this decline in muscle strength is responsible for much of the disability observed in older adults, and in the old elderly, as muscle strength is a crucial component of walking ability. It is thought that human life cannot be sustained if levels of body cell mass fall below 60% of the normal levels of young adults. The prevalence, incidence and etiology of sarcopenia are currently unknown, and therefore require further study. Decreasing physical activity and GH levels are two likely contributing factors to the advancement of sarcopenia, along with poor nutrition (especially inadequate energy and protein intakes, which may be due to poor food intake or disease), disease and the aging process.

Obesity

Overweight and obesity are common problems in the aged, not because they are an inevitable part of growing older, but because of the associated sedentary lifestyle. Though a less serious problem in older persons than PEM, obesity can impair functional status, increase the risk of pulmonary embolus and pressure sores, and aggravate chronic diseases such as diabetes mellitus and hypertension. Greater body fatness, especially if centrally distributed, increases the risk of insulin resistance, hypertension and hypercholesterolemia in the aged. In contrast, heavier women have a lower risk of hip fracture. This is partly due to 'padding' and better muscle development, but may also be due to maintenance of higher estrogen levels from the conversion of precursor steroids to estrogen in adipose tissue. Abdominal obesity is defined as an abdominal circumference of greater than 102 cm and 88 cm for men

and women, respectively. However, abdominal obesity can be reduced in old age by engaging in some form of daily physical activity. An appropriate body weight is a protective factor in older people with advancing age. Body weight maintenance at a suitable level is desirable to maintain physical strength and activity, resistance to infection and skin breakdown, and quality of life.

Immune function

Infections are a common cause of illness and death among the aged. Aging adults are more susceptible to infection and this is probably due, in part, to the age-associated decline in immune function, but this decline may be preventable with good nutrition and physical activity. A decline in immunity may also increase the risk of cancer and arthritis. The observed decline in immune function with aging may be prevented with nutrient intakes greater than those currently recommended for 'normal' health. Nutrients important in immune function include protein, zinc, vitamin A, vitamin C, pyridoxine, riboflavin and tocopherols. Other food components not considered to be essential for health in earlier life may become more important with age.

The non-essential amino acid glutamine has an important role in DNA and RNA synthesis. It is stored primarily in skeletal muscle, and is utilized by intestinal cells, lymphocytes and macrophages. Because the contribution of skeletal muscle to whole-body protein metabolism declines with age, the rate of glutamine formation and availability may be impaired. As such, it may compromise immune function, resulting in a suboptimal response to infection or trauma. Glutamine can be synthesized from glutamic acid, which is found in wheat, soybeans, lean meat and eggs. Glutathione (a tripeptide) and phytochemicals such as flavonoids and carotenoids also appear to play a role in immune function. Meat is a good source of glutathione, with moderate amounts being found in fruits and vegetables. Whey proteins, although low in glutathione, are capable of stimulating endogenous glutathione production.

Osteoporosis and fractures

Old age is associated with decreased bone mass, and osteoporosis is one of the most prevalent diseases of aging. The incidence of osteoporosis is increasing with the aging population, with females most affected. It has

been estimated that about 25% of the female population over 60 years is affected by osteoporosis, and 70% of the fractures that occur annually in Australia can be attributed to osteoporosis. In 1986, 10 000 hip fractures were recorded in Australia, and this rate is expected to rise to 18 000 per year by 2011. Hip fractures result in both mortality and morbidity. Two types of osteoporosis have been identified. Type I involves the loss of trabecular bone (calcium containing crystals that fill the interior of the bone). Women are more affected by this type of osteoporosis, with the most effective preventive measure being the administration of estrogen for at least 7 years after menopause. Type II osteoporosis progresses more slowly than type I, and involves the loss of both cortical (the exterior shell of the bone) and trabecular bone. As the person ages, the disease becomes evident with compressed vertebrae forming wedge shapes, into what is commonly referred to as 'dowager's hump'. Once again, women are more affected by this disease than men. Women are more prone to osteoporosis than men for two reasons: bone loss is accelerated after menopause, and women have a lower bone mineral density than men. A large study of elderly men and women conducted in Australia found that after the age of 60 years about 60% of women and 30% of men would sustain an osteoporotic fracture. A high intake of calcium appears to prevent or reduce bone loss in postmenopausal women. While adequate intakes of calcium appear to be protective against osteoporosis, other potentially protective factors include vitamins C, D and K, protein, boron, copper and possibly phytoestrogens. Recent evidence indicates that soy consumption may also provide benefits to bone health. A vitamin D supplement from fish liver oil has also been shown to reduce fracture rates in later life. While nutrition and physical activity can maximize peak bone mass during growth, other factors such as excess sodium, caffeine, smoking and alcohol can accelerate bone loss in later life.

Cardiovascular disease

Cardiovascular disease is the most common cause of death and disability in the developed world. Dietary habits may contribute to or provide protection against risk factors associated with cardiovascular disease. In a longitudinal health survey of elderly people living in the Netherlands, an inverse relationship was found between fish consumption and coronary heart disease mortality. Elevated serum

homocystine concentrations have been identified as an independent risk factor for cardiovascular disease. In the Framingham study, elderly adults with better folate status had lower homocystine concentrations. Inadequate intake of folate and vitamins B₆ and B₁₂ can lead to homocystinemia, and then to vascular damage and proneness to thrombosis.

