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**Authorship and publication history**

Attribution of authorship is provided with each part of this work.

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List of appendices on DSO

Readings
TOPIC 1

Nutritional assessment

PREPARED BY THE UNIT TEAM

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Introduction

Assessment of nutritional status is an assessment of the state of health of an individual or population relative to intake and utilisation of food. Nutritional assessment is made following the interpretation of clinical, anthropometric and biochemical data.

Why do we assess nutritional status? In the individual the objective is usually to establish the nature and aetiology of the problem in order to provide the appropriate treatment and to prevent reoccurrence of the problem. In the population the objective is usually to establish the extent and distribution of the problem in the community, and to identify the associated environmental factors in order not only to provide appropriate therapeutic facilities, but also to institute preventive programs.

Learning objectives

At the conclusion of this section you should have an understanding of and be able to describe:

- nutritional assessment
- techniques used to assess nutritional status
- methods used to evaluate dietary intake data
- the importance of integrating information from different methods of assessment
- the use of Recommended Dietary Intakes (RDI) to evaluate nutritional risk.

Learning resources

Deakin Studies Online (DSO)

The following appendices are referred to throughout this unit. You can access them and other additional resources to supplement this unit on DSO <http://www.deakin.edu.au/current-students/>.
Appendix 1.1

Birth to 36 months
Boys, length-for-age and weight-for-age percentiles

Appendix 1.2

Birth to 36 months
Girls, length-for-age and weight-for-age percentiles

Appendix 1.3

2 to 20 years: Boys
Stature-for-age and weight-for-age percentiles

Appendix 1.4

2 to 20 years: Girls
Stature-for-age and weight-for-age percentiles

Appendix 1.5

Body mass index-for-age percentiles:
Boys, 2 to 20 years

Appendix 1.6

Body mass index-for-age percentiles:
Girls, 2 to 20 years

The above six documents are from:


Appendix 2

Nutrient Reference Values for Australia and New Zealand – executive summary

Appendix 3

National Nutrition Survey 1995 – summary of findings

Appendix 4

National Nutrition Survey 1995 – tables
Appendix 5


Online reading

Access the reading listed here using your internet browser.


Methods used in nutritional assessment

Assessment of dietary intake

Nutritionists are most interested in the assessment of dietary intake. The following discussion explores in further detail dietary assessment methodology.

Individual dietary intake

Food or dietary assessments can be divided into two categories; those involving recall of the client’s usual intake and those involving recording of actual intake. There is no one generally accepted method and all methods have their advantages and limitations. Methods are chosen based on how precise and valid the results need to be; that is, whether they are required for clinical practice or research.

1 Food recall methods

(a) Twenty-four hour food recall. This involves a recall of food and fluid intake over a specified period of time (e.g. the previous 24 hours). The information may be collected by interview or self-administered questionnaire. The advantages of a 24-hour food recall include short interview length and only short-term memory recall is required by the individual. The major limitation of this method is that information on ‘usual intake’ is not obtained because a 24-hour time period does not show variation over days or weeks. This type of recall is useful in an individual situation to establish a recent food pattern or in research situations where the mean intake of a group is required.

(b) Diet history. This is an interview to determine the usual food and fluid pattern of an individual over the previous two to three month period. The history involves a recall of the usual eating pattern and a crosscheck in the form of a detailed list of foods. Diet histories are frequently used with adults, because the usual eating pattern is established and can be used as a basis for dietary modification. A diet history is a useful measure of an individual’s usual intake but the method is time consuming, difficult to standardise, relies on the memory of the individual, and is believed by some to have insufficient quantitative value.
(c) Food frequency questionnaire. This involves a self-administered questionnaire to ascertain how frequently common foods are consumed. The questionnaire is limited to selected foods and food quantity may be considered. Food frequency questionnaires can be a standardised method, cheap to administer and do not require highly trained personnel. Limitations of the questionnaires include the limited number of foods included, limited information regarding quantity of foods consumed and the reliability of the respondent. Food frequency questionnaires are generally conducted in a research situation to ascertain the mean nutrient intake of a group.

2 Food records

This involves clients recording current intake over a period of one to seven days. The food intake may be recorded based on weights, household measures or general description.

A weighed record can provide an accurate description of food consumed over a period of time but is time consuming and requires cooperation of the individual. Weighed records are generally only used in research. Records in household measures are less time consuming than weighed records for the respondent and may reflect their usual intake more closely. Because the measurements of food are less precise than weighing, the results may not be as accurate.

Records which only describe the types of food eaten do not provide any quantitative information but they require less time than the above methods and can be carried out over longer periods. Food recording as a method suffers the major limitation of showing only the actual intake which may be different from the usual intake. Because individuals are recording their intake for a purpose they may eat differently.

Dietary intakes of populations

The 1995 National Nutrition Survey (NNS) is the most recent dietary survey on the nutrient intake of Australian adults (ABS 1997; ABS 1998). In 2007, the Children’s Nutrition and Physical Activity Survey (CNPAS) was completed and provides the most recent data on 2- to 16-year-old Australians. The NNS was conducted between February 1995 and March 1996 on people aged two or more years and provides intake data on energy, moisture, macronutrients (protein; fat – total, saturated, monounsaturated, polyunsaturated; cholesterol; carbohydrate – total, starch and sugars; dietary fibre; alcohol) vitamins (vitamin A, thiamin, riboflavin, niacin equivalents, folate, vitamin C) and minerals (calcium, phosphorus, magnesium, iron, zinc, potassium). The CNPAS was conducted between February and August of 2007 and collected dietary, anthropometric and physical activity data from 4487 children, these data supersede the NNS data for under 16-year-olds and will be referred to throughout this unit.

In both national surveys, daily food consumption data were collected by a 24-hour recall, as well as food related habits and attitudes, and physical measurements. These data will be referred to in the remaining topics in this study guide. A summary of the findings of both surveys are included in your appendices located on DSO. In 2008, the Australian Government announced its intention to fund an
ongoing National Nutrition and Physical Activity Survey Program. This survey is
to take place approximately every 5 to 6 years and include adults, children and
Indigenous populations. Usual food consumption will be derived from two 24-hour
recalls and respondents will also be asked questions about dietary habits and food
security. Anthropometric measurements (including blood pressure) and physical
activity will be measured and participants will have the option of providing fasting
blood and urine samples (children aged 5–12 years will be asked for urine samples
only). The first survey will be conducted from 2011 to 2013, with first results
released at the end of 2012. Further information can be accessed on the Department
(retrieved 9 March 2012) and the Australian Bureau of Statistics website

ONGOING EXERCISE

As you read through journal articles while studying this unit, scrutinise the
technique used to measure dietary intake. Was it the most accurate method
used? What are advantages and disadvantages of the methodology used?

Evaluating dietary intake data

Once a dietary assessment has been conducted it needs to be translated into nutrient
terms or analysed typically via country specific nutrition analysis programs. When
using nutrient databases it is important to be aware of sources of error, limitations
of their use, and the often crude estimation of nutrients in foods.

Reference standards

Once the dietary nutrients have been determined, they are generally compared to
some form of reference standard. Standards for comparison may include:

(a) *Nutrient Reference Values for Australia and New Zealand – including
Recommended Dietary Intakes*
(retrieved 9 March 2012).

(b) *Australian Dietary Guidelines— incorporating the Australian guide to healthy
eating* [draft for public consultation] (NHMRC, 2011)
<http://consultations.nhmrc.gov.au/open_public_consultations/dietary-
guidelines> (retrieved 9 March 2012).

(c) *Australian Guide to Healthy Eating* [draft for public consultation] (NHMRC,
guidelines> (retrieved 9 March 2012).

(d) Health organisation recommendations; for example, the National Heart
Foundation.

Dietary guidelines for Australians

Dietary guidelines are an essential tool to support broader strategies to improve
nutrition outcomes in Australia. These guidelines focus more on food groups and
lifestyle patterns rather than specific nutrients. The Dietary Guidelines for Australians have recently been reviewed and at the time of printing this study guide, are only available for public consultation in draft form. A draft copy of the guidelines can be accessed from the National Health and Medical Research Council web site:

Recommended Dietary Intakes (RDIs)
The first known dietary recommended intake was probably recorded during 1795 when the British Admiralty recommended one ounce of lemon juice daily to prevent scurvy in their sailors. This is an example of how the daily intake of a food could help prevent a deficiency disease.

With the increase in knowledge about food constituents and their physiological function, as well as the availability of analytical methods to measure the small amounts of many nutrients in the human body and food, current recommendations are now defined in terms of nutrients rather than food.

The first Australian recommended dietary intakes were published by the National Health and Medical Research Council (NHMRC) in 1954. These early values included those for energy and eight nutrients. They went through a number of revisions and in 2006 new Nutrient Reference Values for Australia and New Zealand were endorsed and released by the Australian NHMRC and the New Zealand Ministry of Health. The executive summary is available as Appendix 2 on DSO or on the NHMRC web site <http://www.nhmrc.gov.au/guidelines/publications/n35-n36-n37> (retrieved 9 March 2012).

Nutrient Reference Values (NRVs)
The Nutrient Reference Values (incorporating the former RDIs) supersede the previous RDIs for Australians. There are a number of new classifications, as well as recommendations including a wider range of nutrients; the recommendations are more in line with official recommended dietary allowances in the United States.

The NRVs are guidelines to the intake of a range of essential macro and micronutrients directed towards the maintenance of good health. This is in contrast to earlier recommendations, which were developed primarily to prevent deficiency diseases in those at risk.

NRVs retain the concept of RDIs, but also attempt to identify the average requirements needed by individuals. For most nutrients an Estimated Average Requirement (EAR) was set from which an RDI could be derived.

It is not always possible to determine the EAR as the evidence may be insufficient or too conflicting to substantiate it. In these cases an Adequate Intake (AI) was set which in effect becomes the RDI. AIs have been set either on experimental evidence or by adopting the current population median intake, assuming that Australian/New Zealand populations were not deficient for that particular nutrient. AIs were based on median population means from the National Nutrition Survey of Australia and New Zealand (see Appendices 3 and 4).
Both the RDI and AI can be used as a goal for individual intake, but there is less certainty about the AI value as it depends on a greater degree of judgment. Therefore, AI should be used with great care.

For each nutrient, an Upper Intake Level (UIL) (including intake from both food and supplements) was also set. This is the highest level of a nutrient that is likely to pose no adverse risk to health in almost all individuals in the specified life stage group. The UIL is not supposed to be the recommended level of intake. The UIL is based on a risk assessment of nutrients which involves establishment of a No Adverse Effect Level (NOAEL) and/or a Lowest Adverse Effect Level (LOAEL), and application of an Uncertainty Factor (UF).

For individuals the EAR can be used to examine the probability that their usual intake is inadequate and the RDI or AI can be used to indicate the intake at which or above which there is a reduced probability of inadequacy. Intakes about the UIL may place an individual at risk of adverse effects from excessive nutrient intake.

**Relationship of NRVs to other public health nutrition tools**

The dietary guidelines have a focus on the prevention of chronic disease, have been designed to be used by the public and health professionals, and are often qualitative in nature. Dietary guidelines focus on food rather than nutrients.

Food selection guides are often based on RDIs and assist the general population in making healthy food choices. When considering nutritional requirements from the consumer’s point of view, health messages are more easily understood when expressed in terms of foods rather than nutrients. In the development of the draft 2011 Australian Dietary Guidelines and Australian Guide to Healthy Eating, a dietary modelling system was used to translate the 2006 NRVs to practical diet recommendations (Dietitians Association of Australia 2011). This modelling system replaced the Core Food Groups (1994) and involved creating theoretical food patterns that met, or came as close as possible to meeting, the 2006 NRVs for 10 key nutrients (protein, thiamin, vitamin A (as retinol equivalents), vitamin C, folate, calcium, iodine, iron, magnesium and zinc) for males and females across the lifespan with varying body size and activity level.

Using computer programmed modelling incorporating food consumption data from the 1995 National Nutrition Survey and the 2007 Australian National Children’s Nutrition and Physical Activity Survey, ‘foundation diets’ were created comprising of ‘composite foods’ that represented food groups: vegetables and legumes; fruits; grain foods; lean meat, poultry, fish, eggs, nuts, seeds, and legumes; milk, yogurt, cheese and alternatives. Foundation diets were designed to provide the estimated energy requirements and meet the RDIs of the above 10 key nutrients for individuals requiring the least energy in each age and gender group (i.e. the shortest and most sedentary individuals). These diet patterns were evaluated using computer simulation where 7-day diets were generated using actual foods (rather than composite foods) to replicate possible real life scenarios. Diet patterns were generally deemed acceptable if 100 7-day diets following the pattern met the EARs of the 10 key nutrients. ‘Total diets’ were then created based on acceptable foundation diets to meet the energy requirements of taller and more active individuals, and these diets were also evaluated with computer simulation of 100 7-day diets incorporating real foods. In the final evaluation of foundation and total diets, EARs of all 10 key nutrients were met across all age and gender groups.
except for the EARs of iron in pregnant women and in pregnant adolescents, which were too high to meet while complying with dietary energy limits.

The resulting dietary models demonstrate the amounts and types of foods needed by a range of Australians to meet their nutritional needs, and along with a commissioned literature review (evidence report) and input from stakeholders, were used as evidence for the dietary guidelines (Dietitians Association of Australia 2011). Details of the dietary modelling and the evidence report can be found on the NHMRC website <http://consultations.nhmrc.gov.au/open_public_consultations/dietary-guidelines> (retrieved 9 March 2012).

### Table 1.1 Definitions

<table>
<thead>
<tr>
<th>Definitions</th>
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<tbody>
<tr>
<td><strong>EAR</strong></td>
<td>Estimated Average Requirement</td>
</tr>
<tr>
<td>EAR is the median usual intake level estimated to meet the requirements of half the health individuals in a life stage/gender group.</td>
<td></td>
</tr>
<tr>
<td><strong>RDI</strong></td>
<td>Recommended Dietary Intake</td>
</tr>
<tr>
<td>RDI is the average dietary intake level sufficient to meet the nutrient requirements of nearly all healthy individuals (97–98%) in a life stage/gender group. If the requirement for the nutrient is normally distributed and the standard deviation (SD) of the EAR is available, RDI = EAR + 2 SDEAR. If the data about variability are insufficient to calculate a SD, a coefficient of variation of 10% is assumed and used to estimate SD (SD = 10). Therefore RDI = 1.2 × EAR (EAR + 2 (10) / EAR).</td>
<td></td>
</tr>
<tr>
<td><strong>AI</strong></td>
<td>Adequate Intake</td>
</tr>
<tr>
<td>Where an EAR (and therefore an RDI) for the nutrient cannot be determined because of limited or inconsistent data then an Adequate Intake (AI) is determined. AI can be used as a goal for individual intake but is based on observed or experimentally-determined approximation or estimates of nutrient intake by a group (or groups) of apparently healthy people that are assumed to be adequate.</td>
<td></td>
</tr>
<tr>
<td><strong>EER</strong></td>
<td>Estimated Energy Requirement</td>
</tr>
<tr>
<td>EER is the average dietary energy intake that is predicted to maintain energy balance in a healthy adult of defined age, gender, weight, height and level of physical activity, consistent with good health. In children and pregnant and lactating women, the EER includes needs associated with the deposition of tissues or the secretion or milk consistent with good health.</td>
<td></td>
</tr>
<tr>
<td><strong>UIL</strong></td>
<td>Upper Intake Limit</td>
</tr>
<tr>
<td>UIL is the highest level of continuing daily nutrient intake likely to pose no adverse health effects in almost all individuals in the general population.</td>
<td></td>
</tr>
</tbody>
</table>

(NHMRC, 2006)
Integrating information on nutritional status

Dietary data is only one component of nutrition assessment. Full assessment of either an individual or populations nutrition status can only be achieved after collating information from dietary, clinical, anthropometric and biochemical data. After completing these different components of nutritional assessment, the likelihood of a nutrient deficiency being present can be determined. The example of iron deficiency anaemia can be used to illustrate this point.

1 Dietary assessment: Analysis of an individual’s intake by the method of a dietary history may reveal a long term history of insufficient intake of dietary iron.

2 Biochemical assessment: Analysis of serum ferritin levels indicates a level below the reference range.

3 Clinical assessment: A physical observation reveals pale eye membranes and the individual reports they are experiencing chronic fatigue.

4 Anthropometric: Analysis of growth rate. A reduction in the rate of growth may be indicative of low iron status in young children.

It is important to look at all components of a nutritional assessment as opposed to each one individually in order to rule out other reasons for the presenting condition. For example, pale eye membranes may be indicative of another medical illness unrelated to iron deficiency anaemia.

TEXT QUESTIONS

1 Record your food and fluid intake for one day (refer to Table 1.2) and analyse your intake of macro- and micronutrients using Foodworks, a nutrient analysis program that can be downloaded free of charge for a trial period <http://www.xyris.com.au/>, retrieved 9 March 2012. If you an on-campus student you can access some computers which have the Foodworks program loaded. Details of which computer labs have Foodworks loaded will be provided within lectures or on DSO. Enter your data from Foodworks into Table 1.3.

(a) How did your intake of macro- and micronutrients compare to the NRVs for your own age and sex? (See Appendix 2 for NRVs.)

(b) Were there any nutrients that were below the RDI or AI? If a nutrient was below the RDI, how did it compare to the EAR?

(c) Using a calculator estimate the per cent energy from the macronutrients and alcohol in your diet. NB: Protein provides 17 kJ/g, carbohydrate 16 kJ/g, fat 37 kJ/g and alcohol 29 kJ/g. NB: Does your per cent total energy from macronutrients equal 100%. If not, why not?

(d) What was your per cent energy from protein, fat, carbohydrate and alcohol? How does this compare to the NRV’s and usual intakes from the 1995 National Nutrition Survey for your age group? (See Appendix 3 for 1995 National Nutrition Survey Summary of findings.)
(e) What practical difficulties, if any, did you experience while recording and entering your food intake data? How would these affect the quality of your nutrient intake data?

2 For each of the food recall methods and the food record method, consider how appropriate each would be for different groups within the population. What issues would each method present for young children, adolescents, people with low literacy skills, non-English speaking groups, the aged population or people with disabilities? How would these factors impact the quality of nutrient intake data from these population groups?

Table 1.2 Record your food and fluid intake for one day

Instructions:
- Be as specific as possible with brand names and amounts
- Please write every different food / drink on a new line.

Example one day record

<table>
<thead>
<tr>
<th>Meal</th>
<th>Time</th>
<th>Food/drink</th>
<th>Quantity</th>
</tr>
</thead>
<tbody>
<tr>
<td>B/F</td>
<td>7am</td>
<td>Kelloggs Cornflakes</td>
<td>1 cup</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Milk – Pura light start</td>
<td>½ cup</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sugar – white, table sugar</td>
<td>2 tsp</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Tea – tea bag, weak</td>
<td>200 ml</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Milk – Skinny milk</td>
<td>2 tblsp</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Sugar – brown</td>
<td>1 tsp</td>
</tr>
</tbody>
</table>
Day and date recorded ________________________  ___/___/_____

<table>
<thead>
<tr>
<th>Meal</th>
<th>Time</th>
<th>Food/drink</th>
<th>Quantity</th>
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</tbody>
</table>
Table 1.3  Your daily macro- and micronutrient intake

<table>
<thead>
<tr>
<th></th>
<th>Energy (kJ)</th>
<th>Protein (g)</th>
<th>Fat (g)</th>
<th>Carb (g)</th>
<th>Fibre (g)</th>
<th>Folate (ug)</th>
<th>Fe (mg)</th>
<th>Ca (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Totals</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>2006 RDI or Al</td>
<td></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Your % of 2006 RDI</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<td></td>
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<tr>
<td>2006 EAR (if available)</td>
<td></td>
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<td>Your % of 2006 EAR</td>
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References


National Health and Medical Research Council 1994, *The core food groups*, AGPS, Canberra.


TOPIC 2

Nutrition during foetal life

PREPARED BY THE UNIT TEAM

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Introduction

Gestation is the period during which nutrient status has the potential to have the greatest impact on both organ and tissue development during foetal development and also on future growth and development. The nutrient requirements for foetal growth are often discussed in the context of pregnancy. In this unit, however, we have chosen to discuss separately the nutritional needs of the foetus and those of the mother, because while they are clearly linked they are also quite obviously different.

Learning objectives

At the conclusion of this section you should have an understanding of and be able to describe:

• the various stages of foetal growth and development
• the role of the placenta in foetal development
• the changes in body composition of the foetus during gestation
• the amounts and types of nutrients required by the foetus for growth and metabolism
• the impact of maternal nutrition
• how birth weight can influence morbidity and mortality
• the effects of alcohol, smoking and caffeine on foetal development.

Learning resources

Print reading

The reading listed here is reproduced in the print readings for this unit.


Deakin Studies Online (DSO)

Access the unit site on DSO <http://www.deakin.edu.au/current-students/> for the appendices and other additional resources you can use to supplement this topic.
Periods of prenatal and infant life

Stages of foetal growth

Conception, or successful fertilisation of the ovum, usually occurs in the fallopian tube within 48 hours of ovulation and is followed by rapid cell division. The resulting mass of cells enters the uterus where it undergoes reorganisation to a hollow ball of cells known as the blastocyst which attaches itself to the endometrial lining of the uterus within five to seven days after ovulation. Once attached to the endometrial lining, the outer cells of the blastocyst begin to arrange themselves into a functioning placental unit which grows rapidly throughout gestation in order to support the increasing requirements of the foetus for oxygen and nutrients. The periods of foetal growth and development are illustrated in Figure 2.2 and described below.

Blastogenesis

This stage of foetal growth continues for about two weeks after fertilisation. During this time the cells resulting from the fertilised ovum separate into two distinct layers of cells: an inner cell mass which gives rise to the embryo and an outer layer of cells, the trophoblast, which becomes the placenta.

Embryonic stage

The embryonic stage of foetal development, which is complete by 60 days gestation, is the time during which the basic structures for all of the major organs and tissues develop. It is often described as the ‘critical’ stage of development since it is during this time that the inner cells of the blastocyst differentiate into the three germinal layers which in turn develop into the major organ systems.

The ectoderm, or outer layer, gives rise to the brain, nervous system, hair and skin. The mesoderm, or middle layer, develops into all of the voluntary muscles, bones and some components of the cardiovascular and excretory systems and the endoderm, or inner layer, forms the digestive and respiratory systems and all of the glandular organs. Based on measurements of DNA and protein, growth during the embryonic stage occurs by an increase in the number of cells (hyperplasia).
Foetal stage

The foetal stage of gestation is the period of most rapid growth. Between 13 and 40 weeks of gestation, the weight of the foetus increases nearly 500-fold, from around 6 g to 3000–3500 g at birth. While cell division continues until 34–36 weeks of gestation, growth now also occurs by an increase in cell size (hypertrophy). After 34–36 weeks of gestation, growth in foetal size is solely due to hypertrophy.