Cancer

Specific dietary patterns that protect against cancer remain unclear. However, certain food groups are associated with a reduced risk of cancer; for instance, a high intake of fruit and vegetables appears to be associated with a reduced risk of cancer at many sites. Fruit and vegetables are excellent sources of antioxidants, phytochemicals and dietary fiber. Particular foods that may protect against prostate cancer include soy products, tomatoes and pumpkin seeds. Foods high in resistant starch, dietary fiber and salicylates may protect against colorectal cancer. Foods that appear to increase risk of cancer at specific sites include salt and smoked/cured foods (stomach cancer) and alcohol (esophageal cancer). Factors that occur early in life may affect the risk of breast cancer in later life. For instance, rapid early growth, greater adult height and starting menstruation at a younger age are associated with an increased risk of breast cancer. Although it is unlikely that appropriate interventions could be undertaken to avoid these, other nutritional and lifestyle factors are amenable to change and may reduce the risk of breast cancer. These include consuming diets high in vegetables and fruits, avoiding alcohol, maintaining a healthy body weight and remaining physically active throughout life. There is some evidence that phytoestrogens (compounds found in plants that possess mild estrogenic properties) may reduce the risk of breast cancer. Soy and linseed are two excellent sources of phytoestrogens and recently Australian food manufacturers have been adding soy and linseed to a variety of breads and cereals. The increase in prevalence of nutritionally related immunodeficiency with aging is likely to contribute to the development of neoplastic disease.

Diabetes

Aging is associated with an increased prevalence of NIDDM and glucose intolerance. Two risk factors associated with the development of both these conditions include obesity and physical inactivity. In older adults, modest weight reductions can contribute to improvements in diabetic control. This

is important as retrospective studies indicate that good blood glucose control reduces the likelihood and severity of stroke, cardiovascular disease, visual impairment, nephropathy, infections and cognitive dysfunction. Dietary modification can reduce cardiovascular disease risk; even a relatively small reduction in salt and saturated fat intake can have a substantial effect on cardiovascular disease.

Endocrine function

Aging sees a decline in hormone secretion throughout the body. The decline in the level of human GH seems to play a role in the aging process in at least some individuals. GH also plays a role in body composition and bone strength. Estrogen levels also drop with age. Low levels of estrogen are associated with bone thinning, frailty and disability. Low testosterone levels in the body may weaken muscles and promote frailty and disability. Melatonin responds to light and seems to regulate seasonal changes in the body. As melatonin levels decline with age other changes in the endocrine system may be triggered. Dehydroepiandrosterone (DHEA) is being studied for its effects on immune system decline and its potential to prevent certain chronic diseases such as cancer and multiple sclerosis.

Cognitive function

Prevention of cognitive loss or dementia poses a particular challenge in older people. Some deterioration can be attributed to atherosclerotic disease and thus interventions such as aspirin or particular dietary patterns that reduce cardiovascular risk may also prevent dementia. High educational status early in life and continued mental stimulation may also be protective. Living alone has recently been reported to increase the risk of dementia. It is generally accepted that dementing illnesses and depression have a strong genetic background. However, the genetic susceptibility to a certain disease is strongly influenced by environmental factors. Thus, it may be possible to delay the onset of poor cognitive function in old age if food intake is adequate. For example, cognitive status assessed in a group of older adults from Madrid using Folstein's Mini-Mental State Examination and Pfeiffer's Mental Status Questionnaire was found to be better in those who consumed a more satisfactory global diet. This diet was characterized by a greater intake of total food, fruit and vegetables. Dementia can result in forgetting to eat, indifference to food, failure to see the need to eat, and behavioral

abnormalities such as holding food in the mouth. Changes in smell and taste may also lead to weight loss, which is common in older adults with dementia, or even anorexia in older adults. Long-term moderate (subclinical) nutrient deficiencies are now believed to produce memory impairments and declining immunity in older adults. Certain nutrients or toxic substances may directly affect brain development (e.g. alcohol, folic acid deficiency) or brain function (e.g. alcohol, vitamins B₁, B₂, B₆, B₁₂, C and E, and zinc deficiencies). Brain aging is associated with oxidative stress; thus, antioxidants and pro-oxidants (such as iron) are of particular interest. There is some epidemiological evidence that the antioxidants carotene and carotenoids, ascorbic acid and α -tocopherol may delay brain aging and iron may accelerate it. Vitamin K may also be protective against cognitive decline and Alzheimer's dementia. Depression in the elderly is a very common symptom. There is a growing body of evidence to suggest that n-3 polyunsaturated fatty acids may play an important role in the etiology of depression. Caffeine ingested as either tea or coffee has also been shown to improve mood and reduce anxiety.

Nutritional assessment of the aged

One of the greatest difficulties in making any assessment of the aged is the biological heterogeneity ('biological age'). There are clearly many health problems seen in the aged in some communities that are not seen in others, making them more age related than aging. Nutritional assessment of the aged needs to pay attention to a number of sociodemographic variables and the food culture in which the elderly person has lived. Another challenge for nutritional assessment in the aged is the question as to when nutritional factors will have operated during the lifespan to have had consequences on health in later life. With these considerations taken into account, the areas of nutritional assessment to consider are:

- food and nutrient intake
- anthropometry and body composition
- laboratory investigations by way of biochemistry, hematology and immunology
- nutritionally related risk factors for various health problems in the aged.

In Australia a tool has been developed which identifies older adults at risk of poor nutritional health by giving warning signs (Figure 7.6).

DETERMINE YOUR NUTRITIONAL HEALTH

The Warning Signs of poor nutritional health in the older person are often overlooked. Use this checklist to find out if you or someone you know is at nutritional risk.

Read the statements below. Circle the number in the column that applies to you or the person you know. For each answer, score the number in the box. Total your nutritional score.

	YES	NO
I have an illness or condition that made me change the kind and/or amount of food I eat	2	0
I eat at least 3 meals per day	0	3
I eat fruit or vegetables most days	0	2
I eat dairy products most days	0	2
I have 3 or more glasses of beer, wine or spirits almost every day	3	0
I have 6 to 8 cups of fluids (e.g. water, juice, tea or coffee) most days	0	1
I have teeth, mouth or swallowing problems that make it hard for me to eat	4	0
I always have enough money to buy food	0	3
I eat alone most of the time	2	0
I take 3 or more different prescribed or over-the-counter medicines every day	3	0
Without wanting to, I have lost or gained 5 kg in the last 6 months	2	0
I am always able to shop, cook and/or feed myself	0	2
TOTAL		

Add up all the numbers you have circled. If your nutritional score is ...

0-3	Good! Recheck your nutritional score in 6 months
4-5	You are at moderate nutritional risk. See what can be done to improve your eating habits and lifestyle. Your Council on Ageing or health care professional can help. Recheck your nutritional score in 3 months.
6 or more	You are at high nutritional risk. Bring this checklist the next time you see your doctor, dietitian or other qualified health or social service professional. Talk with them about any problems you may have. Ask for help to improve your nutritional health.

Source: These materials were developed and distributed by the Australian Nutrition Screening Initiative, a project of RACGP, Council on the Ageing, Dietitians Association of Australia, and Self Care Pharmacy, a joint program of the Pharmaceutical Society of Pharmacy Guild of Australia.

Figure 7.6 Example of a checklist to identify older persons at risk of poor nutritional health.

Food and nutrient intake

Assessment of food and nutrient intake is an important tool in health assessment in the aged. Because of a positive decline in memory, instruments used for food intake assessment should be as simple and practical as possible and should involve corroboration from other observers, such as family or friends. Knowing about appetite, the special senses for smell and taste, and the overall food patterns, facilitates an understanding of the various factors that may affect food intake. Food and nutrient intake can alert the health-care worker to possible nutritionally related disease, for example, osteoporosis, by asking 'What do you have in the way of dairy products, fish, sesame based foods?' as sources of calcium. A systematic inquiry about food intake usually requires asking about each episode of eating during the day, the main meals and the snacks.

Anthropometry and body composition

Anthropometry is a simple, non-invasive, quick and reliable form of obtaining objective information about a person's nutritional status.

- **Weight:** Ambulatory elderly persons are weighed on an upright balance beam scale or microprocessor-controlled digital scale. A movable wheelchair balance beam scale can also be used for those elderly who can only sit. A bed scale should be available in geriatric hospitals for measuring the weight of bed-ridden elderly patients. Weights less than 20% of the ideal body weight indicate a significant loss of total body protein requiring immediate investigation and action. They are associated with reduced tolerance to trauma and an increased risk of morbidity, infection and mortality. Low body weight and/or unintended weight loss are significant risk factors as the aging process progresses and require careful intervention and monitoring. General guidelines requiring action would be:

- a 2% decrease in body weight in 1 week
- a 5% decrease in body weight in 1 month (3.5 kg in a 70 kg man)
- a 7% decrease in body weight in 3 months
- a 10% decrease in body weight in 6 months.

Interpretation of the weight of elderly people should be done with circumspection. Increases in body weight may indicate overweight/obesity or edema. Decreases in body weight can signify the correction

of edema, development of dehydration or emergence of a nutritional disorder.

- **Height:** For the elderly who are agile and without stooped posture, height should be measured in an upright position. When this cannot be measured, *knee height* (using a knee height calliper) in a recumbent position can be used to estimate stature. The following formulae are used to compute stature from knee height:

$$\text{Stature for men} = (2.02 \times \text{knee height}) - (0.04 \times \text{age}) + 64.19$$

$$\text{Stature for women} = (1.83 \times \text{knee height}) - (0.24 \times \text{age}) + 84.88$$

The knee height measurement in these equations is in centimeters, and the age is rounded to the nearest whole year. The estimated stature derived from the equation is in centimeters. These equations are derived from observations which presume that elderly people will have lost some height, an inevitability that may not always continue as health-care improves.

- **Arm span** is another substitute for height and happens to be the same as maximal height achieved. It is sometimes necessary to ask for maximum adult height to be recalled by the subject or by a carer. Gradual reduction in height may be an indicator of vertebral crush fractures due to osteoporosis, or it may be due to loss of vertebral disk space.
- **Mid-arm circumference (MAC):** Combined with triceps skinfold (TSF), MAC (taken at the mid-point between the acromion and olecranon) can be used to calculate mid-arm muscle area (MAMA), which is an index of total body protein mass. The equation to estimate MAMA is:

$$\text{MAMA} = [\text{MAC} - (3.14 \times \text{TSF}/10)]^2/12.56$$

The MAC measurement in this equation is in centimeters and the TSF is in millimeters. The calculated MAMA derived from the equation is in centimeters squared. MAMA of less than 44 cm² for men and less than 30 cm² for women may indicate protein malnutrition.