From this sequence of events, it is possible to assess the likely effects of energy and nutrient restriction during different periods of foetal growth. During the first two stages of foetal growth (blastogenesis and embryonic stage) the size of the foetus and therefore the amount of energy and nutrients needed to support foetal development are extremely small. Nevertheless, an interruption to, or a restriction in, the supply or transport of nutrients to the foetus during this period has the potential to result in abnormal tissue or organ development (malformations). In contrast, the restricted availability of energy and/or nutrients after the third month of gestation will not have any teratogenic effects, but could interfere with foetal growth particularly during the last three months of gestation.

Measurements of DNA and protein in embryonic and foetal tissues show that embryonic growth occurs only by an increase in number of cells (hyperplasia). Foetal growth continues in cell number but now also involves an increase in cell size (hypertrophy).

Growth-retarded infants

From the sequence described it is possible to estimate the effects of malnutrition on growth at different stages of gestation. In the early months of pregnancy, a severe limit on the supply of nutrients would have to occur to cause retarded growth, because the quantitative requirements of the embryo are extremely small. Nevertheless, a restriction of nutrients and energy needed for cell synthesis and cell differentiation could produce malformations or cause the embryo to die.

Malnutrition after the third month of gestation would not have teratogenic effects, but it could interfere with foetal growth. Nutrient requirements are the greatest in the last trimester of pregnancy, when cells are increasing rapidly in both number and size. Even a relatively mild restriction could have serious effects at this time.
Figure 2.2a Stages of foetal development

(Worthington-Roberts & Williams 1996, pp. 111–12)
Figure 2.2b  Stages of foetal development (cont.)

(Worthington-Roberts & Williams 1996, pp. 111–12)
The role of the placenta

The placenta is a complex network of tissue and blood vessels designed to provide the efficient transfer of oxygen and nutrients from the maternal circulation to the foetal circulation, and for the efficient disposal of metabolic waste products from the foetal to the maternal circulation. A well-developed and fully functioning placenta is thus crucial for an adequate supply of oxygen and nutrients for foetal growth and development. The placenta also plays an important role in the regulation of maternal growth and development during pregnancy, which will be discussed in the context of maternal nutrition in the pregnancy module.

Structure

The structure of the placenta is shown in Figure 2.3. The placenta develops from the outer layer of the blastocyst (known as the trophoblast) which embeds itself in the wall of the uterus during the first six to seven days after fertilisation. Following implantation, the uterine tissue and blood vessels break down to form small spaces (lacunae) which fill with maternal blood. At the same time, the trophoblast sends out finger-like villi into the lacunae in the uterine wall. The villi contain capillaries through which the exchange of nutrients and metabolic waste occurs. As the pregnancy progresses the villi become both thinner and more branched so that the total surface area over which exchange of materials can take place increases enormously. While the foetal and maternal tissues are closely intermingled within the structure of the placenta, the maternal and foetal circulations are always separated by the placental membrane (Figure 2.3).

(Worthington-Roberts & Williams 1996, p. 106)
Nutrient transfer mechanisms

The efficiency with which placental transfer of oxygen and nutrients occurs is the major determinant of foetal wellbeing. The efficiency of this transfer can be influenced by a number of factors apart from the concentration of oxygen and nutrients in the maternal blood supply. Factors which can limit the supply of nutrients include a small villous surface area, insufficient vascularisation of the placenta and changes in the hydrostatic pressure in the intervillous space.

Nutrients are transferred from the maternal blood supply to the foetal blood supply by the same range of mechanisms that are used postnatally to transfer nutrients from the lumen of the gut to the capillaries and cells lining the gastrointestinal tract; that is, by passive or simple diffusion, facilitated diffusion, active transport and pinocytosis. The only difference is that the foetal capillaries are separated from the maternal blood supply by two layers of cells – those lining the foetal blood vessels (foetal endothelial cells) and those forming the placental membrane (trophoblast).

The major transport mechanism used for different nutrients can be inferred from the relative concentrations of the nutrient in the maternal and the foetal bloodstream. If the concentrations are equal, then transfer is most likely to occur by simple or facilitative diffusion. Oxygen, carbon dioxide, fatty acids, electrolytes and fat-soluble vitamins are transferred by passive diffusion and most monosaccharides by facilitative diffusion. Amino acids, some cations such as calcium and iron, and the water-soluble vitamins are transferred by active transport. This mechanism requires both a carrier protein for the nutrient and metabolic energy to move the nutrient against a concentration gradient. Proteins in general do not cross the placenta because their molecules are too large. An exception is the maternal immunoglobulin IgG which is probably transported by pinocytosis. The benefit of this transfer of IgG to the foetus is that it has the same resistance to infectious diseases at birth as does the mother.

Respiratory and excretory functions

The placenta functions as a conduit for the exchange of respiratory gases and waste products between the mother and the foetus. An adequate supply of oxygen depends primarily on the amount of blood flow through the placental villi. Towards the end of pregnancy, the rate of blood flow through the intervillous space may be as high as 500 mL/min.

Maternal haemoglobin levels influence the rate of oxygen transfer. Each gram of haemoglobin can carry 1.34 mL of oxygen. A blood haemoglobin level of 12 g/L can deliver 16 mL of oxygen per 100 mL, whereas blood with a haemoglobin level of 10 g/L can deliver only 13 mL per 100 mL. While this difference appears to be relatively small, near term when blood flow is around 500 mL/min., it amounts to a difference of almost 20 L of oxygen over 24 hours.

The placenta also provides for the removal of metabolic wastes and is freely permeable to carbon dioxide, water, urea, creatinine and uric acid. Carbon dioxide removal can occur by passive diffusion because during pregnancy the maternal pCO2 is reduced as a result of a ‘resetting’ of the respiratory centre to stimulate maternal overbreathing or hyperventilation.
Maternal blood is forced into the intervillous spaces by pressure in the maternal circulation. Exchange of nutrients and metabolites occurs as blood flows around the villi and then into the endometrial veins. On the foetal side, umbilical arteries carry deoxygenated blood from the foetus to the placenta and the umbilical vein returns reoxygenated blood to the foetus.

**Changes in body composition during gestation**

- Body water reduces from 90% to 70% by term.
- Protein accretion is relatively constant, and increases linearly with weight.
- Amino acids cross the placenta and provide the building blocks for protein synthesis.
- Fat accretion is low early in gestation and increases markedly late in gestation.
- Fat-free weight of the foetus increases progressively during gestation (Figure 2.4). Fat and protein accretion commences at 25 weeks gestation and increases until the end of gestation. For an infant born at term, protein and fat each account for around 15% of weight at birth.

![Figure 2.4](Forbes 1997, p. 106)
Foetal growth

The rate of growth of the foetus, relative to its size, is more rapid than at any other stage of the lifespan (Figure 2.5). In absolute terms, the rate of increase is from around 5 g/day at 15 weeks to around 35 g/day at 35 weeks gestation. Expressed in velocity terms the rate of growth is in excess of 5% of foetal weight per day in early foetal life. This very rapid rate of growth is possible because the foetus is spared the work of breathing and overcoming the effect of gravity and does not need to maintain its body temperature.

Birth weight is the most reliable indicator of an infant’s health, with higher birth weights leading to lower risks for the infants. Rees et al. (1996) have determined that the most favourable birth weight for neonatal survival is 3000–4499 g irrespective of the mother’s age. Three maternal anthropometric factors can influence the birth weight of a term baby: maternal height, weight and weight gain during pregnancy (Luke 1994).

1 Maternal height is correlated positively with infant birth weight.

2 Pregravid weight and weight gained during gestation both have an effect on birth weight. The percentage of infants born with a low birth weight is highest among women with the lowest pregravid weight and the lowest weight gain during pregnancy (Table 2.1). Mean birth weight increases across each category of gestational weight gain and maternal pregravid weight.

3 Smokers have reduced weight gain with all categories.
### Table 2.1 Relative growth rate

<table>
<thead>
<tr>
<th>Pregravid Weight and Smoking Groups</th>
<th>Gestational Weight Gain (lbs)</th>
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<th>21–25</th>
<th>26–35</th>
<th>&gt;36</th>
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<td></td>
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</tr>
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<td>3100</td>
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<td>3374</td>
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</table>

* Mean birthweight given in lbs. oz
Adapted from the National Center for Health Statistics 1980 National Natality Survey. (Luke 1994, p. 96)

Within Australia, Aboriginal mothers are twice as likely give birth to low birth weight babies than other Australian mothers (13.1% compared with 6.1%) (Figure 2.6). Several causal factors have been implicated, with maternal ill health and malnutrition recognised as significant determinants.

**Figure 2.6** Low birthweight babies, by indigenous status of mother and state/territory 2004–2005

(adapted from AIHW, 2007)
Intrauterine growth retardation (IUGR)

Definitions

The term \textit{intrauterine growth retardation (IUGR)} implies that foetal growth has been inhibited and that the foetus does not attain its genetic growth potential. At present there is no standard definition of IUGR and the condition is usually only diagnosed at birth. The size of the newborn is related to the duration of pregnancy. An infant born before term may be small but not necessarily have the diagnosis of IUGR. A relatively small size for gestational age is regarded as a reflection of intrauterine growth inhibition. Low weight for gestational age is usually referred to either as ‘small for dates’ or ‘small for gestational age’ (SGA). The tenth centile for birth weight for gestational age is often used as the cut-off for defining an SGA infant. Low birth weight is defined by the World Health Organization (WHO) as a birth weight <2500 g. A baby may weigh less than 2500 g at birth because it is born too early or because it is small for its gestational age (WHO, 2006).

The size of the foetus at any stage of pregnancy reflects a complex interaction between the time since fertilisation, the rate of foetal cell multiplication and growth in foetal cell size. The effect of growth inhibition thus depends on the timing of the growth-retarding factor. It has been suggested that inhibiting factors which operate early in foetal life lead to a symmetrically growth-retarded foetus, while inhibiting factors, which operate late in pregnancy, cause asymmetrical growth retardation. An example of the first type is a viral infection which affects mitosis in early pregnancy, while utero-placental insufficiency which reduces fat deposition is an example of the second type.

A symmetrically growth-retarded foetus is characterised by a normal ponderal index ((birth weight/birth height) \times 100) but a length, weight, head and abdominal circumference which is below the tenth centile for gestational age. An asymmetrically growth-retarded infant on the other hand is one in whom length and head circumference are appropriate for age, while weight and abdominal circumference are low (due mainly to a lower proportion of visceral and fat tissue) and the ponderal index is also low. These infants have poorly developed muscles and almost no subcutaneous fat. In practice, growth retardation is often a combination of symmetric and asymmetric growth retardation depending on the nature and timing of the growth-inhibiting factors.

Causes of IUGR

The causes of IUGR are multiple and involve many different factors. Studies in humans and animals suggest that the maternal environment is the most important determinant of newborn weight and has a greater effect on birth weight than genetic background. Probably the most important of these factors is the maternal plasma volume. Other factors include multiple gestation, infections and maternal disorders such as anaemia, severe chronic asthma, renal disease, heart disease and hypertension. Cigarette smoking, narcotic addiction and chronic alcoholism are also associated with IUGR. The nutritional status of the mother at conception, and to a lesser extent her energy and nutrient intake during pregnancy, can also result in IUGR. Placental abnormalities and small placental size are also important factors in intrauterine growth retardation. Chromosomal abnormalities in the foetus can lead to IUGR.
Because IUGR children are not a homogeneous group, they are likely to exhibit a broad range of growth, health and developmental outcomes. In general, however, they have higher rates of subnormal growth, morbidity and neuro developmental problems.

**Later effects of IUGR**

Individuals who have experienced IUGR are not a homogeneous group and thus it is not surprising that findings of studies which have looked at growth, morbidity, mortality and health in later life in IUGR infants do not always show consistent results.

The following section will now look at the consequences of low birth weight (LBW) in terms of morbidity and mortality.

**Neonatal morbidity and mortality**

Studies of morbidity and mortality in LBW infants have tended to focus either on problems occurring in the immediate neonatal period or, once survival is secured, and on longer-term developmental outcomes. A review of the available data (Ashworth 1998) indicates that the risk of neonatal death for infants weighing 2000–2499 g at birth is ten times higher than for infants weighing 3000–3499 g and the risk of post-neonatal death four times higher. Prevalence rates for neonatal mortality within Australia vary from state to state, with ranges from seven to 26 deaths per 1000. There is considerable variability between Indigenous and non-indigenous population groups (Figure 2.7).

**Figure 2.7**  
Perinatal mortality by Indigenous status in selected states and territories, 2004–2008

(adapted from ABS 2008)
The fact that neonatal mortality is greater than during the post-neonatal period in part reflects different causes of death in the two periods. In the neonatal period, deaths are due mainly to complications of delivery and congenital abnormalities, while infections are a more frequent cause in the postnatal period. Beyond acting as a risk factor for neonatal death, birth weight is too narrow an indicator for the wider aspects of health and development in the postnatal period and beyond. It is important to consider what influences foetal development and how this then determines effects on function throughout all stages of the life cycle.

Effects on neurological and cognitive development

There is evidence that SGA infants are at an increased risk of mild cognitive deficits and behavioural problems, especially attention deficits later in life. However not all studies demonstrate such an association and this may be attributed to differences in defining and measuring SGA and the fact that other confounding influences on behaviour and cognition exist and can be difficult to control for. For example, there may be gender differences in learning and behavioural outcomes. Additionally, growth restriction is more common in socially disadvantaged populations. Because low socioeconomic status is associated with learning and behavioural difficulties, the extent to which adverse IUGR outcomes is socially determined is unclear.

Body size, body composition and physical performance

A review of the effects of IUGR on growth in childhood found that IUGR infants show partial catch-up in growth relative to control infants during the first one to two years of life. After two years of age, however, IUGR children appear to maintain their position in the distribution and neither catch-up nor fall further behind (Martorell et al. 1998). Between two and seven years, the size of IUGR infants was significantly less than in controls and this was also true at age 17 to 19, when the difference was about 5 cm for height and 5 kg for weight.

The few studies that have examined whether IUGR is related to fatness in later life have shown inconsistent results. Thus, there is currently no support in the literature for a relationship between low birth weight and greater fatness at follow-up.
Table 2.2 from Martorell et al. (1998) shows data from a Guatemalan follow-up study of IUGR infants. In this study, IUGR adolescents at 15 years of age were both significantly shorter and had a smaller fat-free mass but not fat mass than adolescents with birth weights in the middle and upper part of the birth weight distribution. The study also found some evidence for reduced arm strength in the IUGR group.

### Nutrient requirements during foetal life

#### Macronutrient requirements

In absolute terms, the macronutrient requirements of the foetus are small, despite the very rapid rate of growth, because of its small size and low energy costs for maintenance. At 25 weeks, for example, when the foetus weighs around 800 g the total energy requirement over 24 hours is likely to be about 650 kJ, and at 35 weeks about 1500 kJ. Expressed in terms of glucose, which is the main energy source during foetal life, this amounts to a requirement of 40 g and 100 g per 24 hours, respectively.
**Glucose**

The supply of glucose to the foetus is dependent both on placental blood flow and on maternal blood glucose concentration. Because of the continual uptake of glucose by the placenta, maternal glucose levels generally tend to be lower than those of non-pregnant women after a period of fasting. This is relevant because the placenta cannot regulate glucose against a concentration gradient. With a blood flow of around 400–500 mL/min and a fasting blood glucose of around 100 mg/100 mL (5.5mmol/L), one can estimate that the amount of glucose presented to the foetus (580–720 g) normally is well in excess of demand, at all stages of pregnancy.

**Protein**

At 25 and 35 weeks the lean body mass of the foetus is 790 g and 2250 g, respectively (Table 2.3); most of this is water and only 15% is protein. One can estimate, therefore, that the average requirement for protein during the first two trimesters of pregnancy is less than 0.75 g of protein per day and only about 2 g per day in the final trimester. These amounts are negligible in relation to the average maternal intake of protein, and it is difficult to envisage that an inadequate maternal intake of protein might be responsible for a lack of amino acids in the foetus. It is more likely that if there is such a deficiency it is due to a failure of specific amino acid transport mechanisms. The cellular regulation of amino acid transport allows for separate regulation by the maternal and the foetal surface membranes so that most amino acids are concentrated both within the placenta and by the foetal circulation. Under normal circumstances, the foetus produces considerable amounts of urea indicating that the amino acid transport to the foetus greatly exceeds foetal requirements.

**Fat**

Lipids are used by the foetus for oxidation, for synthesis of cell membranes and cell regulators, and in the latter stages of gestation for storage as triglycerides in adipose tissue. In part, foetal lipids are synthesised by the foetus from glucose, but circulating maternal lipids are also an important source. Access to maternal lipids is assisted by the fact that the concentrations of all lipoprotein classes increase in the maternal circulation during pregnancy under the influence of sex hormones. Triacylglycerol in the maternal very low-density lipoprotein (VLDL) is hydrolysed by lipoprotein lipase on the maternal surface of the placental membrane and the free fatty acids are taken up by the placenta or transferred to the foetus.

The essential fatty acids cannot be synthesised in the foetus. Adequate intake of essential fatty acids and their long-chain polyenes (LCPs), arachidonic acid (AA), eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA), is required during pregnancy. The central nervous system is rich in AA and DHA. The foetal brain undergoes a growth spurt during the third trimester of pregnancy and during early childhood, and it is thought an adequate supply of LCPs or their precursors, namely via the maternal diet, is essential for neonatal growth. At term, brain weight makes up about one quarter of the total weight of the foetus and contains some 75 g of lipid (Cockburn 1995). The concentration of essential fatty acids (18:2 (n-6) and 18:3 (n-3)) in brain phospholipids is low at around 1–1.5%, while the concentration of their long chain derivatives (arachidonic acid 20:4 (n-6) and docosahexaenoic acid 20:6 (n-3)) increase in the brain from mid-gestation to term to much higher concentrations (15–30%).
The development of foetal adipose tissue begins in the third trimester. Because it is in part derived from maternal lipids, it tends to reflect the fatty acid composition of the maternal diet. At birth, the total fat content of the infant (including that in the brain) is around 500 g.

**Micronutrient requirements**

**Vitamins**

The foetus also has requirements for both water-soluble and fat-soluble vitamins, but the requirements are not as well documented as are the requirements for the minerals required for tissue growth. It is, however, clearly possible to estimate the amounts of vitamins available to the foetus from information on the rate of blood flow through the placenta and from information on the concentration of the vitamin in the maternal blood. In general, the concentration of the water-soluble vitamins in maternal plasma decreases during pregnancy, while that of the fat-soluble vitamins increases or stays the same. These differences are consistent with the different transfer mechanisms, since water-soluble vitamins are transferred by active diffusion; the concentrations do not need to be maintained at high levels in the maternal plasma, whereas for fat-soluble vitamins (which are transferred by passive diffusion) concentrations need to be maintained at levels above those found in the foetal circulation.

**Vitamin A**

Vitamin A, needed in small amounts, is important in early development because it is essential for both growth and cellular differentiation. The liver vitamin A concentration of newborn infants is relatively low and only requires the storage of 5 mg of retinol over the whole of the pregnancy. This amount constitutes only about 2% of the maternal liver store and an inadequate supply to the foetus is unlikely. An excessive supply is more likely to be a problem if the mother consumes large amounts of vitamin A during pregnancy. While no direct cause-effect relationship has been established, a small number of cases of birth defects have been reported in women who have consumed large doses (more than ten times the RDI) in pregnancy and it is recommended that during pregnancy maternal intake should not exceed 2800 µg RE per day in 14–18 year olds and 3000 µg RE per day in 19–50 year olds. Azais-Braesco and Pascal (2000) review the requirements of vitamin A during pregnancy. Note that the studies on vitamin A toxicity and birth defects are limited due to ethical reasons. One prospective study has been conducted to date which demonstrated that an intake of 3000 µg RE vitamin A significantly increased the risk of malformations. However, this study contradicts retrospective studies and may be flawed in the classification of malformations.

**Folic acid**

This section covers the role of folate in the development of the foetus. Additional information is also found in Topic 8 (nutrition during pregnancy).

Folic acid in the form of tetrahydrafolate is a carrier of carbon molecules essential for the synthesis and repair of DNA and RNA. These roles are critical for gene expression and cell proliferation. An adequate supply of folic acid to the foetus,
who is undergoing rapid growth and development, is therefore also essential. The fact that maternal folate status often deteriorates during pregnancy suggests that the foetal demand for folate is relatively high in relation to maternal stores.

Folic acid and neural tube defects

Neural tube defects (NTDs) are abnormalities of development of the central nervous system – predominantly anencephaly and spina bifida. Over 600 pregnancies in Australia every year are affected by NTDs. There is an association between dietary folic acid and the incidence of NTDs and the number of cases of NTDs has been shown to be reduced by up to two-thirds with adequate maternal intakes of folate (FSANZ, 2011). The neural tube closes around the 27th day after fertilisation and thus any influence affecting closure of the neural tube must be present before this early stage of pregnancy.

A randomised, controlled clinical trial carried out by the Medical Research Council Vitamin Study Group in the United Kingdom has demonstrated the protective effect, on recurrence of neural tube defects (NTD), of periconceptional supplementation with 4 mg folate daily (Medical Research Council 1991). Other data, including a study from Australia, suggest that lower doses may also be protective in reducing the incidence of first occurrence of these conditions (Bower & Stanley 1989).

Over a period of 15 years, 13 out of 14 studies have shown a protective effect of maternal folate intake and the incidence of NTDs (Bower 1996).

Exactly how folate provides protection against NTDs is still unclear. The balance of evidence indicates that folate does not prevent NTDs by correcting a nutritional deficiency during the periconceptional period. It appears that the preventive role may arise when folate is consumed in an amount equivalent to a pharmacological ‘dose’ (i.e. an amount several times the RDI) and acts to compensate for an error in a biochemical pathway involving folate metabolism in susceptible individuals.

Given the ethical issues associated with denying women access to folic acid, no further trials have been conducted since Bower’s 1996 paper. In 2001, a Cochrane systematic review was published by a team of Melbourne epidemiologists. They calculated that periconceptional folate reduces the prevalence of NTDs substantially. The reduction was similar for current and recurrent defects (Lumley et al. 2001). It should be noted, however, that the 2006 Nutrient Reference Values have set an EAR (estimated average requirement) of 520 µg and an RDI of 600 µg dietary folate equivalents per day during pregnancy to cover the increased dietary requirement for folate; however, they state that ‘this recommendation does not include consideration of additional needs to prevent neural tube defects as the neural tube is formed before most women know they are pregnant. The data indicate that maximal protection from NTD is obtained when the mother is consuming 5000 µg of dietary folic acid supplements in the month preceding conception and during the first trimester’ (NHMRC 2006; Wald et al. 2001).

From 1995 voluntary fortification of a limited range of staple foods with folate has been permitted. Mandatory fortification of bread-making flour with folic acid was introduced in Australia in 2009. The aim of this initiative is to lower the incidence of NTDs in pregnancy. It is currently too early to assess the impact of these fortification strategies on the occurrence of NTDs.
**Minerals**

Table 2.3 shows the accretion rates for the major elements accumulated in foetal tissues at various times during foetal life. The daily accretion rate of calcium at 35 weeks (330 mg) amounts to between one-third and one-half of the usual maternal intake and that for iron (3.5 mg) is also close to one-third of the usual maternal dietary intake and both amount to considerably more than is usually absorbed from the maternal diet. In contrast, the accretion rates for zinc, magnesium and nitrogen account for less than 10% of usual maternal intake.

From these data it is clear that calcium and iron are the nutrients for which foetal needs are significant. Meeting these needs are challenging and a marked increase in rates of absorption of these nutrients during pregnancy is observed. The supply of these nutrients to the foetus need not be compromised during a normal pregnancy in a healthy woman (see Topic 8) provided that she has adequate stores at the outset.

**Table 2.3 Foetal size and element accretion at selected times during foetal life**

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<tr>
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<td>Body weight (g)</td>
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<td>Lean body mass (g)</td>
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<td>Zinc</td>
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</table>

(Forbes 1987, p. 118)

Iron is needed for the production of haemoglobin in red blood cells of both the foetus and mother. The foetus will accumulate 250 mg of body iron stores by term. The foetus derives this iron from maternal iron stores. Infants born to mothers with moderate to severe iron deficiency anaemia had poor iron stores at birth when compared to infants born to non-anaemic mothers (Singla et al. 1996). The most common cause of iron deficiency anaemia in an infant is a premature delivery, whereby there is insufficient opportunity to lay down iron stores. Iron deficiency anaemia in pregnancy is a risk factor for preterm delivery and subsequent low birth weight, and possibly for inferior neonatal and infant health and development (Allen 2000).
Iodine and thyroxine are transferred from maternal supplies to the foetus during pregnancy and adequate supplies are required to ensure normal brain development and prevention of cretinism and mental retardation in the offspring. The weighted results from the National Iodine Nutrition Survey show that the median urinary iodine concentration in Australia is 96 µg/L, which is classified as mildly deficient by WHO criteria (Australian Population Health Development Principal Committee, 2007). Studies specific to the iodine status of pregnant women in Australia are limited. A recent study conducted in Australia indicated that 8% of neonates and 5% of pregnant women surveyed in Sydney had below normal values of urinary iodine. Iodine deficiency in pregnancy is emerging as a significant public health problem as low levels of iodine in the foetus (due to low levels in the mother) has a negative impact on the nervous system of the unborn baby and increases the risk of infant mortality (Zimmerman 2009). Strategies to ensure adequate iodine intake have recently been introduced in Australia including mandatory fortification of non-organic bread with iodised salt from October 2009. Additionally, the NHMRC recommends that women who are considering pregnancy should take an iodine supplement of 150 µg per day and continue taking this throughout pregnancy and breastfeeding.

Smoking, alcohol consumption and caffeine consumption during pregnancy

Alcohol

Maternal alcohol consumption can result in a spectrum of harm to the foetus. A number of alcohol related birth defects and neurodevelopmental disorders have also been described as a result of exposure to alcohol during pregnancy and, together with FAS, are collectively termed Foetal Alcohol Spectrum Disorders (FASD). FASD has been described in children exposed to high levels of alcohol in utero (around 2 g alcohol/kg body weight per day) as a result of chronic or intermittent maternal alcohol use. FAS is characterised by anomalies of the eyes, nose, heart and central nervous system, and is accompanied by growth retardation, a small head circumference and mental retardation. The condition is unlikely to lead to death but is associated with permanent physical and intellectual disabilities. The mechanisms by which alcohol produces effects on the foetus are not understood. Since alcohol can pass across the placenta and nearly equal concentrations in the mother and the foetus can be attained, it has been postulated that alcohol levels have a direct toxic effect on the foetus, particularly during the early phases of pregnancy. Alternatively, women who consume large quantities of alcohol may have micronutrient deficiencies due to displacement of more nutritious foods from the diet. These micronutrient deficiencies may contribute to developmental, behavioural and cognitive problems seen in FASD. However, not all children exposed to alcohol during pregnancy are adversely affected or affected to the same degree.

When considering moderate intakes of alcohol intake, a meta-analysis conducted found the relative risk of having a spontaneous abortion increased following consumption of one to two drinks per day in women aged 35 years and over (Polygenis et al. 1998). Surprisingly, the relative risk of having a low birth weight infant, a premature delivery or an infant with intrauterine growth retardation
decreased when compared to abstainers. Moderate alcohol consumption during the first trimester of pregnancy was not associated with an increased risk of foetal malformations compared with an intake below this level. Other studies regarding maternal alcohol intake and birth weight are conflicting. Two studies show 1.2 standard drinks per day (Mariscal 2006) or 3.6 standards drinks per day (Chiaffarino et al 2006) to be associated with risk of LBW or SGA, respectively. A large prospective cohort study which followed women through pregnancy showed no association between alcohol consumption and SGA (Jaddoe et al. 2007). Due to limitations in the available evidence and the fact that a ‘no-effect’ level of alcohol consumption has not been established, it is not possible to set a ‘safe’ or ‘no risk’ drinking level for pregnant women to avoid harm to their unborn children, although the risks to the foetus from one or two drinks per week are likely to be low (NHMRC 2009). In Australia, a conservative public health approach has been taken in recommending that ‘not drinking alcohol is the safest option’ for pregnant women.

Caffeine
It has been estimated that 75% of pregnant women consume caffeine. Caffeine administered in large single doses to rodents has teratogenic effects. When translated to humans these doses are quite large and are unlikely to be achieved under normal circumstances. For example, a woman weighing 60 kg would need to consume ten to 14 cups of coffee in one sitting to achieve plasma caffeine concentrations comparable to those observed in the rodents. Much lower doses, however, have been implicated in an increased risk of miscarriage. Maternal caffeine intake has been reported to be associated with a reduction in birth weight, but the precise level of intake above which the risk is increased remains unknown. A large prospective cohort study in the UK (2635 women) published by the CARE study group in 2008 confirmed previous research findings that a maternal caffeine intake of >300 mg/day is associated with low birth weight or foetal growth restriction. This group found that an average caffeine intake of >100 mg/day was associated with a reduction in birth weight of 66–88 g in the third trimester. This group concluded that although the threshold at which risk increases is not well characterised, the association of foetal growth restriction with caffeine is reduced for those consuming <100 mg/day (one coffee per day). On publication of these findings, the NHMRC in Australia pledged to determine a recommended intake of caffeine for pregnant women in Australia and the UK Food Standards Agency lowered its recommended upper limit from 300 mg/day to 200mg/day.

Smoking
The effects of smoking on foetal growth and birth weight are well recognised and are as marked as those of severe food restriction during pregnancy (Abel 1980). For example, the infants of women who smoke during pregnancy are on average lighter by 150–250 g than those of non-smokers. Smoking is also associated with a number of poor pregnancy outcomes such as spontaneous abortion, foetal death, complications of pregnancy and increased perinatal loss. When combined with other risk factors such as alcohol, drug abuse or malnutrition, smoking appears to have multiplicative rather than merely additive effects on perinatal mortality. The adverse effects of smoking during pregnancy increase with the number of cigarettes smoked. Good nutritional status and increased weight gain in pregnancy can reduce the extent of foetal growth retardation but are not substitutes for not
smoking, as the prevalence of the major causes of increased foetal and neonatal mortality in smokers remains the same irrespective of weight gain during pregnancy.

For many years the mechanism by which smoking caused foetal growth retardation was thought to be through its effect on maternal appetite and weight gain. However, on average, smokers and non-smokers tend to have similar levels of food intake and weight gains and the effect is more likely to be due to the direct effects of carbon monoxide and/or nicotine in cigarette smoke on placental function. Infants of maternal smokers have been found to be smaller at birth (Conter et al. 1995; Ong 2002), however, show complete compensatory catch-up growth over the first 6-12 months of life if smoking is not associated with other unfavourable risk factors during pregnancy (Conter et al. 1995). It is still possible however that deficits in bone mineral density and susceptibility to respiratory disease persist in offspring of smokers. Additionally, rapid catch up growth after a period of foetal growth restraint may predispose to obesity later in life (Ong 2002).

**Artificial sweeteners**

Food Standards Australia and New Zealand (FSANZ) approves the use of food additives including artificial sweeteners in the food supply where evidence demonstrates that no harmful effects will occur from their use. Pregnant women are included in its assessment of the safety and suitability of all additives including sweeteners. Saccharin is known to pass across the placenta to the foetus. Currently, there is no evidence to suggest that saccharin is harmful to the foetus. Aspartame use during pregnancy is considered safe. Aspartate, one of the metabolites of aspartame, does not pass across the placenta unless consumed in large quantities. Phenylalanine, the second metabolite of aspartame, can pass across the placenta but current intakes are well below toxic levels. Methanol, the third metabolite, is ingested in small amounts and is unlikely to cause any harm (American Dietetic Association 1993).

**Nutritional influences on foetal growth**

It is not possible, for obvious reasons, to examine maternal–foetal relationships directly at the molecular level in humans. However, we can gain some information from animal studies. For example:

1. **Growth failure.** Although a number of prenatal influences affect foetal growth, maternal malnutrition can be one cause of growth failure that results in low birth weight.

2. **Nature of tissue effects.** Animals malnourished from restrictions of their mothers’ diet throughout most of gestation are characterised by reduced number and size of cells in the placenta, reduced brain cell number and head size, proportionally reductions in the size of other organs and alterations in normal cell constituents and biochemical processes.

3. **Influencing factors.** The foetal consequences depend on the timing, severity and duration of the maternal dietary restriction. These consequences may be reversible if the restriction primarily affects growth in cell size, but a reduction
in the number of cells may be permanent if the restriction is maintained throughout the entire period of hyperplastic growth.

It should be noted that the effects seen in animals may be different in humans as the relative rates of growth are slower in humans compared to animals, the timing of maximum growth also differs, the number and size of foetuses that a mother must nourish in utero compared to her own body size and nutritional reserves are much smaller in humans and, finally, the magnitude of dietary deprivation is not normally seen in human populations under normal circumstances.

**Foetal growth related health outcomes**

It is estimated that 10 to 20% of low birth weight infants are due to intrauterine growth failure and that there is no one cause for the growth retardation. When the term ‘foetal malnutrition’ is applied to human infants, it simply means that there was a reduction in the maternal supply or placental transport of nutrients, so that foetal growth was retarded significantly below genetic potential.

It does not necessarily mean that the mother’s nutrition was the cause. There is no way to know how many growth-retarded infants are the result of maternal malnutrition.

**Foetal growth and adult disease**

Please read the article by Kimm (2004), ‘Fetal origins of adult disease: the Barker hypothesis revisited – 2004’.

This review raises doubts regarding the relationship between inadequate nutrition resulting in low birth weight and the subsequent development of cardiovascular disease. Evidence for the idea that some adult diseases may have their origins in utero is reviewed by Leon (1998) and Barker and Clark (1997). An association between smaller size at birth and increased risks of coronary heart disease, stroke, type 2 diabetes, adiposity, the metabolic syndrome and osteoporosis in adult life has been replicated by studies conducted by Barker and colleagues and occasionally other researchers (Kensara, 2005). Whether this effect is related to nutritional factors, genetic factors or problems with maternal/foetal blood supply is not known and peri-natal events appear to exert effects which are independent of environmental effects in adults (Rich-Edwards, 1997; Frankel, 1996). The association between birth weight and cardiovascular disease is likely to involve a range of factors including genetic factors, maternal health and nutritional status and the time in which the in utero exposure to environmental factors occurs. Slow growth in utero may be associated with the increased allocation of nutrients to adipose tissue during early development which may then result in accelerated weight gain during childhood (Bhargava 2004; Barker et al 2005) which may then infer greater risks for coronary heart disease, hypertension, obesity (and therefore some cancers), and type 2 diabetes.

We need a greater understanding of cellular and molecular processes involved to enable progress in this area. In particular we need to know:
what factors limit the delivery of oxygen and nutrients to the foetus
how the foetus adapts to a reduced supply of oxygen and specific nutrients
how these adaptations ‘program’ the structure and physiology of the body
by what molecular mechanisms nutrients and hormones alter gene expression.

QUESTIONS
1 Why is restriction in nutrient supply before ten weeks gestation, when absolute requirements for nutrients are very small, likely to have more adverse effects on foetal development than nutrient deficiencies which occur later in gestation?
2 What are the principal functions of the placenta and why are placental size and efficiency important?
3 What would happen if the placenta failed to adequately transfer nutrients to the foetus during the final trimester?
4 What are the mechanisms by which nutrients are transferred to the foetus and how are these affected by placental blood flow and maternal plasma concentration?
5 What are some of the factors that enable the foetus to grow at a greater rate in utero than at any time postnatally?
6 What are the major non-nutritional causes of IUGR?
7 Why are the findings of studies on the later effects of IUGR on growth and development during childhood likely to vary?
8 What are the most consistent findings of follow-up studies of IUGR infants on:
   • Weight and height during childhood?
   • Neurological and cognitive ability in later life?
   • Risk factors for disease in later life?
9 What is protein needed for during foetal development and in what quantities? Are protein intakes during pregnancy sufficient to meet the requirements of the foetus? What would happen if the mother did not consume one of the essential amino acids for a period of one week?
10 Is caffeine consumption during pregnancy likely to affect foetal development?

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National Health and Medical Research Council 1993, *Revised statement on the relationship between dietary folic acid and neural tube defects such as spina bifida*, 115th session National Health and Medical Research Council, June, AGPS, Canberra.

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**TOPIC 3**

**Nutritional requirements in infancy**

**PREPARED BY THE UNIT TEAM**

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Introduction

Infancy is defined as the period of life from birth to 12 months, and is a time of phenomenal growth and development. During the first few months after birth, tissue and organ synthesis rates are high, and tissues are undergoing a process of maturation. All of these factors dictate the intake of energy, macro- and micronutrients in specific quantities for the infant.

Learning objectives

At the conclusion of this section you should have an understanding of and be able to describe:

• the physiological characteristics of infants that govern their energy and nutrient needs
• the normal patterns and measurement of growth in infants
• the recommended daily intakes for energy, macro- and micronutrients
• the nutritional composition of breast milk and infant formula, and the role of solid foods in the diet of infants
• the major nutritional concerns in infants.

Learning resources

Print readings

The readings listed here are reproduced in the print readings for this unit.


Online reading

Access the reading listed here using your internet browser.

Deakin Studies Online (DSO)
Access the unit site on DSO <http://www.deakin.edu.au/current-students/> for the appendices and other additional resources you can use to supplement this topic.

Basic concepts

- Nutritional support of early physical growth and development provides the foundation for a healthy adult life.
- Infants are born with the ability to digest and absorb nutrients from human milk or formula. The digestive system matures during infancy, so that a wide variety of foods can be used by the end of the first year.
- Individual energy and nutrient needs reflect rapid growth demands for fuel, building materials and basal metabolism.
- Infant feeding behaviour follows a defined developmental sequence.
- Maturing oral structures and function determine developing infant eating skills and appropriate textures of food.

(Worthington-Roberts 2000)

Physiological characteristics of infants influencing energy and nutrient needs

The small body size of infants, their immaturity with respect to a number of physiological functions and their rapid rate of growth have important implications for the kind of diet that infants require in order to meet their energy and nutrient requirements.

Small body size

Small body size is associated with three features, each of which has important implications for infant feeding.

1. Limited gastric capacity
   A small gastric capacity limits the amount of food that can be consumed at any one time. The effect of this constraint makes it necessary for infants to consume small, frequent feeds.

2. Large surface area to volume ratio
   The surface area to volume ratio of an infant is approximately twice that of an adult. This means that losses of both heat and water are relatively greater in the infant and that the infant has a higher requirement per unit of body weight for energy and water than the adult.

3. High resting metabolic rate
   The resting metabolic rate of the infant is also about twice that of the adult calculated per unit of body weight. In effect this is a consequence of the small
body size and large surface area, which means that because heat production
(to maintain body temperature) and nutrient turnover are higher than in the
adult, the infant also has a higher requirement for nutrients per unit of body
weight.

**Immature physiological functions**

A number of physiological functions are not fully developed in the infant at birth
and have implications for the way in which the infant is fed.

1. **Kidney function**

One of the most critical aspects of the infant’s physiological immaturity is the
limited ability to concentrate urine. Older infants are able to concentrate their urine
to a level of 1000 mOsmol/L, but the younger infant may only be able to achieve a
concentration of 600 mOsmol/L. This means that the young infant requires more
water to excrete a given quantity of waste products.

2. **Swallowing reflex**

The infant at birth has a set of reflexes – the rooting, suckling and extrusion
reflexes – which enable the infant to coordinate suckling, breathing and
swallowing, but not yet move solid or semi-solid food from the front to the back of
the mouth. Stroking the cheeks and lips of an infant stimulates the rooting reflex
whereby the infant will turn toward the stimulus to eventually come in contact with
the nipple. Projection of the tongue following contact with the nipple and a
rhythmic suckling action ensure maximal and efficient intake of breast milk or
infant formula.

3. **Gastrointestinal function**

Pancreatic lipase activity is low in the newborn. Fat digestion is initially achieved
by lipases found in breast milk, and secreted from the tongue (lingual lipase) and
the stomach (gastric lipase). Maltase, isomaltase and sucrase activity reaches adult
levels by 28 to 32 weeks gestation. Lactase reaches adequate levels by term.
Pancreatic amylase levels are low or absent from birth to the age of four months.
Starch hydrolysis can be achieved by the presence of glycosidase and
glucoamylase present in the brush border of the small intestine.

**Body composition**

Changes are observed in the components of tissues, particularly changes in total
body water, lean body mass and body fat. Total body water as a percentage of body
weight decreases throughout infancy from approximately 70% at birth to 60% at
one year of age, which is predominantly due to a reduction in extracellular water
(Figure 3.1). Intracellular water is observed to increase, which is associated with
the increase in lean body mass.

The composition of the infant’s lean body mass is also immature at birth. Overall, it
contains more water and less protein than in later life and this means that during
early growth, protein is required not only for the deposition of new tissue, but also
for the maturation of that tissue.
Total body fat as a percentage of body weight also accumulates rapidly during the period of infancy. Fat accounts for 16% of body weight at term and this increases to 25% by one year of age (Figure 3.2).

**Figure 3.1** Changes in body water as a % of body weight in boys during the first two years of life

![Figure 3.1](image1)

(adpated from Butte et al. 2000)

**Figure 3.2** Changes in % fat mass in boys and girls during the first two years of life

![Figure 3.2](image2)

(adpated from Butte et al. 2000)

**Rapid growth rate**

Infancy is a period of rapid growth, with birth weight trebling and length increasing by 50% by the end of the first year of life. In order to assess growth, serial measurements of weight, length and head circumference are made at defined periods. Weight is generally recorded using a weighing scale with the naked infant in a horizontal position. For infants and children less than two years of age, recumbent length is measured, generally with a wooden measuring board.
Monitoring growth

Growth charts are the tools used to monitor growth from birth until the individual reaches the age of twenty years (see the Centers for Disease Control and Prevention growth charts in Appendices 1.1 to 1.6 on DSO). Measurements for weight and length are plotted on age and gender specific growth charts and compared with the percentiles on the chart. For example, a boy aged 6 months and weighing 8 kg is at the 50th percentile; he is heavier than half the reference population and lighter than half the reference population. The rate of growth is the most important factor, rather than one off measurements, as growth trends reveal more about a child’s nutritional status. A child who is on approximately the same percentile for height and weight and who is growing at a rate parallel to the next percentile line is very unlikely to have a serious nutrition or chronic health problem. If a child’s growth percentile is changing – and particularly when it is near or crossing the upper or lower extremes, the 5th and 95th percentiles – a reason should be sought. A trend towards weight loss over a month or more should prompt efforts to establish a nutritional cause or the existence of an underlying problem. However it is important to remember that growth charts are tools, not a diagnostic instrument and further clinical evaluation is required to determine if there is a medical concern.

The current Centers for Disease Control and Prevention (CDC) reference values are based on measurements which were largely derived from infants who were formula fed. As some infants are now breastfed exclusively until the age of six months and not formula fed, it is important that the growth of breastfed infants is examined separately to formula fed infants. If you refer to section 5.2.2 in the draft Australian Dietary Guidelines, you will note that an international study determined that the growth of breastfed infants was slightly below that of formula fed infants. Furthermore, in Australia, the growth of Aboriginal infants who are exclusively breastfed follows the CDC reference until six months of age then begins to fall away. Growth retardation is consistently noted in Aboriginal infants. By the age of 12 months, the weight increase in Aboriginal infants is around 0.5 Z scores below the reference, and this relatively poor growth has been found to persist in older children. In 2006, the World Health Organization (WHO) released updated growth charts based on both longitudinal and cross-sectional studies conducted in multi-communities in five countries around the world (3 developing and 2 developed countries). Approximately 14,000 0- to 5-year-old children took part in the study and at least 20% were breastfed for 0–3 months. At the time of printing this study guide, the WHO growth charts have been adopted by the US for children aged under 2 years but the growth charts have had limited uptake within Australia and the CDC growth charts remain the most common growth reference used in Australia (for example, they are being used in the Northern Territories, however,
Victoria continues to use the CDC growth charts). Therefore we are continuing to use them in this unit. However it is possible that in the near future the WHO charts will be adopted for use in Australia. You can access a complete copy of these charts and the methodology from <http://www.who.int/childgrowth/standards/en/> (retrieved 9 March 2012).

The practical implications of rapid growth rate, small body size and physiological immaturity of the infant for the type of diet required can be summarised as follows:

- The infant has to be fed frequently, usually at least three hourly initially, in order to meet the requirements for water and energy in the face of a limited gastric capacity.
- The diet has to have a high energy and nutrient density in order to meet the high requirements for energy and nutrients per unit of body weight.
- The high requirement for water and the need for a liquid diet combined with a high energy and nutrient density mean that the diet also has to have a high water content.

The following sections will discuss in detail infant requirements for energy, macronutrients, micronutrients and water.

**Energy requirements**

Growth requires energy for synthesis of growing tissues. In the first three months of age this is about 35% of total energy needs, falling to 5% at 12 months, 3% in the second year of life, 1–2% until mid-adolescence and then negligible in late teens and onwards. The best method of assessing energy needs is the doubly-labelled water technique. When this method is applied over a 24-hour period it includes estimates of the metabolic response to foods and the energy cost of tissue synthesis. For adults this equates to the daily energy requirements, but during infancy and childhood additional energy needs for growth are required and are estimated from growth velocity or weight gain equations. Energy needs in infancy are dependent on basal metabolic rate, activity levels and growth.

**Basal metabolic rate**

The energy requirements for infants at rest are twice as high per kilogram of body weight as those for adults. Part of this energy is needed to maintain body temperature, which is related to the large surface area to volume ratio of infants.