- **Calf circumference:** Calf circumference (taken at the largest circumference using inelastic flexible measuring tape) in the absence of lower limb edema can be used to calculate weight in a bed-ridden patient.

Several anthropometric measurements, apart from calf circumference itself (Calf C), are required to compute weight. They are knee height (Knee H), MAC and subscapular skinfold thickness (Subsc SF) (taken at posterior, in a line from the inferior angle of the left scapula to the left elbow). There are separate equations for men and women:

Body weight for men

$$= (0.98 \times \text{Calf C}) + (1.16 \times \text{Knee H}) \\ + (1.73 \times \text{MAC}) + (0.36 \times \text{Subsc SF}) \\ - 81.69$$

Body weight for women

$$= (1.27 \times \text{Calf C}) + (0.87 \times \text{Knee H}) \\ + (0.98 \times \text{MAC}) + (0.4 \times \text{Subsc SF}) \\ - 62.35.$$

All measurements should be in centimeters and the resulting computed weight is in kilograms. Calf circumference is expected to have increasing application for assessment of lean mass. It can also be used as a measure of physical activity in the aged.

- **Anthropometric indices:** BMI has been used widely to estimate total body fatness. BMI can be obtained by using the formula: $\text{BMI} = \text{Weight (kg)} / \text{Height (m}^2\text{)}$. BMI can be calculated to help classify whether the subject is in the reference range. Interobserver errors are possible. Height and weight have coefficient of variations in the order of less than 1%, may be altered by kyphosis in the aged and make interpretation of BMI invalid. The *abdominal* (taken at the midpoint between lower ribcage and iliac crest) and *hip* (taken at the maximal gluteal protrusion) *circumferences ratio* (AHR) is another anthropometric index to estimate fat distribution and the one now recommended by the WHO. It is fat distribution reflected in abdominal fatness, which may account for a number of chronic non-communicable diseases in the elderly if the ratio is above 0.9 for men and 0.8 for women. Several studies are now showing that umbilical measurements alone can be used to safely decide whether weight loss is necessary to reduce the risk from diseases such as heart disease and diabetes. Statistical analyses of umbilical circumferences of Caucasian men and women aged 25–74 years indicated that the ideal circumference for men is less than 102 cm and for women less than 88 cm. These conclusions are drawn from Caucasian subjects and thus may not apply in ethnic groups where the build is slight, such as in many Asian countries, and

where a lesser degree of abdominal fatness may still put the person at risk of developing chronic diseases.

Laboratory investigations by way of biochemistry, hematology and immunology

Biochemical, hematological and immunological assessments are useful to confirm nutritional disorders and to identify specific complications that accompany them in the elderly (see also the chapter on metabolic and nutritional assessment in *Clinical Nutrition* in this series).

Various nutritionally related risk factors for health problems in the aged

Elderly people tend to have different degrees of risk factors (see section on Nutritionally vulnerable 'at-risk' groups).

7.9 Guidelines for healthy aging

Sometimes the assumption is made that, after we turn 65 or 70 years, perhaps lifestyle changes will no longer confer significant benefits. Are the remaining years sufficient to reap the benefits of modifications to food choice or exercise patterns? Several recent intervention and survival studies reveal that improvements in nutrition and regular exercise can benefit health even in advanced old age. For example, older muscles are just as responsive to strength-training exercises as are young muscles. Nonagenarians have shown impressive increases in muscle mass, muscle strength and walking speed with weight-training programs. Chronological age is, in itself, clearly no justification for deciding whether it is worthwhile to pursue lifestyle change. Behavioral risk factors (e.g. regularly not eating breakfast, lack of regular physical activity, overweight, smoking) have been shown to remain predictors of 17 year mortality even in people aged over 70. If elderly people pay attention to aspects of their lifestyle (physical and social activity) other than eating, they may be able to make nutritional errors with less consequence.

Physical activity

The type of physical activity can play an important role in the health of older people. The two principal forms of physical activity or exercise important in promoting health and well-being are endurance/aerobic exercise and strength training. Endurance activities improve heart and lung fitness and psychological functioning, while strength training enhances muscle size and

strength, thus preventing muscle atrophy. The level of physical activity required for older adults to achieve optimal health benefits has not yet been established. Resistance (or strength) training prevents lean muscle atrophy more effectively than aerobic activity, especially during weight loss, whereas aerobic exercise may be more involved in improving psychological functioning in older people (although group membership may also be a factor). Strength training in older adults seems particularly promising in reducing or preventing the decline in muscle mass observed with aging. It can improve walking ability and balance and its associated risk for falls. Strength training also contributes to improved tendon and ligament strength, bone health and improvements in blood sugar levels. The benefits of physical activity such as strength training should make activities of daily living easier for older people. Such activities might include climbing stairs, getting out of a chair, pushing a vacuum cleaner, carrying groceries and crossing a road with sufficient speed. Research suggests that endurance activities should be performed daily (e.g. a walk of 30 min duration or three bouts of 8–10 min), along with some strength training. Endurance activities do not have to be continuous but can be accrued throughout the day through short bursts of activity.