**Activity**

Energy for activity increases as the infant grows older. Less time is spent on sleep and more on movement. Requirements for activity are, however, individual – sleepy, placid babies will use less energy than restless, crying infants. The ability to estimate total energy expenditure by means of doubly-labelled water has made it possible for the first time to obtain an accurate estimate of the energy expended in physical activity. Using this technique, Wells and Davies (1998) have found that the proportion of energy expended on activity increased from 5% at six weeks to 34% at 12 months. These authors also observed that by 9–12 months, because of
the different levels of activity, energy expenditure varies considerably between infants, and the energy requirements of individual infants can no longer be predicted with any accuracy from body size alone (Wells, Hinds & Davies 1997).

**Growth**

Infancy is a period of rapid growth and as a consequence energy requirements expressed as kilojoules per kilogram of body weight are higher than at any other time in life. However, even within the normal range, there are large variations in the rate of growth among infants. Because of these uncertainties the energy requirements of infants are usually based on the observed intakes of healthy infants rather than on estimates of the amount of energy required for basal metabolism, activity and growth. It is possible, however, to derive an estimate of the amount of energy required for growth based on experimental data for body composition, reference weights for infants and estimates of the energy cost of tissue synthesis as described in the readings by Davies (1998) and Wells and Davies (1998).

**Table 3.1**

Estimated energy requirements of infants and young children

<table>
<thead>
<tr>
<th>Age (months)</th>
<th>Reference weight</th>
<th>EER (kJ/day)</th>
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<tbody>
<tr>
<td></td>
<td>Boys</td>
<td>Girls</td>
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<tr>
<td>1</td>
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</tr>
<tr>
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<td>24</td>
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**Rationale:** For infants and 1-2 yr olds, the equations used for estimating energy expenditure were those produced by the Food and Nutrition Board in developing the US/Canadian DRIs values (FNB/IOM, 2002). There are some 14 DLW studies in infants (Butte, 2001), mostly done in the UK and the USA. This method involves consideration of gender, age, body weight and height/length and use of these to derive total energy expenditure (TEE). Physical activity level (PAL) categories are not used in calculating the requirements of infants. To the TEE estimate (89x weight of infant in kg +100), requirements for growth are added (FNB/IOM, 2002) assuming an additional need of 730kJ/day for 0-3 months; 230kJ/day for 4-6 months; 90kJ/day for 7-12 months and 85kJ/day 1-2 years using the estimates of energy content of tissue deposition from Butte et al (2000b) in conjunction with the 50th percentile for weight gain at various ages (Guo et al, 1991).

Four studies with breast-fed and formula fed infants have shown higher total energy expenditures (TEE) in formula fed infants. (Butte et al, 1990, 2000; Jiang et al, 1998; Davies et al, 1990) averaging +12% at 3 months, +7% at 6 months, +6% at 9 months and +3% at 12 months. At 18 and 24 months, no differences were seen (Butte et al, 2001).

(Table adapted from FNB/IOM 2002; reference weights from Kuczynski et al. 2000. Rationale from NHMRC 2006)
The fall in energy requirements between three and six months, which is maintained until nine months, represents a period when the very high growth rate characteristic of the first three months of life has declined, but is not yet balanced by an increase in physical activity. As the infant’s growth rate slows down during the first year of life, the energy requirement per kilogram of body weight at first decreases and then again increases as the level of activity increases towards the end of the first year. Irrespective of these changes, the amount of energy required per kilogram of body weight throughout infancy is three to four times greater than that of adults.

Breastfed infants have lower requirements for total energy expenditure and energy cost of growth than do formula-fed infants. The energy requirements in infancy is primarily determined by body size, physical activity and growth rate, and these all vary widely, therefore the ranges of energy needs are large.

### Macronutrients

#### Protein

Infants require protein for synthesis of new body tissue during growth, as well as synthesis of enzymes and hormones. The 2006 adequate intake (AI) for protein for infants at 0–6 months is lower than the 1991 recommendations (1.43 g/kg/day versus 2.0 g/kg/day).

The amount of protein required for growth and maturation of tissues can be calculated from the increase in body weight (3.5–10.0 kg) and the increase in protein content of the tissues (11–15%) that occurs over the first year of life. The requirement for growth and maturation constitutes a high proportion (60–75%) of the total protein requirement in early infancy, but rapidly decreases to around 40–50% at six months and as little as 15% at one year of age.

The dietary requirement for protein has two components: a requirement for a total amount of nitrogen and requirement for certain essential amino acids. If one or more essential amino acids are deficient in the diet, the rate of protein synthesis is reduced and as a consequence growth rate declines. An infant requires histidine, and some infants may also require cystine and taurine in addition to the nine essential amino acids required by adults.

#### Carbohydrate

Carbohydrate provides energy, spares protein as a source of energy and prevents ketosis. The main carbohydrate in both breast milk and cow’s milk is lactose. Breast milk contains 6–7% lactose as compared to 4.5–5% in cow’s milk, and infants fed breast milk obtain 35% of their energy from lactose. Breast milk also contains around 1% of oligosaccharides and other nitrogen-containing carbohydrates. One of these, L-bifidus factor, is present in much greater concentrations in human than in cows’ milk. Lactose appears to facilitate the absorption of calcium and magnesium as well as preserving an acid medium in the lower intestine. The acid medium facilitates the growth of a Lactobacillus bifidus flora and decreases the growth of Escherichia coli and other bacteria responsible for some forms of diarrhoea.
Fat

Please now read sub-section ‘3.1.4.1 Infants’ (p. 81) of section ‘3.1 Limiting intake of foods and drinks containing saturated and trans fat’ in the draft Australian Dietary Guidelines.

The dominant supply of energy during the foetal stage is glucose, whereas after birth this switches to fat, specifically triglycerides (Lindquist & Hernell 2010). In newborn preterm infants, it has been estimated that 20–30% of dietary fat may be malabsorbed and excreted in the stools.

Fat from breast milk or infant formula is the principal source of energy for the newborn infant, supplying about 50% of total energy intake. It is an important component of infant diets for several reasons:

• Fat provides a concentrated source of energy at a time when growth rate is high and there is only a limited capacity for food intake.

• Fat provides essential fatty acids including linoleic acid and α-linolenic acid.

• Fat is a vehicle for the fat-soluble vitamins A, D, E and K.

• Fat provides a source of energy without increasing the renal solute load (e.g. protein) or lead to hyperosmolar effects in the small intestine (e.g. disaccharides).

Micronutrients – vitamins, minerals and trace elements

Vitamins – general considerations

Recommended daily intakes for vitamins and minerals in the first six months of life are based on the estimated intake from breast milk. Since not all babies are, or can be, breastfed, other considerations also apply in making recommendations for nutrient intake in this age group. Some nutrients – for example thiamin, vitamin B₆ and vitamin C – are heat labile and may be destroyed during the preparation of infant formula. In such cases the recommended amount may take this into account and thus be greater than the amount received by a breastfed infant. In the case of other nutrients such as calcium, iron and zinc, for which the absorption from human milk is greater than from cow’s-milk-based formulas, a range of vitamins may be given in order to allow for differences in absorption. Yet another situation arises where the requirement for a nutrient is governed by the composition of the diet. For example, the higher the protein content of the formula, the higher the requirement for vitamin B₆; or the higher the polyunsaturated fatty acid content, the higher the requirement for vitamin E.

In the second six months of the first year of life additional considerations apply. The principal one is that milk, either breast milk or infant formula, is usually no longer the sole source of nutrients in the diet. In general, the introduction of other foods is associated with a decrease in both the availability and the absorption of nutrients from a mixed diet as compared with milk alone and this has to be taken
into account. The body size of the infant is also greater than in the first six months of life and this also needs to be considered.

**Fat-soluble vitamins**

**Vitamin D**

Normal bone and tooth formation depend on adequate amounts of vitamin D. Australian children who are out in the sunshine for much of the year will obtain adequate amounts of vitamin D from sunlight. However, infants who get little or no exposure to sunlight rely on a supply of vitamin D from milk. Breast milk contains only a small amount of vitamin D (0.5 µg/L) and some breastfed infants may need a vitamin D supplement. In the case of little or no exposure to sunlight, the 2006 AI is for 5 µg/day and commercial formulas contain this amount.

Infants who do not receive sufficient vitamin D develop rickets. Nutritional vitamin D deficiency was found to be a health problem in young children born to migrant parents living in Melbourne and were either exclusively or predominantly breastfed (Pillow et al. 1995). Infants born to mothers with vitamin D deficiency or low plasma vitamin D, who are exclusively breastfed are at risk of developing vitamin D deficiency, as the vitamin D content of breast milk will be very low. However, toxicity can occur with overuse of vitamin D supplements and this can result in failure to thrive, vomiting, bone changes and mental retardation.

**Vitamin K**

Vitamin K is found both in human milk and synthesised by the bacterial flora in the bowel. The colonisation of the bowel by bacteria, however, does not occur immediately after birth and may take a few days. During these first few days after birth the infant may therefore have low levels of prothrombin and other blood-clotting factors, and for this reason vitamin K is usually given intramuscularly immediately after birth to prevent haemorrhage.

Breast milk contains approximately 2.5 mg/L of vitamin K and after the neonatal period this appears to be adequate. The 2006 AI is 2–2.5 µg/day for 0- to 12-month-old infants. This figure assumes that all infants receive prophylactic vitamin K by injection. Infant formula contains much higher amounts than that found in breast milk (50–100 µg/L). No upper tolerable intake for vitamin K has been proposed.

**Water-soluble vitamins**

**Vitamin C**

Vitamin C is easily destroyed by heat and oxidation. Insufficient amounts of this vitamin in the diet lead to scurvy as a result of defective collagen synthesis which in turn causes poor wound healing and fragile capillaries. In infancy, scurvy can be mistaken for the ‘battered baby’ syndrome. Scurvy in infants is now rarely seen but when reported usually occurs in infants fed heat-treated cow’s milk but no other food. Cow’s milk has low vitamin C content, whereas breast milk from mothers who have an adequate diet contains 30–50 mg/L.

The 2006 AI for infants is 25–30 mg/day. Infant formula contains higher amounts of vitamin C to allow for loss on mixing and storage.
Minerals

Calcium, phosphorus and magnesium

Vitamin D and the minerals calcium, phosphorus and magnesium are necessary for bone and tooth formation. Bone contains most of the body’s calcium and phosphorus and half the magnesium. During times of rapid growth, as in infancy, the requirements for these minerals are high in relation to body weight.

Mature breast milk contains 300–350 mg calcium and about 150 mg phosphorus per litre, with a calcium to phosphorus ratio of 2.3:1, which is thought to be optimal. However, healthy full-term infants can adjust to a range of phosphorus levels that are found in commercial infant formula and infant diets.

The dietary requirement for calcium is affected by the amount of vitamin D in the diet. Calcium absorption can be upregulated with optimal vitamin D status and the presence of lactose and some amino acids may also enhance absorption. The 2006 Australian recommendations are for an AI of 210–270 mg/day for infants between birth and 12 months. The AI for 0–6 months was set by multiplying the average intake of breast milk (0.78 L/day) and the average calcium concentration in breast milk (264 mg/L). Formula-fed infants require additional amounts – approximately 350 mg/day – as calcium is less bioavailable in formula. The AI for infants 7–12 months was set by adding the estimate for calcium from breast milk to the estimate of intake from supplementary foods (140 mg), assuming a volume of 600 ml of breast milk and calcium content of 210 mg/L (126 mg).

The phosphorus AI is derived in a similar manner and the 2006 AI is 100 mg for 0–6 months, then 275 mg for 6–12 months.

Sodium and potassium

ONLINE READING

Please now read sub-section ‘3.2.4.2 Infants’ (p. 86) of section ‘3.2 Limit intake of foods and drinks containing added salt’ in the draft Australian Dietary Guidelines.

Sodium and potassium are found in human and cow’s milk. Human milk provides an average of 7 mmol/L of sodium, as do most commercial breast milk substitute formulas, whereas unmodified cow’s milk is higher in sodium, containing 21 mmol/L. Sodium deficiency only occurs if there is excessive loss by vomiting or diarrhoea. The 2006 AI for sodium are 120 mg/day for infants 0–6 months and 170 mg/day for those 6–12 months, calculated from the content found in breast milk.

Potassium is found in breast milk at about 15 mmol/L. The 2006 AI are for 400 mg/day for infants 0–6 months and 700 mg/day for those 6–12 months.

Trace elements

Iron

Iron is found as a component of haemoglobin in red blood cells, and is required to transport oxygen around the body. Iron accumulates in utero in proportion to body size. The pre-term and low birth weight baby at birth will have limited reserves that will rapidly deplete during postnatal growth. Without sufficient iron, haemoglobin levels will be low and anaemia will result.
Breast milk contains only about 0.7 mg iron/L, but this is very easily absorbed; in full-term infants with adequate iron stores it is generally sufficient until approximately six months of age, when additional iron from other foods should be introduced. Pre-term infants with low iron stores will, however, require an iron supplement during the first few months of life.

Cow’s milk contains less iron (about 0.5 mg/L), and infants fed cow’s milk for long periods of time without other iron-containing foods may become anaemic. Iron is added to commercial baby formulas in the form of iron salts, in varying amounts. The rationale for this is that augmentation of iron stores early in life safeguards against iron deficiency during the weaning period. The 2006 AI for infant for iron is 0.27 mg/day up to the age of six months with an additional intake recommended for infant formula due to the reduced absorption. From 7–12 months there is a large jump in requirements and the proposed RDI is 11 mg/day with an even greater intake recommended for those following a vegetarian diet.

Zinc
Colostrum has a high zinc content, which falls as transitional and mature milk appear. Mature breast milk contains 2–5 mg/L and reduces over time. Infants fed sufficient amounts of milk, either breast or cow’s, have an adequate zinc intake.

Iodine
Iodine occurs in breast milk in amounts ranging from 20–120 µg/L. The 2006 AI is 90–110 µg/day based on a level of 115µg/L in human milk. Children born to mothers who have goitre arising from a deficiency of iodine may have hypothyroidism at birth which, if not treated, may result in cretinism and permanent mental retardation. Iodine deficiency can result in major birth abnormalities and is associated with increase prenatal and infant mortality, mental deficiency and psychomotor effects. In the past iodine deficiency was not a concern in Australia as many households used iodised salt for domestic use. Recently, however, the use of iodised salt has significantly reduced, and it is estimated that it is only purchased by 10% of households. In addition, iodised salt is not used in the food industry. In a study conducted in Australia, urinary iodine excretion was measured as an indicator of iodine status in school children, healthy adults, pregnant women and patients with diabetes (Li et al. 2001). Sixty per cent of the pregnant women had urinary iodine levels consistent with mild to moderate iodine deficiency, and this is a concern for the development of the infant. In another study conducted in northern Sydney, it was found that a population of neonates and pregnant women might have mild iodine deficiency (McElduff et al. 2002). Recently the Australian government has approved mandatory iodine fortification of certain foods. The result of this initiative is that salt used in the manufacture of most bread (organic varieties are exempt) within Australia and New Zealand must be fortified with iodine. It is expected that this fortification strategy will help to improve the iodine status of the general population within Australia, however, the NHMRC still recommends that all women who are pregnant, breastfeeding or considering pregnancy, take an iodine supplement of 150 micrograms (µg) each day.

NHMRC 2010
Water requirements

Water intake is very important in infancy. Not only do infants have high body water content at birth (approximately 70% of body weight) but water loss by evaporation is much greater than in adults because both the surface area and the basal metabolic rate per kilogram of body weight are approximately twice that of the adult.

Water intake must cover the following:

- loss from skin and lungs by evaporation
- loss in faeces
- urinary water loss
- an allowance for growth.

Evaporative water loss

Evaporative water loss constitutes the major route of water loss, and ranges from 30–70 mL/kg/day in healthy full-term infants. However, in high environmental temperatures (40˚C), evaporative water loss increases by 50–100%. It also increases when body temperature is raised, as in fever, by 12% for each degree Celsius above 37˚C.

Faecal water loss

Faecal water loss averages 10 mL/kg/day. If the infant has diarrhoea then this will increase depending on the severity of the diarrhoea.

Urinary water loss and renal solute load

The amount of water lost in the urine depends not only on the level to which the infant is able to concentrate the urine but also on the diet. Waste products such as non-metabolisable dietary components, especially electrolytes and metabolic end products in excess of body needs, must be excreted in the urine. The collective name for these products is renal solutes. The total renal solute load is measured in milliosmoles (mOs) per day. The renal solute load consists of the nitrogenous end products of protein metabolism, sodium, potassium, phosphorus and chloride. A rough estimate of the renal load can be made using a rule of thumb which assumes that each gram of protein intake leads to the excretion of four mOsmol of urea and each millimole of electrolyte (Na, K and Cl) to the excretion of one mOsmol of renal solute. Most adults are able to concentrate urine to 1300 mOsm/L, but a health newborn may only be able to concentrate urine at 600–900 mOsm/L. An isotonic urine of 280–310 mOsm/L is the goal.

Difficulties with the renal solute load are unlikely in normal infants fed human milk, but low fluid intake, concentrated feeds or additional evaporative water loss all increase the renal solute load and can lead to dehydration.

Under normal circumstances full-term infants require 150 mL/kg until three months, 130 mL/kg from 3–6 months, 120 mL/kg from 7–9 months and 110 mL/kg from 10–12 months. The 2006 AI for water indicate that fully breast/bottle fed infants do not require any supplemental water as breast milk is 87% water and the
recommended intake (AI) is 0.7 L breast milk or formula for 0–6 months and 0.8 L for 7–12 months.

The following sections will now examine the practical aspects of feeding during the first year of life. It will look at breast feeding, infant formulae, introduction of solids and the emergence of feeding skills throughout the first year of life.

**Infant feeding: birth to six months**

Exclusive breast feeding is recommended for around the first 6 months of life with infant formula recognised as the only suitable substitute for breast milk during this period.

**Advantages of breastfeeding**

Please now read section ‘5. Encourage and support breastfeeding’ (pp. 131–43) in the draft *Australian Dietary Guidelines*.

The advantages of breastfeeding for both the mother and child include:

- immunological advantages
- nutritional advantages
- emotional advantages
- practical advantages.

**Immunological advantages**

Breast milk consists of many different components, which interact together to provide an effective defence system against disease for the newborn infant, whose immune system is not fully developed.

Breast milk contains growth factors for *L. bifidus* which, when it colonises the bowel, creates an acid medium and retards the growth of enteric pathogens such as *E. coli* and *Shigella*.

Immunoglobulins found in breast milk include IgA, IgM, IgG, IgD and IgE. The main immunoglobulin, IgA, is found as secretory IgA particularly in colostrum and during the first few months of lactation. Secretory IgA, like lactoferrin, is resistant to digestion by digestive enzymes and is found in high concentrations in the stools of breastfed infants. IgA coats the mucous membrane of the intestine and acts as a barrier to a variety of toxic agents of both bacterial and viral origin.

The breastfed baby is much less likely to suffer from bowel upsets than the formula-fed baby. This is in part due to the immunological properties of breast milk but also because bottles, teats and artificial milk feeds can become contaminated with pathogenic bacteria. Breastfeeding also provides protection against respiratory infection, reducing the risk of developing allergies.
Nutritional advantages

Breast milk is well suited to the growth rate and nutritional requirements of the full-term infant. It contains adequate amounts of energy, protein, carbohydrate, fat and micronutrients for normal growth. The protein has a high whey-to-casein ratio which is easier to digest, and also supplies the essential amino acids in the required amounts. The low solute content minimises the renal solute load. The lipids in breast milk contain long-chain polyunsaturated fatty acids, as well as shorter-chain fats, and these are more easily absorbed when compared to the fats in cow’s milk. Breast milk contains the essential fatty acids linolenic acid and linoleic acid. The longer chain derivatives arachadonic acid and docosahexanoic acid (DHA) are also present. Breastfed babies have higher levels of DHA present in plasma membranes and this may be associated with increased visual acuity and cognitive development (Agostini & Giovannini 2001). The article by Auestad et al. (2003) describes a long term follow-up trial looking at the efficacy of n-3 and n-6 long chain polyunsaturated fatty acids (LCPUFA) on cognitive and motor development in infants. What did the authors conclude about the addition of LCPUFAs to infant formula?

Emotional advantages

Breastfeeding promotes a feeling of security in the infant and encourages a close relationship between mother and child. Many women find breastfeeding enjoyable and as they gain confidence, enjoy nurturing their child. It should be stressed, however, that formula-fed babies can also achieve effective bonding with their parents. Eye-to-eye contact at the time of formula feeding helps to create a bond between parent and baby.

Practical advantages

For the infant, the practical advantages of breastfeeding include proper development of the jaw and teeth, because breastfed infants have to suck harder than formula-fed babies to obtain their milk.

Problems that may occur with breastfeeding

Babies who fail to grow and gain weight but who show no signs of physical illness may not be receiving adequate supplies of milk. This is most readily determined if infants are weighed regularly in the first few months of life. If there is any doubt that the infant is receiving sufficient milk, every effort should be made to increase the milk supply before supplementary bottle-feeding is commenced.

Breastfeeding trends

Breastfeeding rates reached an all time low in 1970 across a number of countries (Figure 3.3). By 1975 an upward trend not only in the number of babies being breastfed but also in the duration of breastfeeding began to show in the Victorian records, and by 1980 Tasmania, South Australia and New South Wales all showed a dramatic increase in the prevalence of breastfeeding. The Australian Institute of Health and Welfare report A picture of Australia’s children provides recent data on breast feeding rates in Australia (AIHW 2009). These national data indicate that...
overall 88% of children aged 0–3 years had been breastfed (exclusive or complementary feeding) at some stage. The general trend indicates that rates of breastfeeding decreases with increasing age. Eighty-six per cent of children were breastfed up to one month, but only 57% and 51% and 4 and 6 months, respectively. When comparing to Indigenous children, 80% were breastfed (exclusive or complementary) at one month and 62% and 48% at 4 and 6 months, respectively.

Figure 3.3 Changes in the proportion of infants' breastfed at different ages in developed countries

a) UK breast feeding rates, 1965–2006

( Symonds & Ramsay 2010, p. 108)

b) UK breast feeding rates, 1975–2000

(Langley-Evans 2009, p. 113)
Factors associated with the initiation and duration of breastfeeding

There are several possible reasons why mothers either do not breastfeed or give up breastfeeding after a few weeks and some of these reasons are given below.

- **Education.** The higher the education level attained by the mother, the more likely she is to breastfeed and for a longer period of time. Sixty-four per cent of mothers with post-school qualifications were breastfeeding their infants at six months compared with 41% of mothers with no post-school qualifications (ABS 2007).

- **Age.** Mothers who are greater than 25 years of age are more likely to choose to breastfeed and to breastfeed for a greater period of time. The ABS report, Australian Social Trends (ABS 2007), reported 55% of mothers over 30 years still breastfed their infants at 6 months compared with 38% of those aged 18–29 years.

- **Prenatal breastfeeding intentions.** Women who had decided to breastfeed prior to pregnancy were more likely to breastfeed after the birth of the child when compared to women who had made this decision either during or after the pregnancy.

The Scott et al. (2006) article in your readings discusses the myriad of factors associated with the initiation and duration of breastfeeding in a Perth-based cohort study and will be discussed in further detail in Topic 9.

Infant formula

Not all infants are able to be breastfed for the first six, let alone, the first 12 months of life. Some infants cannot be breastfed at all.

**Cow’s milk: why is it unsuitable?**

There are a number of reasons why cow’s milk is unsuitable as the main source of nutrition for infants. They are:

- Both the quality and quantity of protein is unsuitable for human infants. The concentration of protein is 3.3g/100mL, which is 2.5 times higher than that of breast milk.

- The predominant protein is casein (casein:whey ratio is 80:20) which is difficult to digest.

- The fat concentration is the same as for breast milk but the type of fat (butter fat) is poorly absorbed.

- The carbohydrate is the same type (lactose) as in breast milk but at a lower concentration. Cow’s milk has 4.6 g/100mL compared to 7g/100mL in breast milk.

- The renal solute load of cow’s milk is very high:

  | Cow’s milk: 22.8 mOsmol/100mL |
  | Breast milk: 7.0 mOsmol/100mL  |
  | Infant formula: 9.1–12.3 mOsmol/100mL |
• Calcium is less readily absorbed.
• Phosphorus is less readily absorbed.
• Intestinal blood loss occurs in a substantial percentage of infants fed whole cow’s milk before 12 months of age (cause unknown).
• The levels of vitamin A, C, D, E, niacin and iron are lower than in breast milk.
• No immune factors.

Low fat milks are unsuitable for infants, as the reduced fat content will result in a low-energy feed and provide insufficient fat-soluble vitamins. Table 3.2 shows the difference in nutrient content between human milk, infant formula and cow’s milk.

Table 3.2  Composition (per 100 mL) of mature breast milk and cow’s milk, and compositional guidelines for infant feeding

<table>
<thead>
<tr>
<th>Component</th>
<th>Mean value for mature human milk</th>
<th>Infant formulaa</th>
<th>Cow’s milk</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy (kJ)</td>
<td>280</td>
<td>282</td>
<td>291</td>
</tr>
<tr>
<td>Energy (kcal)</td>
<td>67</td>
<td>67</td>
<td>70</td>
</tr>
<tr>
<td>Protein (g)</td>
<td>1.3</td>
<td>1.5</td>
<td>3.5</td>
</tr>
<tr>
<td>Fat (g)</td>
<td>4.2</td>
<td>3.7</td>
<td>3.5</td>
</tr>
<tr>
<td>Carbohydrate (g)</td>
<td>7.4</td>
<td>7.1</td>
<td>6.4</td>
</tr>
<tr>
<td>Sodium (mg)</td>
<td>15</td>
<td>18</td>
<td>37</td>
</tr>
<tr>
<td>Chloride (mg)</td>
<td>43</td>
<td>45</td>
<td>97</td>
</tr>
<tr>
<td>Calcium (mg)</td>
<td>26</td>
<td>45</td>
<td>100</td>
</tr>
<tr>
<td>Phosphorus (mg)</td>
<td>12</td>
<td>32</td>
<td>92</td>
</tr>
<tr>
<td>Iron (mg)</td>
<td>0.26</td>
<td>0.9</td>
<td>Trace</td>
</tr>
<tr>
<td>Vitamin A (ug)</td>
<td>31</td>
<td>87</td>
<td>56</td>
</tr>
<tr>
<td>Vitamin C (mg)</td>
<td>3.8</td>
<td>7.1</td>
<td>0</td>
</tr>
<tr>
<td>Vitamin D (ug)</td>
<td>0.01</td>
<td>1.0</td>
<td>0.5</td>
</tr>
<tr>
<td>Potassium (mg)</td>
<td>50</td>
<td>66</td>
<td>146</td>
</tr>
<tr>
<td>Magnesium</td>
<td>3.4</td>
<td>5.3</td>
<td>10</td>
</tr>
<tr>
<td>Renal solute load</td>
<td>Average 290</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* Values for Nurture Starter 1 infant formula (whey dominant formula) from Heinz.

(NHMRC 2006)
Types of formulas

Infants under six months of age who are not breastfed should receive a commercial infant formula from one of the following categories:

- Whey dominant formulas.
- Casein dominant formulas.
- Soy-based formulas.
- Follow-on formulas.

Whey dominant formulas

This category of formula is made from modified cow’s milk with additional vitamins and minerals (refer to Table 3.2 for nutritional composition).

Energy: Equivalent to breast milk (280 Kj/100mL).

Protein: amount equivalent to human milk and modified to be whey dominant. The whey-to-casein ratio is similar to that of breast milk (60:40).

Fat source: oleic oil.

Casein dominant formulas

Energy, fat, carbohydrate, vitamin and mineral levels are the same as those of breast milk and whey dominant formula. The amount of protein is equivalent to breast milk and whey dominant formula, but is predominantly casein (whey:casein ratio of 20:80). There is no nutritional advantage of having a whey dominant over a casein dominant formula, and both are suitable from birth.

Follow on formulas

This class of formula has higher protein levels than human milk (closer to cow’s milk) with added vitamins and minerals. They also have a higher renal solute load and higher levels of sodium, potassium, iron and zinc relative to human milk. They are only suitable for infants greater than six months of age.

Soy formulas

Soy-based formulas use soy isolates as the protein source. This type of formula is often used in the management of cow’s milk protein allergy; however, it should be noted that approximately 40% of infants allergic to cow’s milk protein also have an allergy to soy protein. Soy protein can be used if the infant has lactose intolerance as the predominant carbohydrate in soy formula is sucrose.

All commercial formulas are fortified with iron, often to high levels. The rationale for this is that the added iron is not absorbed well by infants and therefore a higher level of supplementation is required. The Australian Food Standards Code specifies that infant formula should contain not less than 100 µg/100kJ and not more than 480 µg/100kJ of iron. This is equivalent to not less than 2.8 mg/L and not more than 13.4 mg/L assuming an average energy content of 280 kJ/L. The commercial breast-milk substitutes are all fortified with vitamins and minerals, but lack the immune bodies found only in breast milk.

Additional information about the nutritional composition of each infant formula can be obtained directly from the manufacturers.
WHO International Code of Marketing of Breast-Milk Substitutes

The World Health Organization International Code of Marketing of Breast-Milk Substitutes was formulated to promote breastfeeding and to protect the public from over-zealous promotion and use of breast-milk substitutes (WHO 1981). The guidelines on the implementation of the Code cover the responsibility of health personnel in educating the public about the advantages of breastfeeding. They require that, while information and educational materials should give clear advice about the advantages of breastfeeding, they should not disadvantage those women who decide not to breastfeed, and information should be made available on suitable feeds for these babies.

Under the Code, companies who market breast-milk substitutes should not advertise the superiority of their product over breast milk, or give out free samples to pregnant women. In the guidelines, the responsibility for educating health workers in regard to their responsibilities in promoting good infant-feeding practices lies with the government.

Problems that can occur with formula-feeding

Microbial contamination
The risk of infection among formula-fed babies is greater than among those who are breastfed. All formula, once mixed with cool boiled water, must be refrigerated and should not be kept for longer than 24 hours. Any formula left over after a feed should be discarded rather than reheated and used at a later stage.

Incorrect concentration
Powdered formulas should be measured accurately using the scoop provided in the tin for that particular formula. Unfortunately not all scoops are the same size, and difficulties arise when a brand is changed and the wrong scoop size is used. The instructions given on the tin should be followed for each brand of formula.

Overfeeding and obesity
Incorrect preparation of feeds can lead to high energy intakes if too much milk powder is added to water. Formula-fed babies are often encouraged to drain the bottle, thus exceeding their energy requirements.

Constipation
Whereas the problem of constipation is rare in the breastfed infant, parents of formula-fed infants often complain that ‘constipation’ is a problem. There is, however, a need to clarify what is meant by constipation. Infants who pass an infrequent, but soft, stool do not fall into this category. Constipation in infants can be defined as a delay or difficulty in defecation, present for two or more weeks. Constipation can be caused by a formula that is too concentrated, or a lack of fluid.

Cow’s milk protein intolerance
During an allergic reaction, the immune system mistakenly believes that a harmless substance, in this case a food item, is harmful. In its attempt to protect the body, it creates specific IgE antibodies to that food. The next time the individual eats that food, the immune system releases large amounts of chemicals and histamines in order to protect the body. These chemicals trigger a cascade of allergic symptoms that can affect the respiratory system, gastrointestinal tract, skin, or cardiovascular
system. The incidence of cow’s milk allergy, one of the most common types of allergies, is approximately 2% in the paediatric population. The mechanism for this is probably that one or more intact proteins from cow’s milk, for example casein or β-lactoglobulin, pass through the gut and induce an allergic reaction. Allergic reactions to cow’s milk protein also occur much more commonly in babies born into atopic families. Allergic reactions can occur in infants when solids are introduced too early, due to intestinal immaturity. Symptoms following an allergic reaction include vomiting, diarrhoea, abdominal pain, rhinitis, asthma, a topic dermatitis, urticaria and anaphylactic reactions. There is dramatic improvement on a cow’s-milk-free diet.

Relationship of infant feeding to development of allergies
There has been some concern that types of infant feeding may be related to the development of allergies in older children. A recent review assessed the impact of early feeding (breast milk and/or cow’s milk and/or formula) on development of atopic disease. The review group concluded that breastfeeding seems to protect from the development of atopic disease. The effect appears even stronger in children with atopic heredity (van Odijk et al. 2003).

Docosahexaenoic acid (DHA) and arachidonic acid (ARA) supplementation
Healthy term infants who are not breastfed may need long-chain polyunsaturated fatty acids (LCPUFA) in their formula. A ‘meta-analysis’ assessed the effect visual acuity at four months. The two main dietary determinants of DHA status are the linolenic acid and the preformed DHA consumed. The authors concluded that there is a significant relation between the total DHA equivalents provided and effectiveness as defined by visual acuity measurements at four months of age (Uauy et al. 2003).

Infant feeding: six months

Need for foods other than milk

Please now read sub-sections ‘2.1.4.2 Infants’ (p. 30), ‘2.2.6.2 Infants’ (p. 44), ‘2.3.4.2 Infants’ (p. 50), ‘2.4.4.2 Infants’ (p. 60), ‘2.5.4.2 Infants’ (p. 67), and ‘2.6.4.2 Infants’ (p. 72) of section ‘2.1 Eat a wide variety of nutritious foods’ in the draft Australian Dietary Guidelines.

By about six months of age infants are physiologically able to handle foods other than breast milk or infant formula. Infants are able to move finely mashed and smooth food from the front to the back of the mouth for swallowing. Before this time the presence of the extrusion reflex causes infants to reject food placed on the tip of the tongue. The foetal iron stores accumulated during pregnancy supply the majority of iron required during the first six months. Infants will become susceptible to iron deficiency if appropriate foods are not included in the diet after this time. It is during this time that iron fortified infant cereals can be introduced, to maintain body iron stores, followed by fruits and vegetables. Foods should be introduced one at a time in order to determine if the infant is having an allergic reaction. Maturation of the kidneys will mean the infant will be able to tolerate
larger osmolar loads of protein and electrolytes. Infants are also able to digest and absorb cereal products. Introduction of solid foods is an educational process, breast milk or infant formula will continue to meet the majority of the infant’s nutritional requirements.

**Infant feeding: From around 6–9 months**

Infants at this age should be encouraged to take foods, which have a few lumps in them and are of a coarser texture. Foods that can be chewed such as crusts of bread, pieces of fruit and strips of meat are suitable foods. The draft Dietary Guidelines note that there is no particular order or rate for the introduction of new foods but recommend that the early foods be iron rich. Breast milk or infant formula continues to be the main source of nutrition for the infant until about nine months of age.

**Infant feeding: 9–12 months**

It is generally recommended that salt and sugar should not be added to foods prepared for infants. The salt merely adds to the solute load that has to be excreted, while the sugar adds energy without other nutrients. It is has also been suggested that the preference for sweet and salty foods can be influenced by the early introduction of these flavours (Beauchamp et al. 2009).

**Commercial tinned and bottled baby foods**

Commercially prepared baby foods are widely available in supermarkets. They have a variety of different food and nutrient contents. The varieties targeted to infants aged six months have a homogenised appearance and a smooth texture, whereas the varieties for 6 to 12-month-old infants are a mixture of homogenate and discrete food particles.

Advantages of commercially prepared baby foods:

- Parents find commercial baby foods easy and convenient to use.
- Microbiologically sound.
- Nutritionally sound with no added salt or sugar.

Disadvantages of commercially prepared baby foods:

- They are an expensive way of purchasing nutrients.
- They are limited in texture.
- The homogenised content is bland and does not differentiate flavours; hence the infant does not learn to recognise separate tastes.
Recommended dietary patterns for infant feeding

The draft Australian Guide to Healthy Eating (NHMRC 2011b) includes recommended food patterns for infants aged 7–12 months as displayed in Table 3.3. These recommended patterns meet the RDIs of iron and zinc for infants and the AIs of all other nutrients (for infants, only iron and zinc have RDIs; all other nutrients have only AIs as reference values). However, as nutritional requirements vary between individuals, the weight gain and development of each infant should be used as an indication of whether their dietary intake is adequate or excessive, and the recommended patterns modified as appropriate. The serving sizes recommended for infants are smaller than that recommended for toddlers and adults.

Table 3.3 Recommended food patterns that will provide nutrient needs for infants aged 7–12 months*

<table>
<thead>
<tr>
<th>Food</th>
<th>Serving size</th>
<th>Recommended serves per day</th>
<th>Recommended serves per week</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vegetables and legumes/beans</td>
<td>20 g</td>
<td>1½ – 2</td>
<td>10 – 14</td>
</tr>
<tr>
<td>Fruit</td>
<td>20 g</td>
<td>½</td>
<td>3 – 4</td>
</tr>
<tr>
<td>Grain (cereal) foods</td>
<td>40 g bread equivalent</td>
<td>1½</td>
<td>10</td>
</tr>
<tr>
<td>Infant cereal (dried)</td>
<td>20 g</td>
<td>1</td>
<td>7</td>
</tr>
<tr>
<td>Lean meat, poultry, fish, eggs</td>
<td>30 g</td>
<td>1</td>
<td>7</td>
</tr>
<tr>
<td>Breast milk or formula</td>
<td>600 ml</td>
<td>1</td>
<td>7</td>
</tr>
<tr>
<td>Yoghurt, cheese and/or</td>
<td>20 ml yoghurt or 10 g cheese</td>
<td>1½</td>
<td>3 – 4</td>
</tr>
<tr>
<td>alternatives</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

* There is also an allowance for 5 g of unsaturated spreads or nut/seed paste. Whole nuts and seeds are not recommended for infants due to the risk of choking.

(NHMRC 2011)

PRINT READING

Please read Golley (2010), ‘Understanding the role of infant and toddler nutrition on population health: epidemiological resources in Australasia.

Please read this paper and note the role of diet during infancy on health outcomes.
Common nutritional concerns

Iron deficiency anaemia

Children with iron deficiency anaemia in infancy are at risk of developmental delay during childhood.

Introduction of fruit juice

Please now read sub-section ‘3.3.4.1 Infants’ of section ‘3.3 Limit intake of foods and drinks containing added sugars’ in the draft Australian Dietary Guidelines, p. 91.

Fruit juice is unnecessary in the diets of infants. Excessive fruit juice consumption has been associated with dental caries, chronic diarrhoea, decline in milk intake, failure to thrive and obesity (Dennison 1996).

Vegetarian diets

Strict macrobiotic and fruitarian diets may contain insufficient levels of energy, iron, calcium and vitamin B12. It is difficult to provide sufficient nutrients and energy for an infant on a totally vegetarian diet without milk, eggs and milk products. A parent should be appropriately counselled by an expert in nutrition, for example a dietician who specialises in paediatrics, before they consider placing an infant on a vegetarian diet.

Text questions

1. Explain what is meant by renal solute load and how it may be estimated.
2. Under what circumstances, other than high environmental temperatures, might a healthy breastfed baby become dehydrated?
3. Plot the growth of the following two children using the growth curves in Appendix 1 on DSO. You will need to work out their age at different dates to be able to plot individual weights and lengths. Give an opinion of the growth of each child. Why is it necessary to weigh an infant so regularly (every few months)?
Child 1  Ronny Bloggs  Date of birth: 15/9/98

<table>
<thead>
<tr>
<th>Date</th>
<th>Weight (kg)</th>
<th>Length (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>15/9/98</td>
<td>3.5</td>
<td>50</td>
</tr>
<tr>
<td>29/9/98</td>
<td>3.3</td>
<td>52</td>
</tr>
<tr>
<td>22/10/98</td>
<td>3.6</td>
<td>55.5</td>
</tr>
<tr>
<td>15/11/98</td>
<td>4.2</td>
<td>57</td>
</tr>
<tr>
<td>13/3/99</td>
<td>7.0</td>
<td>68</td>
</tr>
<tr>
<td>29/6/99</td>
<td>8.2</td>
<td>71.5</td>
</tr>
<tr>
<td>18/9/99</td>
<td>9.2</td>
<td>76</td>
</tr>
</tbody>
</table>

Child 2  Edwina Montgomery  Date of birth: 1/1/00

<table>
<thead>
<tr>
<th>Date</th>
<th>Weight (kg)</th>
<th>Length (cm)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1/1/00</td>
<td>3.6</td>
<td>50</td>
</tr>
<tr>
<td>14/1/00</td>
<td>3.8</td>
<td>51.5</td>
</tr>
<tr>
<td>31/1/00</td>
<td>4.6</td>
<td>53.5</td>
</tr>
<tr>
<td>9/3/00</td>
<td>5.5</td>
<td>59.5</td>
</tr>
<tr>
<td>16/6/00</td>
<td>7.6</td>
<td>66</td>
</tr>
<tr>
<td>1/9/00</td>
<td>9.9</td>
<td>71</td>
</tr>
<tr>
<td>1/2/01</td>
<td>12.2</td>
<td>75</td>
</tr>
</tbody>
</table>

4 Why is it customary to express the energy and protein requirements of infants per kilogram of body weight, rather than per day, as for other age groups?

5 Recommended energy intakes for infants expressed per kilogram of body weight are lower at 6–9 months of age than at 1–2 months or at 11–12 months of age. What is believed to be the basis for this pattern of energy needs?

6 On average, how much energy per kilogram of body weight does 850 mL of human milk provide for an infant on the 50 percentile for weight at age six months?

7 Explain why breast milk, which contains only 6–7% of energy from protein, is adequate for the rapid rate of growth which occurs in early infancy.

8 What are the major advantages of a diet providing a higher proportion of energy from fat (50–54%) than from carbohydrate (40–43%) in infancy?

9 Why is a distinction sometimes made between recommended intakes for breastfed and formula-fed babies in early infancy?
10 Under what circumstances are mineral imbalances most likely to arise in Australian infants?

11 Table 3.4 is the nutrient composition per 100 mL of breast milk and three unknown milks. Identify the class of each unknown milk/formula. Explain the main differences in comparison to breast milk.

Table 3.4 Nutrient composition of milk per 100 mL

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Breast milk</th>
<th>Milk 1</th>
<th>Milk 2</th>
<th>Milk 3</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Energy (kJ)</strong></td>
<td>280</td>
<td>280</td>
<td>291</td>
<td>274</td>
</tr>
<tr>
<td>Protein total (g)</td>
<td>1.3</td>
<td>1.5</td>
<td>3.5</td>
<td>1.8</td>
</tr>
<tr>
<td>lactalbumin (%)</td>
<td>60</td>
<td>18</td>
<td>18</td>
<td>soy protein isolate</td>
</tr>
<tr>
<td>casein (%)</td>
<td>40</td>
<td>82</td>
<td>82</td>
<td></td>
</tr>
<tr>
<td>Fat total (g)</td>
<td>4.2</td>
<td>3.6</td>
<td>3.5</td>
<td>3.2</td>
</tr>
<tr>
<td>fat source</td>
<td>linoleic acid, linolenic acid, DHA, EPA</td>
<td>oleo, coconut, soy</td>
<td>milk fat</td>
<td>oleo, coconut, soy, oleic</td>
</tr>
<tr>
<td>Carbohydrate total source</td>
<td>7.0 lactose</td>
<td>7 lactose</td>
<td>6.4 lactose</td>
<td>6.9 Corn syrup solids (75%) sucrose (25%)</td>
</tr>
<tr>
<td>Renal solute load (mOsm/L)</td>
<td>77</td>
<td>96</td>
<td>226</td>
<td>110</td>
</tr>
<tr>
<td>Calcium (mg)</td>
<td>35</td>
<td>46</td>
<td>101</td>
<td>60</td>
</tr>
<tr>
<td>Phosphorus (mg)</td>
<td>15</td>
<td>36</td>
<td>92</td>
<td>42</td>
</tr>
<tr>
<td>Iron (mg)</td>
<td>0.026</td>
<td>1.2</td>
<td>trace</td>
<td>1.2</td>
</tr>
<tr>
<td>Sodium (mg)</td>
<td>15</td>
<td>18</td>
<td>37</td>
<td>20</td>
</tr>
<tr>
<td>Potassium (mg)</td>
<td>60</td>
<td>62</td>
<td>146</td>
<td>70</td>
</tr>
</tbody>
</table>

12 A parent is making a bottle of S26 infant formula, usually made with 240 mL water and four level scoops of powder. Unfortunately this parent is confused and is only adding in three instead of four scoops. What are the consequences of this error if this practice continues for a few months?

13 A father is concerned that his 8-month-old daughter is not consuming sufficient protein. The father has decided to stop giving fruit and vegetables because he has heard these foods are low in protein. Instead he is giving more dairy foods, meats and chicken. In addition, he is adding a protein powder to the infant formula. Comment on the dietary practices of this father.

14 The local childcare centre has made a change to the feeding practices in the babies' room (children aged six weeks to one year) and is asking for your expert opinion. The childcare centre has replaced infant rice cereal with Weet-Bix for the infants who are aged six months. In addition, they are also introducing a bottle of orange juice with the Weet-Bix. Discuss the implications of this change. (Hint: it would be useful to look at the nutrition
composition of both Weet-Bix and rice cereal. You can obtain this information from the supermarket.)

15 Rickets is known to occur in exclusively breastfed infants of Muslim mothers. It is typical for Muslim women to cover their entire body with clothing (except for their face) and to spend long periods indoors. Explain how rickets would occur in this situation.

References


Food and Nutrition Board: Institute of Medicine 2002, Dietary reference intakes for energy, carbohydrates, fiber, fat, fatty acids, cholesterol, protein and amino acids, National Academy Press, Washington, DC.


TOPIC 4

Nutrition during the preschool years

PREPARED BY THE UNIT TEAM

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Introduction

The preschooler includes children aged one to five years, whereas the toddler is a term often used and includes children aged one to three years of age. This age group is characterised by a period of slower growth and the emergence of feeding skills that allow independence in eating. In a similar way to infants, preschool aged children continue to have high energy and nutrient needs to meet their requirements for growth and development.