Protective foods and food variety

The first consideration when it comes to nutritional matters is that enough food is available for basic energy and nutritional needs. Food variety is another important consideration in terms of nutritional adequacy and health outcomes. Food variety has been demonstrated to be an accurate predictor of the nutritional adequacy of the diet, and is invariably linked to food availability. Consuming a wide variety of foods (especially plant foods) has been associated with longevity. It is suggested that an ideal way to increase variety in the diet is to choose foods from across all five food groups, and a wide selection within each of these groups. Several studies have shown that energy and total food intakes decline with age, making a nutritionally adequate diet more difficult to achieve. Older men consume about 800 kcal less than younger men and older women about 400 kcal less than younger women. A reduction in BMR is partly responsible for this decline, but physical inactivity appears to be the major cause for reduced food intake. Compared with

younger adults, older adults need to reach at least the same levels of intake (and in some cases, higher levels) of most vitamins, minerals and protein. Since this usually needs to be obtained from substantially lower overall food intakes, however, a nutrient and phytochemically dense diet becomes a high priority in later life. In other words, given the tendency for activity levels to decline and total food intakes to fall with advancing years, there is less room for energy-dense foods (e.g. indulgences, treats) which supply few of the essential nutrients that our bodies continue to need. Therefore, older adults need to be selective about what they eat to avoid excessive fat gain and to prefer foods that are nutrient dense and high in protein (e.g. nuts, lean red meat, low-fat dairy products, legumes, seeds). This principle also applies to younger adults who are sedentary. Eating in a traditional food culture context can provide a measure of food security for the aged. This is one of the arguments for Food-based Dietary Guidelines (FBDGs) for the aged. The consumption of nutrient-dense foods reduces the risk of essential nutrient deficiencies. These foods include:

- eggs (little if any effect on serum cholesterol if not eaten with saturated fat)
- liver
- lean meat
- meat alternatives such as legumes (especially traditional soy products, e.g. tofu and tempeh) and nuts
- fish
- low-fat milk and dairy products
- fruits, vegetables, plant shoots
- wholegrain cereals
- wheat germ
- yeast
- unrefined fat from whole foods (nuts, seeds, beans, olives, avocado, fish)
- refined fat from liquid oils (cold pressed, from a variety of sources, predominant in n-3 and/or n-9 fatty acids).

The protective nutritional value of fruits and vegetables is derived particularly from their content of phytochemicals, which are multifunctional compounds, usually of health benefit (antioxidant, antimutagenic, antiangiogenic, immunomodulatory, phytoestrogens). In late 1999, the Australian government, based on the National Health and Medical Research Council report, released a set of dietary guidelines for older Australians (Table 7.6).

Table 7.6 Dietary guidelines for older Australians, 1999

1. Enjoy a wide variety of nutritious foods
2. Keep active to maintain muscle strength and a healthy body weight
3. Eat at least three meals every day
4. Care for your food: prepare and store it correctly
5. Eat plenty of vegetables (including legumes) and fruit
6. Eat plenty of cereals, breads and pastas
7. Eat a diet low in saturated fat
8. Drink adequate amounts of water and/or other fluids
9. If you drink alcohol, limit your intake
10. Choose foods low in salt and use salt sparingly
11. Include foods high in calcium
12. Use added sugars in moderation

Source: National Health and Medical Research Council (1999).
© Commonwealth of Australia. Reproduced with permission.

The FBDGs were also recently published in conjunction with the WHO. They address traditional foods and dishes and most importantly cuisine, making such guidelines more practical and user-friendly at the individual level. These principles will need to be addressed by the various countries around the world when developing their own country/culture-specific FBDGs.

To summarize, the nutritional factors involved in healthy aging include food variety, nutrient and phytochemical density. A 'Mediterranean' food pattern may also reduce the risk of death in older adults. In the frail elderly there should be more emphasis on the need for support and increased nourishment and the prevention of malnutrition. The best and main message for an older person at home is to be well nourished, to be as active as possible without overdoing it, to eat better, not less, to keep their weight up and to drink plenty of fluids every day.

7.10 Perspectives on the future

While maximal lifespan is probably genetically determined, the probability of reaching that lifespan in good health seems largely determined by environmental and lifestyle factors. Thus, if humans are to continue to increase their lifespan and associated quality of life, they will most likely have to make alterations, even if very small, in the way they live their day-to-day life. This may take the form of consuming more fruit and vegetables, reading or taking a daily walk. Owing to decreased energy requirements in old age, diet plays an integral part in maintaining health and vitality. The quality of the diet is critical in ensuring that nutritional needs are met. The diet should consist of nutrient rich, low energy dense foods, which are generally low in fat.

There is not much room for indulgences in an elderly person's diet, especially if they are sedentary. Even though energy requirements are lower, nutrient needs are the same as or higher than those in younger adults. To reduce abdominal obesity and the development of subclinical deficiencies, older adults must choose foods wisely and maintain appropriate physical activity levels.