Learning objectives

At the conclusion of this section you should have an understanding of and be able to describe the following about preschool aged children:

- growth patterns
- emerging developmental characteristics
- nutritional requirements
- factors influencing food choices
- common nutritional concerns
- venues where preschool aged children consume food
- sources of nutrition information for parents.

Learning resources

Print reading

The reading listed here is reproduced in the print readings for this unit.


Online readings

Access the readings listed here using your internet browser.


Deakin Studies Online (DSO)

Access the unit site on DSO <http://www.deakin.edu.au/current-students/> for the appendices and other additional resources you can use to supplement this topic.

Growth

Growth charts are used to monitor the growth of preschool aged children, as discussed in the previous section. Excessive or inadequate intakes of energy are reflected as accelerations or decelerations in weight gain, respectively.

The rapid rate of growth observed during infancy is followed by a deceleration during the preschool aged years. A male toddler tracking along the 50th centile for both weight and length will gain 2.4 kg in weight and 12 cm in length in the second year of life.

Compare this to 7.0 kg and 25 cm gained in the first year of life for a male infant also progressing along the 50th centile.

Changes in body composition

A number of changes are observed in proportions of LBM (lean body mass), body fat, water and tissue development. There is an increase in percentage LBM and a decrease in percentage body fat. Preschool children generally become leaner as they age, although females carry more subcutaneous fat than males. Figure 4.1 shows the changes in average accretion rates for weight, FM (fat mass), FFM (fat-free mass), TBW (total body water) and BMC (bone mineral content) in boys over the first two years of life. In summary accretion rates are greatest at birth and decrease with increasing age. Expected changes in body composition are gender specific; however a similar pattern of growth is seen in girls (Ellis 2002). Figure 4.2 outlines the growth in weight and height during the first five years of life. The greatest growth rate occurs in the first year of life, where the infant will triple in body weight and height will increase by approximately 75%. After this, growth rate slows, and is similar for both boys and girls (Figure 4.2). The proportion of body weight that is water reaches adult levels by the age of 2–3 years of age. The extracellular fluid is observed to decrease while the amount of intracellular fluid increases. This shift in body fluid can be attributed to the growth of new cells. The concurrent decrease in ratio of body surface to body mass will mean the preschooler is less susceptible to dehydration. By the time the child is two, brain growth is 75% complete. Bone growth results in an increase in stature of the preschooler.
Figure 4.1  Changes in the accretion rates for Wt, FM, FFM, TBW, BMC in boys (age in months)

(Butte 2000, cited in Ellis 2002)

Figure 4.2  Rates of growth in preschool children

(Freeman et al. 1995, cited in Langley-Evans 2009, p. 125)
Emerging developmental characteristics

The preschool years are characterised by emerging developmental characteristics, many of which impact on the feeding process.

Motor skills
By 18 months of age the toddler will have well defined chewing patterns, ulna deviation of the wrist enabling them to self feed from a spoon. By the age of 24 months the toddler can lift a cup and drink from it without spillage.

Social skills
A toddler will understand the routine of mealtimes, from as early as 12 months of age. The location of meals, who participates in meals and the atmosphere around mealtimes will become evident to the toddler.

Language skills
The toddler at three years of age can construct 3–4 word sentences, which will enable them to verbally accept or reject food.

Nutritional requirements, intakes and nutritional concerns

Nutritional requirements relative to body weight are reduced when compared to infants; however, they are high when compared to adults.

Energy intake, fat intake, growth failure and obesity

Energy requirements
Two techniques can be used to determine the energy requirements of preschoolers:

1. Energy requirements can be determined by extrapolating from energy intakes of healthy children growing normally.
2. Measurement of energy expenditure using the doubly-labelled water technique. (Davies 1996)

Total energy requirements are dependent on BMR (basal metabolic rate), rate of growth, thermogenesis and levels of physical activity. BMR is primarily dependent on LBM, which varies with age but not by gender, until the children reach the age of ten years. The energy cost of growth is small and is approximately 21 kJ/g tissue gained (<10% of total energy intake). The contribution from physical activity will vary both between children and within the one child. As a general guide, if a child's growth is adequate, then it can be assumed their diet is sufficient in energy. The best method of assessing energy needs is the doubly-labelled water technique. When this method is applied over a 24-hour period, it includes estimates of metabolic response to food and the energy cost of tissue synthesis. For adults, this equates to daily energy requirements. The additional needs in infancy and childhood, in adolescence, pregnancy and lactation need to be estimated from growth velocity or weight gain equations. In the 2006 Nutrient Reference Values, the Estimated Energy Requirement for Maintenance (EERM, or actual energy...
requirement) is the dietary energy intake that is predicted to maintain energy balance (plus extra needs for pregnancy, lactation and growth) in healthy (i.e. no chronic illness) individuals or groups of individuals at current levels of body size and level of physical activity; and the Desirable Estimated Energy Requirement (DEER, or energy ‘reference value’) is the dietary energy intake that is predicted to maintain energy balance (plus extra needs for pregnancy, lactation and growth) in healthy individuals or groups of individuals of a defined gender, age, weight, height and level of physical activity consistent with good health and/or development. Use of, and distinction between, these two terms is necessary because of the various ways in which estimates of energy requirements are used and because of the risk of over-prescription of ‘desirable’ energy intakes in people who do not follow recommendations for increased physical activity.

Under the 2006 NRVs, for children and adolescents a method was used that estimates energy expenditure at any physical activity level (PAL), similar to that used in the previous Australian/New Zealand RDI (NHMRC, 1991). This approach is limited by the choice of equation (Schofield et al. 1985) used to calculate BMR. This approach involves firstly determining body weight and height for each age, gender of the group or individual. To determine actual or maintenance energy requirements (EERM), use the current body weight. To determine desirable energy requirements (DEER), the current body weight is used if it falls within the healthy weight range (i.e. Body Mass Index (BMI) in the range 18.5–24.9 kg/m² for adults or BMI centiles for children and adolescents). Where the BMI is 25.0 kg/m² or above, determine the desirable body weight by assuming a BMI of 22.0 kg/m² or in the range 18.5–24.9 kg/m² as appropriate. Next, the BMR of the group or individual is determined using indirect calorimetry, or predicted from the Schofield equations. To account for activity, the approximate PAL of the group or individual is estimated from the amount of time spent in different activities and energy expenditure by multiplying the BMR by the level of activity (PAL; expressed as a multiple of BMR). A PAL above 1.75 is considered compatible with a healthy lifestyle. To this is added an estimate of extra energy requirements for growth of 85kJ/day for 4–8 years, and 105 kJ/day for 9–18 years, using the estimates of energy content of tissue deposition, in conjunction with the 50th percentile for weight gain at various ages. The estimate of energy requirement is then corrected for the composition of the Australian/New Zealand diet.

Energy intake

Table 4.1 summarises mean energy intakes and the percentage contribution from the three macronutrients, as determined by the 2007 Children’s Nutrition and Physical Activity Survey (CNPAS) (Table 4.1). Carbohydrates provide the majority of energy for young children. Refined carbohydrates (total sugar) contribute a large proportion of energy, 24–26% for both males and females at all age groups, this is a modest decrease from the 1995 National Nutrition Survey (NNS) where total sugar intakes were 27–31% for this age range. Compared with the 1995 National Nutrition Survey, there has been a decrease in percent energy from fat (down from 32–33% total energy), and carbohydrate (down from 52%) and an increase in percent energy from protein (previously 13.9–14.3%).
### Table 4.1 Mean energy intakes and % contribution from the three macronutrients

<table>
<thead>
<tr>
<th></th>
<th>Males (2–3 years)</th>
<th>Males (4–8 years)</th>
<th>Females (2–3 years)</th>
<th>Females (4–8 years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean daily energy intake (kJ)</td>
<td>6290</td>
<td>7740</td>
<td>6043</td>
<td>7030</td>
</tr>
<tr>
<td>Mean % contribution</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Protein (%)</td>
<td>16.5</td>
<td>16.4</td>
<td>16.7</td>
<td>16.0</td>
</tr>
<tr>
<td>Fat (%)</td>
<td>30.2</td>
<td>30.4</td>
<td>30.6</td>
<td>30.5</td>
</tr>
<tr>
<td>Saturated Fat (%)</td>
<td>14.2</td>
<td>13.9</td>
<td>14.2</td>
<td>13.7</td>
</tr>
<tr>
<td>Carbohydrate (%)</td>
<td>49.3</td>
<td>49.3</td>
<td>48.7</td>
<td>49.5</td>
</tr>
<tr>
<td>Total sugar (%)</td>
<td>26.1</td>
<td>24.3</td>
<td>25.6</td>
<td>24.3</td>
</tr>
<tr>
<td>Total starch (%)</td>
<td>22.7</td>
<td>24.5</td>
<td>22.6</td>
<td>24.8</td>
</tr>
</tbody>
</table>

(Commonwealth of Australia 2008)

Table 4.2 outlines the mean per cent contribution to energy intake from the main food groups.

### Table 4.2 Percentage energy intakes from selected major food groups

<table>
<thead>
<tr>
<th>Proportion of today dietary energy from selected major food groups</th>
<th>Males (2–3 years)</th>
<th>Males (4–8 years)</th>
<th>Females (2–3 years)</th>
<th>Females (4–8 years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-Alcoholic Beverages</td>
<td>4.9</td>
<td>6.1</td>
<td>4.6</td>
<td>5.6</td>
</tr>
<tr>
<td>Cereals &amp; Cereal Products</td>
<td>22.7</td>
<td>23.0</td>
<td>21.5</td>
<td>22.0</td>
</tr>
<tr>
<td>Cereal-Based Products &amp; Dishes</td>
<td>12.1</td>
<td>14.9</td>
<td>12.1</td>
<td>16.1</td>
</tr>
<tr>
<td>Fats &amp; Oils</td>
<td>2.9</td>
<td>2.6</td>
<td>3.0</td>
<td>2.9</td>
</tr>
<tr>
<td>Fish &amp; Seafood Products &amp; Dishes</td>
<td>1.3</td>
<td>1.3</td>
<td>1.6</td>
<td>1.4</td>
</tr>
<tr>
<td>Fruit Products &amp; Dishes</td>
<td>7.7</td>
<td>5.6</td>
<td>7.8</td>
<td>6.0</td>
</tr>
<tr>
<td>Egg Products &amp; Dishes</td>
<td>0.6</td>
<td>0.9</td>
<td>0.8</td>
<td>0.8</td>
</tr>
<tr>
<td>Meat, Poultry &amp; Game Products &amp; Dishes</td>
<td>8.4</td>
<td>10.4</td>
<td>8.7</td>
<td>9.4</td>
</tr>
<tr>
<td>Milk Products &amp; Dishes</td>
<td>24.4</td>
<td>17.7</td>
<td>23.9</td>
<td>17.2</td>
</tr>
<tr>
<td>Vegetable Products &amp; Dishes</td>
<td>4.8</td>
<td>5.1</td>
<td>5.2</td>
<td>5.8</td>
</tr>
<tr>
<td>Legume &amp; Pulse Products &amp; Dishes</td>
<td>0.4</td>
<td>0.5</td>
<td>0.5</td>
<td>0.4</td>
</tr>
<tr>
<td>Snack Foods</td>
<td>1.4</td>
<td>3.1</td>
<td>1.7</td>
<td>2.6</td>
</tr>
<tr>
<td>Sugar Products &amp; Dishes</td>
<td>1.6</td>
<td>1.8</td>
<td>1.5</td>
<td>1.8</td>
</tr>
<tr>
<td>Confectionery &amp; Cereal Bars</td>
<td>3.1</td>
<td>4.5</td>
<td>3.1</td>
<td>4.9</td>
</tr>
</tbody>
</table>

(Commonwealth of Australia 2008)
Cereal and dairy foods are a significant source of energy for the preschooler, with the contribution from dairy foods decreasing with increasing age. Of interest is the combined contribution of non-alcoholic beverages (note, this excludes milk), confectionary and cereal bars, which contribute around 8–10% of total energy. These foods can be considered non-core foods yet represent a reasonable proportion of daily energy intake.

**Fat: low fat and high fat diets**

**ONLINE READING**

Please now read sub-sections ‘2.5.4.2 Infants’ (p. 67) and ‘2.5.4.3 Children and adolescents’ (p. 67) in section ‘2.5 Milk, yoghurt, cheese and/or alternatives’ of the draft *Australian Dietary Guidelines*.

Fat continues to be an integral component of the diet in order to support the growth requirements of preschoolers and to provide essential fatty acids, particularly omega-3 fatty acids.

For children under the age of two years, full fat dairy products are recommended and 40% of energy should be derived from fat. Whereas for children aged two to five years, reduced fat milks are recommended but skim milk should be avoided and the recommended fat intake levels reduce to 30% of energy.

Conversely, excessively high fat diets are not recommended as they can lead to energy intakes exceeding energy expenditure. The mean per cent contribution of fat to total energy intake was 30% for both males and females (Commonwealth of Australia 2008).

**Obesity**

A chronic imbalance between energy intake and energy expenditure will lead to a state of positive energy balance and eventually excess weight gain. Obesity is defined when weight is greater than two percentiles above that of the percentile for height, as plotted on the growth curves. Factors that can lead to positive energy balance include high energy intakes, particularly resulting from high fat diets, and insufficient activity levels. The consequences of obesity are both metabolic and psychological in basis. Obesity as a problem emerges during the preschool years and continues to be a health issue throughout every stage of the lifespan.

**Protein**

Dietary protein is needed for tissue maintenance, growth of body tissue and changes in body composition. In comparison to infants, the amount of protein required for growth is relatively small compared to the amount of protein required for tissue maintenance. It should also be noted that growth occurs in spurts and that protein requirements will be higher during these periods of rapid growth. The accumulation of body protein reaches adult levels (18–19% of total body weight) by the age of four years. The decrease in protein requirements, as measured in grams of protein per kilogram of body weight, from 1–3 to 4–7 years is due to the decrease in the rate of growth during this period. In general, mean daily protein intakes exceed estimated requirements.
Fibre and fluid intake, constipation and diarrhoea

Fibre
Adequate consumption of dietary fibre is necessary for normal bowel actions in preschoolers, and may also reduce the future risk of developing cardiovascular disease, some cancers and type 2 diabetes. The 2006 adequate intake (AI) for fibre is 14 g/day for children aged 1–3 years, which increases to 18 g/day for those aged four and eight years. These levels were devised using data from the 1995 Australian National Nutrition Survey and the New Zealand Dietary Survey. The 2007 CNPAS results indicate fibre intakes were above this AI; 16.7 and 19.2 g/day for boys aged 2–3 and 4–8 years, respectively and 15.5 and 18.0 g/day for girls aged 2–3 and 4–8 years, respectively.

Fluid
The preschooler is less susceptible to dehydration when compared to an infant; however, fluid requirements are still high when compared to adults. Young children should be encouraged to drink water when they are thirsty. Sweetened fluids include 100% fruit juice, fruit juice drinks, cordial, cordial made with fruit juice, soft drinks, flavoured mineral waters and vitamin C syrups and should be minimised in the diets of young children. Excessive intake of sweet drinks can displace a child's appetite for more nutritious foods, increase the risk of developing dental caries and is associated with childhood diarrhoea (NHMRC, 1995). A nutrition needs assessment conducted in the Melbourne’s Western Metropolitan Region found that 81% of children aged 2–4 years of age consumed one cup or more of juice per day in a childcare setting, with 20% of these children consuming more than two cups (Department of Human Services 1997). A study by Petter et al. (1995) reported that sweetened fluids in comparison to plain water were more popular in young children, and that 15% of the preschooler group consumed just under 50% of their recommended daily intake from sweet drinks. It has also been suggested from this study that a liking for sweetened fluids can be developed at an early age.

Constipation
Constipation is defined as the passing of a hard bowel motion often with pain and discomfort (Department of Human Services 2002). Thirty-four per cent (2–4 year age group) and 32% (4–6 year age group) of parents indicated their child had had constipation (Department of Human Services 1997). These results must be interpreted carefully as parents will often believe their child has constipation based on the number of bowel actions passed per day and not on the true definition of constipation. The incidence of constipation may actually be less than what was found in this needs assessment. Constipation can be caused by an inadequate intake of fibre and/or fluid, or poor toileting practices.

Diarrhoea
Diarrhoea was reported to occur in 45% of 2- to 4-year-old children (n = 130) and 36% of 4- to 6-year-old children (n = 126) surveyed in the needs assessment (Department of Human Services 1997). It is not known how many incidents of diarrhoea occurred in these children. Excessive consumption of fruit juice and food that has been contaminated with bacteria can cause diarrhoea. Lifshitz et al. (1992) has reviewed the role of juice carbohydrate in the aetiology of chronic nonspecific
diarrhoea in children. Sorbitol and fructose, two carbohydrates found in fruit juice, can be malabsorbed in the gastrointestinal tract leading to diarrhoea.

**Micronutrient requirements**

The following will discuss selected vitamins and minerals, which are of particular importance in this age group.

**Calcium**

Please now read sub-section ‘2.5.4.3 Children and adolescents’ (p. 67) in section ‘2.5 Milk, yoghurt, cheese and/or alternatives (mostly reduced fat)’ from the draft *Australian Dietary Guidelines*.

Between the ages of 1–7 years approximately 100 mg/day of calcium is accumulated in bone. Recommended dietary intakes of calcium are high (500 and 700 mg per day for children aged 1–3 and 4–8 years, respectively), as measured in mg of calcium per kilogram of body weight, when compared to adults. Achieving adequate intakes of calcium can be difficult as calcium is not widely distributed in the food supply, the absorption of calcium is inefficient at times of high requirements, the bioavailability of calcium from the diet is variable and there is continuous excretion of calcium in the urine, faeces and sweat. Average calcium intakes were 829.8, 841.8 mg/day for boys aged 2–3 and 4–8 years, respectively and 780.2 and 747.4 mg/day for girls aged 2–3 and 4–8 years, respectively. Between 93–99% of boys and 99% of 2- to 3-year-old girls obtained the EAR (estimated average requirement) for calcium; however, only 85% of 4- to 8-year-old girls had intakes above the EAR.

**Iron**

Preschoolers have high requirements for iron because of their high growth rates and rapidly expanding blood volume. After infancy, 0.5 mg of iron needs to be accumulated each day until the growth period ceases. The 2006 RDI recommends 11 mg/day for infants 7–12 months and this is reduced to 9 mg/day aged 1–3 years and increases again to 10 mg/day between four and eight years. These NRVs are based on absorption of 10% of dietary iron. Intake data from the 2007 CNPAS indicate intakes of 8.3 and 10.5 mg/day for boys aged 2–3 and 4–8 years, respectively and 7.8 and 9.2 mg/day for girls aged 2–3 and 4–8 years, respectively. Between 99–100% of the population met the EAR for this important nutrient. The potential consequences of iron deficiency anaemia include delayed cognitive and physical development, and decreased resistance to infection (Booth & Aukett 2003). Factors that can lead to the development of iron deficiency anaemia include delayed introduction of iron containing foods during infancy followed by poor dietary habits during the preschool years, including insufficient intake of haem iron containing foods. Overconsumption of cow’s milk, which is a poor source of iron, and fruit juices, can displace the preschooler’s appetite for iron containing foods. To prevent iron deficiency anaemia young children should be given both haem and non-haem sources of iron. The absorption of iron in the gastrointestinal tract will be increased if foods containing vitamin C and haem iron are consumed at the same time. Nguyen et al. 2004 assessed the iron status and dietary iron intake of six
Vietnamese children in Australia and found high rates of non-anaemic iron deficiency (NAID) and as iron deficiency anaemia (IDA). Dietary factors associated with iron IDA were examined by Karr and co-workers (2001) in Australian-born children of Arabic background in central Sydney. Overall, 6% had iron deficiency anaemia, another 9% were iron deficient without anaemia, and 23% were iron depleted. The main nutritional risk factor for iron depletion was a daily intake of greater than 600 mL of cow’s milk.

Dental caries

Dental plaque, found on tooth surfaces, consists of bacteria, extracellular polysaccharides, proteins of salivary and dietary origin, and lipids. Without effective oral hygiene, plaque can cover all surfaces of the teeth. Dental caries are formed when carbohydrates, predominantly mono and disaccharides, are metabolised into acids by plaque bacteria. These acids cause the plaque pH to fall and can cause the tooth structure to dissolve. Plaque is also produced from the metabolism of carbohydrates by bacteria. Plaque causes bacteria to adhere to teeth and reduce the acid-neutralising effect of saliva.

Sucrose is highly cariogenic, whereas glucose and lactose tends to be less cariogenic. These monosaccharides and disaccharides can rapidly diffuse through dental plaque to become available for bacterial fermentation. Dental caries formation is also influenced by the rate at which carbohydrates clear from the oral environment. Sugars in solution (e.g. fruit juice) are less harmful than solids (e.g. confectionery) because they are cleared from the mouth more rapidly. However, if sweetened fluids are consumed over an extended period, then they are likely to cause caries. Sticky foods (e.g. honey, toffee, dried fruit) are generally more cariogenic as they are in contact with the tooth surface for longer periods of time. Milk consumed over extended periods, such as children consuming milk from a bottle while they sleep, is likely to lead to the formation of dental caries.

Vegetarian diets

The American Dietetic Association recently published a position statement on vegetarian diets and health (American Dietetic Association 2009). They concluded that an appropriately planned vegetarian diet can be nutritionally adequate and provide health benefits at all stages of the lifespan.

Vegetarian diets exclude meat, chicken, fish or any products containing these foods. Lacto-vegetarians exclude eggs in addition to meat, chicken and fish. The lacto-ovo vegetarian diet includes both dairy and eggs, and vegans exclude all
animal products including dairy foods and eggs. A macrobiotic diet is predominantly based on a vegan diet and consists of wholegrain cereals, vegetables and pulses and small amounts of seaweed, fermented foods, nuts, seeds and fruits. The macrobiotic diet is typically devoid of animal products.

Preschool aged children need to consume energy and nutrient dense foods at regular intervals during the day to support their requirements for growth and development. Foods that are generally included in a vegetarian diet have low energy content, and this may be a concern for young children following these sorts of diets. A vegan diet will be especially low in energy due to the absence of milk, which is a significant source of fat and therefore energy in the diets of young children.

Lacto-ovo children have growth rates similar to that of non-vegetarian children, although poor growth has been observed in those following very restricted vegetarian diets (American Dietetic Association 2009). Children reared on vegan and macrobiotic diets have lower growth rates in the first five years of life, but catch up growth occurs by the age of approximately 10 years (Sanders & Reddy 1994). These problems can be avoided by offering energy and nutrient dense foods (e.g. full fat soy drink, tofu and vegetables cooked with oil, tahini spread) and protein foods that contain a range of essential amino acids.