Further reading

Growth

- Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. Establishing a standard definition for child overweight and obesity worldwide: international survey. *BMJ* 2000; 320: 1240. <http://www.bmj.com/cgi/content/full/320/7244/1240>
- Gracey M, Hetzel B, Smallwood R *et al*. Responsibility for Nutritional Diagnosis: A Report by the Nutrition Working Party of the Social Issues Committee of the Royal Australasian College of Physicians. London: Smith-Gordon, 1989.
- Lee RD, Nieman DC. Nutritional Assessment, 2nd edn. Sydney: McGraw-Hill, 1996.
- National Health and Medical Research Council. The Australian Dietary Guidelines for Children and Adolescents. Canberra: Australian Government Publishing Service, 1995. On-line scientific background papers for each guideline. <http://www.health.gov.au:80/nhmrc/publications/synopses/n1syn.htm>
- Tienboon P, Rutishauser IHE, Wahlqvist ML. Early life factors affecting body mass index and waist-hip ratio in adolescence. *Asia Pacific J Clin Nutr* 1992; 1: 21–6.
- USDA, 2000. http://www.usda.gov/cnpp/dietary_guidelines.htm

Aging

- Campbell AJ, Buchner DM. Unstable disability and the fluctuations of frailty. *Age and Ageing* 1997; 26: 315–318.
- Chapuy MC, Arlot ME, Duboeuf F, Brun J, Crouzet B, Arnaud S, Delmas PD, Meunier PJ. Vitamin D3 and calcium to prevent hip fractures in the elderly women. *N Engl J Med*. 1992 Dec 3; 327 (23): 1637–1642.
- Fiatarone MA, O'Neill EF, Ryan ND, Clements KM, Solares GR, Nelson ME, Roberts SB, Kehayias JJ, Lipsitz LA, Evans WJ. Exercise training and nutritional supplementation for physical frailty in very elderly people. *NEJM* 1995; 330 (25): 1769–1775.
- Glass TA, de Leon CM, Marattoli RA, Berkman LF. Population based study of social and productive activities as predictors of survival among elderly Americans. *BMJ* 1999; 319 (21): 478–483.
- Grundy E. The epidemiology of aging. In: Textbook of Geriatric Medicine and Gerontology, 4th edn (JC Brocklehurst, RC Tallis, HM Fillit, eds), pp. 3–20. New York: Churchill Livingstone, 1992.
- Khaw K-T. Healthy ageing. *BMJ* 1997; 315: 1090–1096. On-line full text: <http://www.bmj.com/cgi/content/full/315/7115/1090>
- National Health and Medical Research Council. The Australian Dietary Guidelines for Older Adults. Canberra: Australian Government Publishing Service, 1999. On-line scientific background papers for each guideline. <http://www.health.gov.au/nhmrc/publications/pdf/n23.pdf>
- Trichopoulou A, Kouris-Blazos A, Wahlqvist ML *et al*. Diet and overall survival in elderly people. *BMJ* 1995; 311: 1457–1460. On-line full text: <http://www.bmj.com/cgi/content/full/311/7018/1457>
- Wahlqvist ML, Kouris-Blazos A. Requirements in maturity and ageing. In: Food and Nutrition: Australia and New Zealand (ML Wahlqvist, ed.). Sydney: Allen & Unwin, 2002. <http://www.healthyeatingclub.com/bookstore/>

Nutrition and Metabolism

Edited on behalf of The Nutrition Society by

Michael J. Gibney, Ian A. Macdonald
and Helen M. Roche

Blackwell
Science



© The Nutrition Society 2003

Published by Blackwell Science
a Blackwell Publishing company
Editorial offices:

Blackwell Science Ltd, 9600 Garsington Road,
Oxford OX4 2DQ, UK

Tel: +44 (0) 1865 776868

Iowa State Press, a Blackwell Publishing Company,
2121 State Avenue, Ames, Iowa 50014-8300, USA

Tel: +1 515 292 0140

Blackwell Science Asia Pty, 550 Swanston Street, Carlton, Victoria 3053, Australia

Tel: +61 (0)3 8359 1011

The right of the Author to be identified as the Author of this Work has been asserted in accordance with the Copyright, Designs and Patents Act 1988.

All rights reserved. No part of this publication may be reproduced, stored in a retrieval system, or transmitted, in any form or by any means, electronic, mechanical, photocopying, recording or otherwise, except as permitted by the UK Copyright, Designs and Patents Act 1988, without the prior permission of the publisher.

First published 2003

Library of Congress

Cataloging-in-Publication Data

Nutrition and metabolism / edited on behalf of the

Nutrition Society by Michael J. Gibney, Ian MacDonald, and Helen M. Roche. – 1st ed.

p. cm.

ISBN 0-632-05625-8 (pbk. : alk. paper)

1. Nutrition. 2. Metabolism. I. Gibney, Michael J. II. Macdonald, Ian, 1921–

III. Roche, Helen M. IV. Nutrition Society (Great Britain)

QP141.N7768 2003

612.3–dc22

2003015190

ISBN 0-632-05625-8

A catalogue record for this title is available from the British Library

Set in Minion by Gray Publishing, Tunbridge Wells, Kent
Printed and bound in Great Britain using acid-free paper
by Ashford Colour Press, Gosport