In general, if the vegetarian diet contains adequate energy and a variety of plant foods, then protein requirements are likely to be met. Animal proteins contain sufficient levels of all nine essential amino acids. Most plant proteins, except for soy bean, lack one or more of the essential amino acids. A vegan eating a variety of plant products (e.g. cereals, legumes, nuts, seeds, vegetables) would consume sufficient quantities of essential amino acids. If that person was consuming soy products, e.g. soy drink, they would certainly have a sufficient intake of essential amino acids. There would be concern if the diet was very restricted and was limited to 1–2 food sources of protein e.g. corn or rice only. This is likely to be observed in Third World countries during times of famine, and would rarely occur in a developed country. Adequate levels of the essential amino acids can be achieved by consuming the following diet: 60% from grains, 35% from legumes and 5% from leafy greens. Micronutrients that may to be inadequate in a poorly planned vegetarian diet include vitamin B12, iron, calcium and zinc. Iron intake is of particular concern as the majority of iron in plant foods is in the non-haem iron form, and their bioavailability is lower due to the presence of phytates, which can reduce the absorption of iron.

Food requirements for preschoolers

The preceding discussion looked at requirements in terms of the NRVs. This information may be useful for nutritionists, but is often meaningless for the lay population. Food groups translate this information into requirements expressed in household servings. The draft 2011 Australian Guide to Healthy Eating (NHMRC 2011) updated the 1998 recommendations for children aged 4-7 years old (now grouped as children aged 4-8 years) and introduced recommendations for toddlers. These food patterns are summarised in Table 4.3. For these age groups, recommended serving sizes are the same as for adults, although toddlers may eat less in one sitting but eat more frequently.
Food groups are only a guide to the requirements of young children. For example, heavier and more active toddlers may require more food when compared to lighter and more sedentary toddlers. Ultimately, appetite should be the guide to the amounts of food that toddlers or preschoolers should eat. Children should also be offered foods with a range of textures, such as soft and crunchy foods, to develop chewing skills.

Table 4.3 Food requirements for toddlers and young children

<table>
<thead>
<tr>
<th>Food group</th>
<th>Serve definition</th>
<th>Recommended number of serves per day</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Toddlers 13-23 months</td>
<td>Boys and girls 2-3 years</td>
</tr>
<tr>
<td></td>
<td>Boys 4-8 years</td>
<td>Girls 4-8 years</td>
</tr>
<tr>
<td>Vegetables and legumes / beans</td>
<td>1/2 cup cooked vegetables (75 g)</td>
<td>2 – 3</td>
</tr>
<tr>
<td></td>
<td>1 cup salad vegetables</td>
<td>2½</td>
</tr>
<tr>
<td></td>
<td>1 small potato</td>
<td>4½</td>
</tr>
<tr>
<td></td>
<td>½ cup cooked dried or canned beans or lentils, no added salt</td>
<td>4½</td>
</tr>
<tr>
<td>Fruits</td>
<td>1 medium piece of fresh fruit (150 g)</td>
<td>½</td>
</tr>
<tr>
<td></td>
<td>30 g dried fruit (e.g. 4 dried apricot halves)</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>1 cup canned fruit (150 g)</td>
<td>1½</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1½</td>
</tr>
<tr>
<td>Grain (cereal) foods</td>
<td>1 slice of bread or ½ a medium roll or flat bread (about 40 g)</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>½ cup cooked rice, pasta, noodles</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>½ cup cooked porridge or polenta, 2/3 cup breakfast</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>cereal flakes (30 g) or ½ cup museli</td>
<td>4</td>
</tr>
<tr>
<td>Lean meat and poultry, fish, eggs, nuts and seeds, and legumes / beans</td>
<td>65 g cooked lean red meats or ½ cup of lean mince, 2 small chops</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>80 g cooked poultry</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td>100 g cooked fish fillet or 1 small can of fish</td>
<td>1½</td>
</tr>
<tr>
<td></td>
<td>2 large eggs</td>
<td>1½</td>
</tr>
<tr>
<td></td>
<td>170 g tofu</td>
<td>1½</td>
</tr>
<tr>
<td>Milk, yoghurt, cheese and/or alternatives</td>
<td>250 mL cup of milk</td>
<td>1 – 1½</td>
</tr>
<tr>
<td></td>
<td>200 g tub of yoghurt</td>
<td>1½</td>
</tr>
<tr>
<td></td>
<td>40 g or 2 slices of cheese</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>120 g ricotta cheese</td>
<td>1½</td>
</tr>
<tr>
<td>Additional serves from the five food groups or discretionary choices for those who are taller or more active</td>
<td>a</td>
<td>0 – 1</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0 – 2½</td>
</tr>
<tr>
<td></td>
<td></td>
<td>0 – 1</td>
</tr>
</tbody>
</table>

* Toddlers aged 13-23 months have an allowance for 7-10 g of unsaturated spreads, oils, or nut/seed pastes; whole nuts are not recommended due to the risk of choking

(NHMRC 2011)
In addition to looking at macro and micronutrient intakes, the CNPAS provides the opportunity to determine the level of adherence to the basis of the draft Australian Guide to Healthy Eating (2011), the ‘foundation diet’ that was designed to provide the estimated energy requirements and meet the RDIs of 10 key nutrients for the shortest and most sedentary individuals in each age and gender group. Table 4.4 outlines the dietary changes required for preschool-aged children to meet the foundation diet models. Table 4.5 summarises the proportion of children meeting the 2003 Australian Guide to Healthy Eating based on CNPAS data. It is clear from these tables that considerable effort is required to boost the understanding to the importance of a healthy diet early in the lifespan to promote optimal nutrition.

Table 4.4  Changes to dietary patterns required for children to meet the ‘foundation diet’ underlying the draft 2011 Australian Guide to Healthy Eating

<table>
<thead>
<tr>
<th>Required changes to dietary patterns</th>
<th>Ages 2-3 years</th>
<th>Ages 4-8 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total vegetables</td>
<td>90% more</td>
<td>144% more</td>
</tr>
<tr>
<td>Legumes</td>
<td>320% more</td>
<td>320% more</td>
</tr>
<tr>
<td>Fruit</td>
<td>10% less</td>
<td>35% more</td>
</tr>
<tr>
<td>Total cereals</td>
<td>81% more</td>
<td>24% more</td>
</tr>
<tr>
<td>- Wholegrain or high fibre cereals</td>
<td>159% more</td>
<td>164% more</td>
</tr>
<tr>
<td>- Refined or low fibre cereals</td>
<td>3% less</td>
<td>28% less</td>
</tr>
<tr>
<td>Red meats</td>
<td>35% more</td>
<td>70% more</td>
</tr>
<tr>
<td>Other meats and alternatives</td>
<td>50% more</td>
<td>84% more</td>
</tr>
<tr>
<td>Total dairy</td>
<td>25% more</td>
<td>19% more</td>
</tr>
<tr>
<td>- High/medium fat dairy</td>
<td>89% less</td>
<td>77% less</td>
</tr>
<tr>
<td>- Low fat dairy</td>
<td>277% more</td>
<td>373% more</td>
</tr>
</tbody>
</table>

(Dietitians Association of Australia 2011)
**Table 4.5** Proportion of children meeting the serve recommendations of the 2003 Australian Guide to Healthy Eating

<table>
<thead>
<tr>
<th>Dietary Guideline</th>
<th>Measurement criteria</th>
<th>All children 2–3 years</th>
<th>All children 4–8 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Encourage, support breastfeeding</td>
<td>Ever been breast fed</td>
<td>93</td>
<td>89</td>
</tr>
<tr>
<td>Eat plenty of fruits</td>
<td>≥1–3 serves/d excluding juice</td>
<td>(68)*</td>
<td>61</td>
</tr>
<tr>
<td></td>
<td>≥1–3 serves/d including juice</td>
<td>(90)</td>
<td>93</td>
</tr>
<tr>
<td>Eat plenty of vegetables</td>
<td>≥2–4 serves/d excluding potatoes</td>
<td>(5)</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>≥2–4 serves/d including potatoes</td>
<td>(14)</td>
<td>22</td>
</tr>
<tr>
<td>Eat plenty of cereals</td>
<td>≥3–4 serves/day</td>
<td>(5)</td>
<td>20</td>
</tr>
<tr>
<td>Include lean meat, fish, poultry and/or alternatives</td>
<td>%&gt; EAR for protein, iron, zinc</td>
<td>99–100</td>
<td>100</td>
</tr>
<tr>
<td>Include milks, yoghurts, cheese and/or alternatives</td>
<td>%&gt; Calcium EAR</td>
<td>99</td>
<td>89</td>
</tr>
<tr>
<td>Choose water as a drink</td>
<td>%&gt;fluid AI</td>
<td>53</td>
<td>54</td>
</tr>
<tr>
<td>Limit saturated fat and moderate total fat intake</td>
<td>&lt;10% energy from saturated fat</td>
<td>16</td>
<td>19</td>
</tr>
<tr>
<td>Choose foods low in salt</td>
<td>&gt;sodium AI</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Consume only moderate amounts of sugars</td>
<td>%&lt;20% energy from total sugars</td>
<td>21</td>
<td>29</td>
</tr>
</tbody>
</table>

*no national recommendations or guidelines available for this age group, percentage in brackets ( ) calculated based on recommendations for the 4–7 year olds.

(Adapted from Commonwealth of Australia 2008)

**Sources of nutrition information**

The preschool years are a time when parents will seek out information about the nutritional requirements for young children. In the needs assessment conducted by Graham et al. (2000), parents were asked to indicate the five most common sources of nutrition information. It was found that parents with children aged 2–4 years predominantly sought information from family (70%), friends (43%) and childcare centres (9%). Childcare centre staff themselves obtained nutrition information from newspapers/magazines and brochures from food companies. This raises concerns about the quality and inconsistency of nutrition information obtained by parents.

**Venues where preschoolers consume food**

The number of families with two working parents is steadily rising and this will mean that more and more children will be cared for outside the family home. Childcare centres can provide full time care for a child, which can be up to ten
hours a day and five days per week. This will have significant implications on the nutritional intake of young children. Childcare centres may supply all food and fluids based on 50% of the NRVs for all macro and micronutrients. Alternatively, parents are requested to provide all food for their child. Family day care is where a child is cared for by an adult in his or her own home. Childcare centres and family day care arrangements are usually coordinated by local councils. Other care arrangements can also include grandparents and family friends. It is possible that one child may have three different care arrangements during the week. This may have implications on how the child perceives mealtimes if each carer has a different set of values and beliefs.

**TEXT QUESTIONS**

1. Why are preschoolers less vulnerable to dehydration when compared to infants? Consider the physiology of the preschooler in your response.

2. Previous national recommendations for children's energy intakes were based on recorded energy intakes in apparently healthy populations. What are the limitations of this approach and how does using the doubly labelled water method overcome these?

3. The energy expenditure of two children is compared by the method of doubly-labelled water. The children are the same height and weight; however, the energy expenditure of one child is 20% greater than that of the second child. What could explain this difference?

4. Why do the 2006 Nutrient Reference Values for energy for children from the age of three years have such a wide range of values compared to those less than three? When interpreting dietary energy calculated from food records and comparing this to recommended energy intakes, what issues will you need to consider?

5. Calculate the fat intake in g/day for a 2-year-old boy who is on the 50th centile for weight and height. What contribution does three serves of full fat dairy products have to fat intake? How would a change from full fat to low fat dairy products affect fat intake?

6. Explain the mechanism by which fructose, a carbohydrate found in fruit juice, can lead to diarrhoea.

7. Why is iron an important micronutrient in the diets of preschoolers? Can iron deficiency anaemia lead to growth failure?

8. What information would you require to ascertain if a group of preschoolers might be at risk of consuming insufficient quantities of iron? What are the risk factors for iron deficiency and anaemia in preschoolers?
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Introduction

Childhood includes children aged 5–10 years and an individual who falls in the age range of 11–18 years is defined as an adolescent. It is during this stage of the lifespan that we begin to notice greater differences in the development and nutritional requirements of males and females. In this section we will focus on nutritional concerns common in childhood and adolescence, as well as issues that are specific to each individual age range.

Learning objectives

At the conclusion of this section you should be able to describe:

• the changes in body composition
• the pattern of growth during childhood and adolescence
• sexual maturation during adolescence
• the most common diet-related disorders
• nutritional concerns in adolescence, including eating disorders and the special needs of pregnant adolescents
• the evidence for the possible role of nutrition in childhood and adolescence in influencing later health.

Learning resources

Print readings

The readings listed here are reproduced in the print readings for this unit.


Online readings

Access the readings listed here using your internet browser.


Deakin Studies Online (DSO)
Access the unit site on DSO <http://www.deakin.edu.au/current-students/> for the appendices and other additional resources you can use to supplement this topic.

Changes in body composition

Differences exist in accretion of lean body mass (LBM) and fat mass (FM) in children and adolescents. With respect to lean body mass, there is an earlier plateau in accretion of LBM in girls when compared to boys. There is also a later and higher peak in LBM velocity in boys (12–14 years) when compared to girls (10–12 years) (Figure 5.1).

With regards to FM, there is a higher increase in FM with age in girls. Girls have a higher mean percentage body fat at every age range (Figure 5.2). During adolescence the difference in the body fat content between the sexes becomes very marked and persists throughout adult life with a more central fat distribution observed in men and a more peripheral pattern in women. There is evidence to suggest this difference in fat deposition exists before puberty with girls having more hip fat and less waist fat than boys (Taylor 2010). Both the total amount and distribution of FM and LBM, and the time at which this occurs, differs between boys and girls from the pre-pubertal stage to late adolescence. There is a higher increase in fat mass with age in girls. Girls have a higher mean percentage body fat at every age range. Boys on the other hand have an increase in body fat in early puberty and then a decrease later in puberty when lean muscle mass increases – their absolute FM is approximately doubled from pre-puberty to older adolescence but relative fatness (% fat) is highest during early puberty. The end result at maturity is that fat-free weight in males makes up about 85% of a total body weight of around 70 kg, and in females about 75% of a total body weight of around 63 kg. As progression to young adulthood may be a time of considerable fat deposition, particularly centrally deposited fat in males, late adolescence could be an appropriate time for targeted physical activity and dietary interventions to limit fat deposition.

Figure 5.1 Accrual of lean body mass from 8–18 years

Characteristics of growth during childhood and adolescence

Unlike infancy, which is characterised by a rapid decrease in the rate of growth, growth during childhood occurs at a relatively steady rate. The adolescent years, however, are again characterised by rapid changes in the rate of growth.

Changes in body size

During childhood (5–10 years) the rate of increase in weight and height is fairly steady at about 2–3 kg and 5–6 cm per year. At adolescence, however, there is a marked increase in both gain in weight and length – the adolescent growth spurt. Height velocity curves show the average age of maximal growth velocity in girls and boys (Figure 5.3). The spurt in height on average begins at 10–11 years in girls and at 12–13 years in boys, although there is wide variation in the time of onset in individuals. For example, in boys it may begin at any time between 10 and 16 years of age. The growth spurt, however, lasts for about the same length of time (2 to 2.5 years duration), irrespective of when it begins.

During the adolescent growth spurt, boys add around 20 cm in height and 20 kg in weight and girls around 16 cm and 16 kg respectively. The peak velocity for weight gain tends to occur about three months later than that for height. In girls, menarche – the onset of menstruation – generally occurs after the peak in height velocity (Figure 5.3), while in boys sexual development is much less closely related to the adolescent growth spurt (Figure 5.3).
The following sections will look at nutritional requirements, nutrient intakes and common nutritional concerns during childhood and adolescence. Note that nutritional needs for growth in relation to body size are proportionally reduced when compared with the younger ages (infants and preschoolers) but are still high when compared to adults.

**Energy balance, physical activity and obesity**

**Energy requirements**

The energy requirements for basal metabolism and activity increase proportionately with body size during childhood, while the energy requirement for growth is comparatively small after the first year of life (Figure 5.4). From infancy to adolescence there is a sharp fall in the energy cost of growth (Figure 5.4). The average daily energy requirement falls from around 400–500 kJ (100–125 kcal) in the first year to around 100–150 kJ (25–38 kcal) in late adolescence (Butte 2000).

Age-specific nutrient requirements for children and adolescents are not well established and are largely based on requirement data for infants and young adults and on data on the amount of nitrogen and minerals required for the increase in lean body mass with age. Table 5.1 provides some estimates of the average increments in body calcium, iron, nitrogen, zinc and magnesium during the second decade of life and at the time of the peak growth spurt. During the second decade of life, the average adolescent boy accumulates about 1 kg of nitrogen, about 750 g of calcium, 16 g of magnesium, 2 g of iron and 1 g of zinc, while the average adolescent girl
accumulates about 600 g of nitrogen, 400 g of calcium, 8 g of magnesium, 0.85 g of iron and about two thirds of a gram of zinc. The sex difference in growth needs for these elements is very marked and due to the much greater accumulation of lean tissue by adolescent boys than girls. If these amounts are expressed on a daily basis it is clear that the actual amounts required for growth constitute only a small fraction of the total recommended dietary intake for nitrogen and magnesium but a significant proportion of the 'absorbed' dietary intake for calcium, iron and zinc during adolescence.

Figure 5.4  The energy cost of growth in females compared with energy requirements

Table 5.1  Average daily increments in body content of selected nutrients due to growth

<table>
<thead>
<tr>
<th></th>
<th>Age 10–20 years (mg)</th>
<th>At peak of growth spurt (mg)</th>
<th>NRV at 14–18 years (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nitrogen *</td>
<td>M  320</td>
<td>610</td>
<td>10400</td>
</tr>
<tr>
<td></td>
<td>F  160</td>
<td>360</td>
<td>7200</td>
</tr>
<tr>
<td>Calcium</td>
<td>M  210</td>
<td>400</td>
<td>1300</td>
</tr>
<tr>
<td></td>
<td>F  110</td>
<td>240</td>
<td>1300</td>
</tr>
<tr>
<td>Magnesium</td>
<td>M  4.4</td>
<td>8.4</td>
<td>410</td>
</tr>
<tr>
<td></td>
<td>F  2.3</td>
<td>5.0</td>
<td>360</td>
</tr>
<tr>
<td>Iron</td>
<td>M  0.57</td>
<td>1.1</td>
<td>11</td>
</tr>
<tr>
<td></td>
<td>F  0.23</td>
<td>0.9</td>
<td>15</td>
</tr>
<tr>
<td>Zinc</td>
<td>M  0.27</td>
<td>0.50</td>
<td>13</td>
</tr>
<tr>
<td></td>
<td>F  0.18</td>
<td>0.31</td>
<td>7</td>
</tr>
</tbody>
</table>

* Nitrogen is protein divided by 6.25

(Adapted from Forbes 1987, p. 167)
Physical activity

ONLINE READING

Please now read subsection ‘4.4.4 Infants, children and adolescents’ (pp. 125–7) in section ‘4.4 Practical considerations: Achieving and maintaining a healthy weight’ and subsections ‘A4.2 Physical activity recommendations for 5–12 year olds’ (p. 170) and ‘A4.3 Physical activity recommendations for 12–18 year olds’ (pp. 170–1) of ‘Appendix 4. Physical activity guidelines’ in the draft Australian Dietary Guidelines.

The Australian Government Department of Health and Ageing recommend that children between 5–18 years of age accumulate at least 60 minutes, and up to several hours of moderate to vigorous physical activity per day (Commonwealth of Australia 2008). Results from the 2007 Children’s Nutrition and Physical Activity Survey (CNPAS) indicate that over a period of 4 days 87% of 9- to 16-year-old boys and 75% of girls met these guidelines. Consistently across the age groups, girls engaged in less vigorous activity and for fewer minutes per day than boys. However, only 16% of boys and 28% of girls met the guidelines for accumulating no more than 2 hours of screen time per day, with girls more likely to meet these guidelines than boys. On average 9- to 16-year-olds spent 223 minutes (over 3½ hours) screen time per day, with the significant majority if this time watching TV (153 minutes per day).

Energy intake

Refer to table 7 in Appendix 5 on DSO for the mean intakes of energy as measured during the 2007 CNPAS. An increase in mean energy intake with age, consistent with the increase in overall body weight is evident during childhood. However, during adolescence, while intake of energy increases markedly in boys from 9.8 to 11.8 MJ; this is not the case in girls whose mean energy intake changes only from 8.7 to 8.6 MJ between 9–16 years. In part this difference is to be expected given the fact that boys experience a greater increase in height and body weight, as well as a much greater increase in lean tissue mass, than girls. However, the difference is also likely to reflect the greater preoccupation that girls have with diet and dieting during the adolescent period. When mean energy intakes are related to basal metabolic rate it is evident that the level of energy intake (boys 1.6 BMR [basal metabolic rate] and girls 1.4–1.5 BMR) is lower than that needed to maintain the level of energy expenditure considered desirable at this age (1.6–1.8 BMR), thus indicating underreporting, especially in older girls.

Obesity

ONLINE READING

Defining overweight and obesity in individuals

For clinical practice (as opposed to population studies and research), the use of a BMI-for-age chart is used for individuals (see Appendices 1.5 and 1.6 on DSO). An individual can be described in terms of a percentile – for example, above the 85th percentile – and their progress plotted on a chart that forms part of the clinical record. An individual with a BMI above the 85th percentile is considered overweight, while a BMI above the 95th percentile is indicative of obesity. Nevertheless, although they are useful in clinical practice, BMI-for-age charts do have some weaknesses. Firstly, the choice of cut-off points to classify overweight and obesity is arbitrary, without evidence of an association with adverse health outcomes. Secondly, the charts must be derived from a reference population. Australia does not at present have growth reference charts derived from the local population, so a reference from another population must be used. The choice of a suitable reference population is also somewhat arbitrary. Importantly, BMI-for-age reference charts should be derived from the same population as that used for other widely used growth references such as weight-for-age and head circumference charts. The Centers for Disease Control and Prevention (CDC) BMI for age charts, developed in the United States, would meet these criteria. Ideally, child and adolescent overweight and obesity would be defined in terms of the risks of adverse health outcomes. Those data are not currently available, and further research to identify the appropriate cut-off points should be a priority.

Prevalence rates for overweight and obesity in children and adolescents in Australia can be tracked by comparing data collected from three national surveys decades apart (Table 5.2) Overweight and obesity are defined here by using the internationally recognised BMI cut points corresponding to the 85th and 95th growth chart. During this ten year period, the level of combined overweight and obesity in children has more than doubled. Data on the prevalence of obesity among Aboriginal and Torres Strait Islander children are scant. The only measured national data comes from the 1994 National Aboriginal and Torres Strait Islander Survey (NATSIS). This survey found that 13% of Aboriginal and Torres Strait Islander boys and 19% of girls aged 7–15 years were overweight (ABS 1998). However this data is not directly comparable with the data presented in Table 5.2 due to different measurement techniques.