For further information on Blackwell Publishing, visit our website:

www.blackwellpublishing.com

Contents

Series Foreword	xi
Preface	xii
Contributors	xiii
1. Core Concepts of Nutrition	1
<i>IA Macdonald and MJ Gibney</i>	
1.1 Introduction	1
1.2 Balance	1
1.3 Turnover	2
1.4 Flux	3
1.5 Metabolic pools	3
1.6 Adaptation to altered nutrient supply	5
1.7 Perspectives on the future	5
Further reading	5
2. Molecular Aspects of Nutrition	6
<i>HM Roche and RP Mensink</i>	
2.1 Introduction	6
2.2 Core concepts in molecular biology	7
2.3 Gene expression: transcription and translation	12
2.4 Research tools to investigate molecular aspects of nutrition	17
2.5 Effects of the genetic code on the response to nutrients	22
2.6 Nutrient regulation of gene expression	25
2.7 Perspectives on the future	28
Further reading	29
3. Integration of Metabolism 1: Energy	30
<i>XM Leveille</i>	
3.1 Introduction	30
3.2 Energy metabolism at the cellular level	31
3.3 Energy metabolism in the body as a whole	39
3.4 Perspectives on the future	42
Notes	42
Further reading	42
4. Integration of Metabolism 2: Protein and Amino Acids	43
<i>JT Brosnan and VR Young</i>	
4.1 Introduction	43
4.2 Protein and amino acid turnover	44
4.3 Protein synthesis	45
4.4 Regulation of the translation phase of protein synthesis	47
4.5 Post-translational events	48
4.6 Protein degradation	49
4.7 Selectivity of protein turnover	50

4.8	An integration of these processes of turnover with respect to amino acid metabolism	52
4.9	Regulation of amino acid metabolism	52
4.10	Amino acid synthesis: the dispensable amino acids	54
4.11	<i>In vivo</i> aspects of protein and amino acid turnover	55
4.12	<i>In vivo</i> rates of protein turnover	57
4.13	Mechanisms and factors responsible for alterations in protein turnover	59
4.14	Interorgan amino acid metabolism	61
4.15	Amino acid and peptide transport	62
4.16	Disposal of dietary amino acids and roles of specific organs	65
4.17	Catabolic illnesses	68
4.18	Non-proteinogenic metabolic functions of amino acids	69
4.19	Perspectives on the future	72
	Further reading	73
5.	Integration of Metabolism 3: Macronutrients	74
	<i>KN Frayn and AO Akanji</i>	
5.1	Introduction: fuel intake and fuel utilization	74
5.2	Regulatory mechanisms	76
5.3	Hormones that regulate macronutrient metabolism	77
5.4	Macronutrient metabolism in the major organs and tissues	82
5.5	Substrate fluxes in the overnight fasting state	87
5.6	Postprandial substrate disposal	91
5.7	Short-term and longer term starvation	93
5.8	Perspectives on the future	95
	Further reading	95
6.	Pregnancy and Lactation	96
	<i>JMA van Raaij and CPGM de Groot</i>	
	Section I: Pregnancy	97
6.1	Introduction	97
6.2	Physiological stages of pregnancy and their nutritional demands	97
6.3	Principles for estimating nutritional needs in pregnancy	98
6.4	Physiological adjustments that may effect energy and nutrient needs of pregnancy	100
6.5	Ways in which mothers may deal with the energy and nutrient needs of pregnancy	102
6.6	Dietary recommendations for pregnancy	103
6.7	Perspectives on the future	105
	Section II: Lactation	105
6.8	Regulation of milk production	105
6.9	Colostrum, transitional and mature milk	106
6.10	Protective aspects of human milk	106
6.11	Maternal nutrition and lactational performance	107
6.12	Recommended intakes during lactation	108
6.13	Energy and nutrient inadequacies	111
6.14	Perspectives on the future	111
	Further reading	111

7.	Growth and Aging	112
	<i>ML Wahlqvist, A Kouris-Blazos, KA Ross, TL Setter and P Tienboon</i>	
7.1	Introduction	112
7.2	Growth and development	112
7.3	Nutritional factors affecting growth	116
7.4	Nutrition and the life cycle	118
7.5	Effects of undernutrition	119
7.6	Effects of overnutrition	121
7.7	Growth during childhood and adolescence	123
7.8	Aging	126
7.9	Guidelines for healthy aging	142
7.10	Perspectives on the future	144
	Further reading	144
8.	Nutrition and the Brain	145
	<i>JD Fernstrom and MH Fernstrom</i>	
8.1	Introduction	145
8.2	General organization of the mammalian nervous system	146
8.3	The blood-brain barrier	148
8.4	Energy substrates	150
8.5	Amino acids and protein	151
8.6	Vitamins and minerals	159
8.7	Perspectives on the future	166
	Further reading	166
9.	The Sensory Systems: Taste, Smell, Chemesthesis and Vision	168
	<i>CM Delahunty and TAB Sanders</i>	
9.1	Introduction to taste, smell and chemesthesis	168
9.2	The taste system	169
9.3	The olfactory system	172
9.4	Chemesthesis	174
9.5	Role of saliva	176
9.6	Adaptation	176
9.7	Cross-modal sensory interactions	177
9.8	Flavor preferences	178
9.9	Changing function of the chemical senses across the lifespan	180
9.10	Introduction to the visual system	182
9.11	Outline of the physiology of the visual system	182
9.12	Role of the retina in signal transduction and the specific roles of retinol, docosahexaenoic acid and taurine in the visual process	183
9.13	Evidence for a specific requirement for docosahexaenoic acid in the visual process	184
9.14	Specific problems associated with the visual process	185
9.15	Cataract	185
9.16	Age-related macular degeneration	187
9.17	Epidemic optic nerve neuropathy	187
9.18	Perspectives on the future	187
	Further reading	189

10.	The Gastrointestinal Tract	190
	<i>M Mañas, E Martínez de Victoria, A Gil, M Yago and J Mathers</i>	
10.1	Introduction	190
10.2	Structure and function of the gastrointestinal system	190
10.3	Motility	193
10.4	Secretion	195
10.5	Digestion and absorption	196
10.6	Water balance in the gastrointestinal tract	198
10.7	The exocrine pancreas	199
10.8	Diet and exocrine pancreatic function	202
10.9	Interactions between the endocrine and exocrine pancreas	206
10.10	Physiology of bile secretion and enterohepatic circulation	207
10.11	Adaptation of the biliary response to the diet	210
10.12	Growth, development and differentiation of the gastrointestinal tract	211
10.13	The large bowel	217
10.14	Perspectives on the future	222
	Further reading	223
11.	The Cardiovascular System	224
	<i>G Riccardi, A Rivellese and C Williams</i>	
11.1	Introduction	224
11.2	Factors involved in a healthy vascular system	225
11.3	Pathogenesis of cardiovascular disease	233
11.4	Risk factors for cardiovascular disease	234
11.5	Dietary components and their effect on plasma lipids	237
11.6	Diet and blood pressure	240
11.7	Effects of dietary factors on coagulation and fibrinolysis	242
11.8	Homocysteine	242
11.9	Diet and antioxidant function	243
11.10	Insulin sensitivity	244
11.11	Perspectives on the future	245
	Further reading	246
12.	The Skeletal System	247
	<i>JM Pettifor, A Prentice and P Cleaton-Jones</i>	
12.1	Introduction	247
12.2	Bone architecture and physiology	247
12.3	Bone growth	254
12.4	Teeth	260
12.5	Nutritional rickets	265
12.6	Bone loss with aging	274
12.7	Specific nutrients and their effects on bone health	277
12.8	Lifestyle factors and bone health	281
12.9	Perspectives on the future	283
	Further reading	283

13.	The Immune and Inflammatory Systems	284
	<i>P Yaqoob and PC Calder</i>	
13.1	Introduction	284
13.2	The immune system	285
13.3	Why should nutrients affect immune function?	290
13.4	Assessment of the effect of nutrition on immune function	290
13.5	Impact of infection on nutrient status	291
13.6	Malnutrition and immune function	292
13.7	The influence of individual micronutrients on immune function	293
13.8	Dietary fat and immune function	297
13.9	Dietary amino acids and related compounds and immune function	302
13.10	Probiotics and immune function	303
13.11	Breast-feeding and immune function	304
13.12	Perspectives on the future	304
	Further reading	306
14.	Phytochemicals	307
	<i>A Cassidy and FS Dalais</i>	
14.1	Introduction	307
14.2	Historical perspective	307
14.3	The phenolic phytochemicals	308
14.4	Carotenoids	314
14.5	Phytosterols	314
14.6	Sulfur-containing compounds	316
14.7	Phytochemical toxicity	317
14.8	Perspectives on the future	317
	Further reading	317
15.	The Control of Food Intake	318
	<i>A Drewnowski and F Bellisle</i>	
15.1	Introduction	318
15.2	Theories of control	318
15.3	The neuroscience of food intake	319
15.4	Motivational states	320
15.5	The preload paradigm	321
15.6	The role of energy density	321
15.7	Psychosocial factors	322
15.8	Implications for aging and body-weight management	322
15.9	Perspectives on the future	323
	Further reading	323
16.	Overnutrition	324
	<i>L Bandini and A Flynn</i>	
	Section I: Obesity	324
16.1	Introduction	324
16.2	Identification	324
16.3	Skinfold thickness	325

16.4	Body mass index	325
16.5	Energy balance	326
16.6	Etiology	328
16.7	Pathological syndromes	331
16.8	Consequences of obesity	332
16.9	Perspectives on the future	333
Section II: Vitamin and mineral overconsumption		333
16.10	Introduction	333
16.11	Adverse effects of vitamins and minerals: concepts	333
16.12	Derivation of the tolerable upper intake level	335
16.13	Use of tolerable upper intake levels as dietary reference standards	339
16.14	Perspectives on the future	340
	Further reading	340
17.	Undernutrition	341
	<i>M Vaz</i>	
17.1	Introduction	341
17.2	Definition and classification of undernutrition	341
17.3	Adaptation and chronic energy deficiency	343
17.4	Changes in body composition in chronic energy deficiency	343
17.5	Energy metabolism in chronic energy deficiency	343
17.6	Regulatory processes in chronic energy deficiency	346
17.7	Functional consequences of energy deficiency	346
17.8	Perspectives on the future	347
	Further reading	348
18.	Exercise Performance	349
	<i>AE Jeukendrup and LM Burke</i>	
18.1	Introduction	350
18.2	Energy expenditure during physical activity	350
18.3	Carbohydrate and performance	353
18.4	Fat metabolism and performance	358
18.5	Effect of exercise on protein requirements	360
18.6	Physique and sports performance	361
18.7	Weight maintenance and other body-weight issues	362
18.8	Vitamins and minerals	364
18.9	Fluid and electrolyte loss and replacement in exercise	366
18.10	Nutritional ergogenics	372
18.11	Dietary supplements and failed drug tests	376
18.12	Practical issues in nutrition for athletes	377
18.13	Perspectives on the future	377
	Further reading	378
Index		379