Overweight and obesity in childhood and adolescence is associated with hypertension, dyslipidemia, insulin resistance and diabetes, and a range of associated medical conditions including respiratory and orthopaedic problems. In addition overweight and obese children have a lower self esteem, are more likely to be subject to bullying, teasing, lower socioeconomic prospects, and suffer from disordered eating and body image concerns (Carlson 2004). In females, actual body weight correlates negatively with self-esteem in girls as young as five years of age (Davidson & Birch 2002) and in adolescents (Duncan et al. 2002).
Table 5.2 Prevalence of overweight and obesity in Australian children

<table>
<thead>
<tr>
<th></th>
<th>Age (years)</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>7–11</td>
<td>12–15</td>
</tr>
<tr>
<td><strong>Boys</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Australian Health &amp; Fitness Survey 1985</td>
<td>Overweight</td>
<td>9.7%</td>
<td>8.8%</td>
</tr>
<tr>
<td></td>
<td>Obese</td>
<td>1.5%</td>
<td>1.9%</td>
</tr>
<tr>
<td>National Nutrition Survey 1995</td>
<td>Overweight</td>
<td>11.6%</td>
<td>20.0%</td>
</tr>
<tr>
<td></td>
<td>Obese</td>
<td>3.7%</td>
<td>6.1%</td>
</tr>
<tr>
<td><strong>Girls</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Australian Health &amp; Fitness Survey 1985</td>
<td>Overweight</td>
<td>11.0%</td>
<td>10.1%</td>
</tr>
<tr>
<td></td>
<td>Obese</td>
<td>1.9%</td>
<td>1.3%</td>
</tr>
<tr>
<td>National Nutrition Survey 1995</td>
<td>Overweight</td>
<td>17.2%</td>
<td>14.5%</td>
</tr>
<tr>
<td></td>
<td>Obese</td>
<td>6.3%</td>
<td>4.4%</td>
</tr>
<tr>
<td></td>
<td></td>
<td>9–13 years</td>
<td>14–16 years</td>
</tr>
</tbody>
</table>

(The causes of overweight and obesity are complex and multifactorial. Increased sedentary behaviours, decreased physical activity, a higher intake of sugar, fat, takeaway foods and an increased total energy intake are all implicated in the aetiology of weight gain.

Prevention of obesity is much easier than treatment. The most effective preventive measure is monitoring growth patterns in childhood and taking appropriate steps if weight shows a tendency to exceed height on centile charts.

In the treatment of obesity in childhood, two factors need to be stressed. First, very low-energy diets are not suitable for the growing child as it is difficult to ensure an adequate intake of essential nutrients. Food intakes should therefore be sufficient to provide an adequate protein, vitamin and mineral intake.

Second, activity levels need to be investigated. Goran et al. (1998) have followed, over a period of five years, the total energy expenditure, physical activity and body composition of 11 boys and 11 girls initially aged between five and six years. Total energy expenditure (TEE) increased each year in the boys, but declined by 50% between six and nine years of age in the girls while their consumption of food did not change. The authors comment that increased physical activity in all children, as opposed to decreased energy consumption, may be a more effective way of...
improving their health. There is a lack of survey trend data for activity levels in adolescence in Australia however, a 1997 survey of NSW girls in years 8–10 showed that girls were involved in less vigorous physical activity than boys and 70% of girls did not maintain adequate physical activity levels during winter. Particularly low rates of physical activity were seen in girls from Middle Eastern and Asian cultural backgrounds (NSW Health 2010).

Obese children are often inactive by nature. Sedentary activities such as television viewing may reduce the time spent in more active pastimes, such as cycling, ball-games or swimming. Participation in such activities by children and adolescents is more likely to increase if there is also active parental involvement however physical activity levels in Australian adults have declined in the last decade also.

**Calcium and peak bone mass**

**ONLINE READING**

Please now read sub-section ‘2.5.4.3 Children and adolescents’ (p. 67) in section ‘2.5 Milk, yoghurt, cheese and/or alternatives (mostly reduced fat)’ of the *Australian Dietary Guidelines*.

**PRINT READING**

Please read Weaver (2008), ‘Current calcium recommendations in North America’.

Osteoporosis is a condition that is responsible for considerable disability and immobility in later life because of the increased risk of fractures associated with this condition. In turn fracture risk is dependent on bone mineral accretion during growth which is an important determinant of bone mass in later life. Although genetic factors appear to be primary determinants of bone mass, environmental factors such as physical activity and nutrition also contribute. Specific nutritional risk factors for osteoporosis include inadequate calcium and vitamin D status.

Bone growth begins during the second month of gestation and continues until the latter half of the third decade of life. Thereafter some thinning of bone with age occurs in all individuals and can only be slowed, and not prevented by, oestrogen therapy, calcium supplements or regular weight-bearing exercise. The achievement of an individual’s maximum peak bone mass in early adult life is therefore an important first step in prevention. To achieve this, adequate calcium intake during the first 30 years of life is considered to be especially important however controversy exists around the long term efficacy of calcium supplementation to facilitate increased bone density in childhood and adolescence (Gafni & Baron 2007). The reading by Weaver (2008) discusses current calcium requirements and sex and ethnicity differences in calcium accretion. As you read through this article summarise why there are no differences between the calcium recommendations between boys and girls despite the significantly larger skeletal mass in boys.

**Calcium intake**

Refer to Table 9 in Appendix 5 on DSO and note how the median intakes of calcium intake compare to the 2006 NRVs.
Median intakes for 9–13 and 14- to 16-year-olds were 989 and 1143mg/day for boys and 792 and 826 mg/day for girls. As the EAR (estimated average requirement) for this stage of the lifespan is 800 to 1050 mg/day and RDI 1000–1300mg/day (for boys and girls depending on age) the intakes for Australian adolescent girls is alarmingly low. The 2007 CNPAS also determined that only 50–65% of our adolescent boys and 11–45% of adolescent girls had calcium intakes that met the EAR, indicating that inadequate calcium intake is common in this age group. What strategies would you recommend to increase calcium intakes in this important lifespan stage?

Iron deficiency anaemia

Adolescents of both genders are at risk of iron deficiency not primarily because of their diet but because of their rapid rate of growth which is accompanied by a major increase in blood volume and thus by an increased requirement for iron. Female adolescents are at particular risk not only because of the increased demands made by growth but because these are immediately followed by the onset of menstruation and consequent regular loss of iron from the body. Look up the NRVs and compare the RDI for iron for girls and boys 9–13 years and girls and boys between 14–18 years. What are the reasons for the difference in RDIs?

Signs and symptoms of iron-deficiency anaemia include pale gums and lips, loss of appetite, listlessness and irritability. These symptoms can easily be missed in the absence of appropriate blood tests for anaemia. Work done in western New South Wales and in Northern Territory in the 1980s showed that the rate of anaemia (11–12%) in Aboriginal children was much higher than the estimated 3% in non-Aboriginal children (NHMRC 2000).

The 2007 CNPAS found average iron intakes for boys were above both the EAR and RDI and 99–100% of the population sampled achieved intakes at or above the EAR. However, although average iron intakes were above the EAR for 14- to 16-year-old girls, 11% of the population did not meet the EAR compared with 98% of younger girls.

PRINT READING

Please read Gibson et al. (2002) in your print readings.

Summarise are the main issues highlighted in this article.
Dental caries

ONLINE READING
Please now read subsection ‘3.3.4.2 Children’ (p. 92) in section ‘3.3 Limit intake of foods and drinks containing added sugars’ of the Australian Dietary Guidelines.

Dental caries is one of the most common nutritional diseases today. It affects children of all socioeconomic levels and all ages. Once the teeth have erupted through the gum they are vulnerable to the effects of bacteria present in the mouth. These bacteria are responsible both for the formation of dental plaque, and the fermentation of dietary carbohydrates, to produce acids which dissolve the tooth enamel and thus cause cavities.

Sugar, especially in a form such as caramel or chocolate which clings to the tooth surface, is the principal substrate for plaque and acid fermentation. The amount of caries produced by a given foodstuff is, however, not merely a function of sugar content but is determined by a combination of factors including:

- frequency of use
- effects on saliva production
- time of retention on the tooth
- effects on dental plaque formation
- ability to dissolve enamel.

Eating (or drinking) sugar between meals has a more cariogenic effect than eating it with meals, presumably because the flow of saliva is less and there is less buffering action than with meals.

Fluoride is normally present in tooth enamel and protects against caries. In areas where fluoride is found naturally in small amounts in the water supply, or where it is added to a level of 1 ppm, the prevalence of dental caries in children is significantly less than in non-fluoridated areas.

A combination of a fluoridated water supply and a decrease in between-meal eating of high-sugar foods which adhere to the dental plaque is the best approach to reducing the prevalence of dental caries in children. The Dental Statistics and Research Unit of the Australian Institute of Health and Welfare (AIHW) regularly collates data on the caries status of Australian children and these indicate that throughout the 1990’s there was a decline in the rate of new disease in both 6 and 12 year old children (Figures 5.3, 5.4). However, from 1999 a steady incline in disease was observed in 6 year old children (Figure 5.3). A similar increase was observed in 12 year old children from 2003 (Figure 5.4). Children’s dental health in Australia is better than many other developed countries. However, there is a strong socioeconomic influence on dental health with economic disadvantage and living in a rural area being the two greatest risks for poor dental health in Australia. Children from disadvantaged backgrounds are found to have significantly poorer dental health (AIHW 1998). The incidence of decayed, missing and filled teeth in aboriginal children is almost double that of non-Aboriginal children. Indigenous children have more than twice as many caries as non-indigenous children and their oral health has
continued to worsen over recent decades in contrast to non-Indigenous children (National Advisory Committee on Oral Health 2004).

**Vegetarian diets**

Vegetarian diets are generally low in energy density and high in bulk when compared to omnivorous diets. In adults a number of benefits to health have been reported to be associated with a vegetarian diet although it is by no means clear to what extent these benefits may be due to other aspects of a ‘vegetarian’ lifestyle.

There is no similar evidence for health benefits of vegetarian diets during early childhood. In contrast there is, instead, some concern that vegetarian diets may, in some circumstances, compromise the intake of energy and nutrients required for growth such as iron, zinc and calcium. Iron deficiency anaemia is probably the main nutritional risk associated with vegetarian diets in children because of the low intake of haem sources of iron even in lacto-ovo-vegetarian diets. Concerns over protein intake and quality are hardly ever warranted unless the diet is a restricted vegan diet. Vegetarianism is not uncommon in adolescents as increasingly self-managed values play out through what is viewed as acceptable food sources. Vegetarianism and veganism are common dietary practices among many religious and ethnic groups in multicultural Australia. Vegetarian adolescents should be supported in the planning of a balanced nutritional intake.

**Figure 5.5** Caries experience of 6-year-old Australian children from 1990 to 2004

(Armfield et al. 2009, p. 44)
Specific nutritional concerns in adolescents

Eating disorders

ONLINE READING
Please now read subsection ‘4.4.8 People with eating disorders’ (pp. 129–30) of section ‘4. Achieve and maintain a healthy weight’ in the Australian Dietary Guidelines.

The most common eating disorders are anorexia nervosa and bulimia nervosa. It is usual for psychological factors to influence the development of eating disorders. Eating disorders are more common in females but they increasingly also occur in males. The age of onset of eating disorders is becoming younger as society has an increasing preoccupation with an ‘ideal’ body shape (Dietitians Association of Australia 2011b).

Anorexia nervosa is an extreme form of starvation as weight-control behaviour which appears to be very resistant to treatment. It is seen most commonly in young women, typically those from achievement-oriented and successful families. Bulimia is the term used to describe the sequence of binge eating followed by vomiting and/or use of laxatives which is one of the methods of weight control adopted in anorexia nervosa but which may also be used by some individuals to maintain a near-normal weight-for-height status.

Adolescent pregnancy
The nutrients that are at risk of being inadequate during adolescence are likely to be further compromised by pregnancy. Pregnancy in adolescence is more frequently associated with obstetric complications, low birth weight infants and perinatal mortality, than pregnancy in the third decade of life. The reasons for this are threefold. First, the adolescent, although sexually mature, may not have completed her skeletal
growth and consequently pelvic structure may not be optimal for pregnancy. Secondly, the nutritional demands of pregnancy are superimposed on those for growth, and thirdly, the socioeconomic environment frequently associated with teenage pregnancy makes it less likely that appropriate antenatal care is sought.

**Nutrition in childhood and later health**

There is now general recognition of the fact that conditions both during foetal life and during childhood can influence physical and psychological development and ultimately the health of the adult.

There are clearly many factors that can influence later health. Here we concern ourselves only with specific nutritional influences. However, it is important to recognise that nutritional influences do not operate in isolation and are often conditioned by, or the direct consequence of, environmental and lifestyle factors. Non-nutritional factors can often have a much greater impact on health than nutritional factors, for example the effects of poor sanitation and smoking on health status in general greatly outweigh the effects of any associated nutritional factors.

Specific nutrients and dietary components which have been implicated in the development of disease in later life, and which may therefore also exert an influence in childhood, include sodium, the amount and nature of dietary fat, and dietary fibre.

**Sodium**

There is now a considerable body of evidence indicating that a high salt intake contributes to the development of hypertension and that current salt intake is the major factor increasing blood pressure and therefore cardiovascular disease.

Studies in children that have attempted to relate sodium intake and/or excretion to blood pressure have not given consistent results but this may, in part, relate to the difficulty of estimating sodium intake and excretion in children. Intervention studies in children have also shown relatively little effect on blood pressure. However, it is clear that blood pressure rises during childhood and that children in the upper part of the blood pressure distribution at earlier ages also tend to be in the upper part of the distribution at later ages. This tendency for blood pressure to ‘track’ may, however, be genetic rather than related to environmental factors such as diet. In infants and young children the return to breastfeeding, the widespread use of commercial breast-milk substitutes and the reduced sodium content of infant foods have resulted in a lower sodium intake than in the earlier part of the twentieth century, but the effect, if any, of this reduction in sodium intake in early life on later blood pressure has yet to be determined. Average intakes from the 2007 CNPAS indicate levels of consumption well above current recommendations. The adequate intake (AI) for this life span is between 400–920 mg/day, the UL is 2000–2300 mg/day and the suggested daily target (appropriate for >14years) is to restrict sodium intake to <1600 mg/day. Average intakes were 2890–3672 mg/day for boys and 2490–2623 mg/day for girls. The tracking of blood pressure and liking for high sodium products into adulthood means these levels of intake present significant health concerns. In addition, evidence from the UK shows that children who have more salt in the diet consume more sugar-sweetened soft drink which in itself is related to childhood obesity (Feng 2008).
Dietary fat

**ONLINE READING**

Please now subsection ‘3.1.4.2 Children and adolescents’ (p. 81) of section ‘3.1 Limiting intake of foods and drinks containing saturated and trans fat’ in the *Australian Dietary Guidelines*.

In adults hypercholesterolaemia is an important risk factor for coronary heart disease (CHD) and is related to the consumption of a diet high in saturated fat. However, the time at which serum cholesterol level becomes influenced by the diet, and a risk factor for later CHD, is not well established. For example, in infancy serum cholesterol levels appear to be similar irrespective of the type of feeding, while at 7–8 years of age they already appear to reflect differences in dietary fat content.

Similarly, the role of nutritional factors in the thickening of the intima of the coronary arteries, which has been described even in very young children and which it is possible may predispose to atherosclerosis, is not known. It is known, however, that human milk provides more than 50% of its energy from fat and that approximately 48% of this is in the form of saturated fat. It is also clear that dietary fat restriction in early life can interfere with growth, by limiting energy intake and the supply of fat-soluble vitamins and essential fatty acids.

Dietary fat has also been implicated in the development of a number of cancers which occur in later life, in particular colon and breast cancer, but again the time at which fat intake becomes a risk factor is not yet clear.

**Dietary fibre**

A low intake of dietary fibre in childhood is frequently associated with constipation, and the longer-term risks of a low-fibre diet are reported to include diverticular disease, cancer of the colon, coronary heart disease and obesity in later life. The role of a low-fibre diet as opposed to a high-fat diet is not clear in relation to the development of these longer-term problems.

**ONLINE READING**

Please now read subsections ‘2.3.4 Practical considerations: Eat grain (cereal) foods, mostly wholegrain’ (pp. 48–50) and ‘2.2.6.3 Children and adolescents’ (pp. 44–5) in section ‘2. Eat a wide variety of nutritious foods’ of the *Australian Dietary Guidelines*.

A sufficient intake of dietary fibre from adequate amounts of cereals, breads, fruits and vegetables is the most appropriate strategy to prevent constipation in childhood. Adequate intake levels set for children are 18 g per day for 4- to 8-year-olds, 20 g per day for 9- to 13-year-old girls, 24 g/day for 9- to 13-year-old boys, 22 g per day for 14- to 18-year-old girls and 28 g day for 14- to 18-year-old boys. Data from the National Nutrition Survey in 1995 shows that dietary fibre intake in children and adolescents was largely inadequate.

**Alcohol**

Children aged under 15 years are at the greatest risk of harm from alcohol consumption and in the Australian Dietary Guidelines, parents and carers are
cautioned that not drinking alcohol is particularly important for children. Alcohol consumption among younger people can impede brain development and the Australian Dietary Guidelines states:

For young people aged 15–17 years, the safest option is to delay the initiation of drinking for as long as possible. (NHMRC 2011)

**Food requirements for children and adolescents**

The recommended food patterns for children and adolescents differ according to gender and age, as presented in Tables 5.3 and 5.4. As the recommendations are for those of average height and with sedentary to moderate activity levels, children and adolescents who are taller or more active will need additional serves to meet their extra energy requirements.

**Table 5.3 Recommended dietary patterns for male children and adolescents of average height and with sedentary to moderate activity levels**

<table>
<thead>
<tr>
<th>Food group</th>
<th>Serve definition</th>
<th>4 – 8 years</th>
<th>9 – 11 years</th>
<th>12 – 13 years</th>
<th>14 – 18 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vegetables and legumes / beans</td>
<td>1/2 cup cooked vegetables (75 g)&lt;br&gt;1 cup salad vegetables&lt;br&gt;1 small potato&lt;br&gt;½ cup cooked dried or canned beans or lentils, no added salt</td>
<td>4½</td>
<td>5</td>
<td>5½</td>
<td>5½</td>
</tr>
<tr>
<td>Fruits</td>
<td>1 medium piece of fresh fruit (150 g)&lt;br&gt;30 g dried fruit (e.g. 4 dried apricot halves)&lt;br&gt;1 cup canned fruit (150 g)</td>
<td>1½</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Grain (cereal) foods</td>
<td>1 slice of bread or ½ a medium roll or flat bread (about 40 g)&lt;br&gt;½ cup cooked rice, pasta, noodles&lt;br&gt;½ cup cooked porridge or polenta, 2/3 cup breakfast cereal flakes (30 g) or ½ cup muesli</td>
<td>4</td>
<td>5</td>
<td>6</td>
<td>7</td>
</tr>
<tr>
<td>Lean meat and poultry, fish, eggs, nuts and seeds, and legumes / beans</td>
<td>65 g cooked lean red meats or ½ cup of lean mince, 2 small chops&lt;br&gt;80 g cooked poultry&lt;br&gt;100 g cooked fish fillet or 1 small can of fish&lt;br&gt;2 large eggs&lt;br&gt;170 g tofu</td>
<td>1½</td>
<td>2½</td>
<td>2½</td>
<td>2½</td>
</tr>
<tr>
<td>Milk, yoghurt, cheese and/or alternatives</td>
<td>250 mL cup of milk&lt;br&gt;200 g tub of yoghurt&lt;br&gt;40 g or 2 slices of cheese&lt;br&gt;120 g ricotta cheese</td>
<td>2</td>
<td>2½</td>
<td>3½</td>
<td>3½</td>
</tr>
<tr>
<td>Additional serves from the five food groups or discretionary choices for those who are taller or more active</td>
<td>0 – 2½&lt;br&gt;0 – 3&lt;br&gt;0-3&lt;br&gt;0-3</td>
<td>(NHMRC 2011)</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
**Table 5.4**

<table>
<thead>
<tr>
<th>Food group</th>
<th>Serve definition</th>
<th>4 – 8 years</th>
<th>9 – 11 years</th>
<th>12 – 13 years</th>
<th>14 – 18 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vegetables and legumes / beans</td>
<td>1/2 cup cooked vegetables (75 g) 1 cup salad vegetables 1 small potato ½ cup cooked dried or canned beans or lentils, no added salt</td>
<td>4½</td>
<td>5</td>
<td>5</td>
<td>5</td>
</tr>
<tr>
<td>Fruits</td>
<td>1 medium piece of fresh fruit (150 g) 30 g dried fruit (e.g. 4 dried apricot halves) 1 cup canned fruit (150 g)</td>
<td>1½</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Grain (cereal) foods</td>
<td>1 slice of bread or ½ a medium roll or flat bread (about 40 g) ½ cup cooked rice, pasta, noodles ½ cup cooked porridge or polenta, 2/3 cup breakfast cereal flakes (30 g) or ½ cup muesli</td>
<td>4</td>
<td>4</td>
<td>5</td>
<td>7</td>
</tr>
<tr>
<td>Lean meat and poultry, fish, eggs, nuts and seeds, and legumes / beans</td>
<td>65 g cooked lean red meats or ½ cup of lean mince, 2 small chops 80 g cooked poultry 100 g cooked fish fillet or 1 small can of fish 2 large eggs 170 g tofu</td>
<td>1</td>
<td>2½</td>
<td>2½</td>
<td>2½</td>
</tr>
<tr>
<td>Milk, yoghurt, cheese and/or alternatives</td>
<td>250 mL cup of milk 200 g tub of yoghurt 40 g or 2 slices of cheese 120 g ricotta cheese</td>
<td>1½</td>
<td>3</td>
<td>3½</td>
<td>3½</td>
</tr>
<tr>
<td>Additional serves from the five food groups or discretionary choices for those who are taller or more active</td>
<td>0 – 1</td>
<td>0 – 3</td>
<td>0 – 3</td>
<td>0 – 2½</td>
<td></td>
</tr>
</tbody>
</table>

(NHMRC 2011)

**Adherence to the dietary guidelines**

As with childhood dietary patterns, changes to dietary patterns are required to meet the foundation diet models underlying the 2011 Australian Guide to Healthy Eating and the ability to adhere to the 2003 dietary guidelines is poor. Table 5.5 summarises the dietary changes needed to meet the foundation diet and Table 5.6 below describes the proportion of the population meeting the dietary guidelines in the 2007 CNPAS.
### Table 5.5  
**Changes to dietary patterns required for children to meet the ‘foundation diet’ underlying the draft 2011 Australian Guide to Healthy Eating**

<table>
<thead>
<tr>
<th>Dietary Guideline</th>
<th>Measurement criteria</th>
<th>Ages 4–8 years</th>
<th>Ages 9–13</th>
<th>Ages 14–16</th>
<th>Ages 17–18</th>
</tr>
</thead>
<tbody>
<tr>
<td>Encourage, support breastfeeding</td>
<td>Ever been breast fed</td>
<td>90</td>
<td>92</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eat plenty of fruits</td>
<td>≥1–3 serves/d excluding juice</td>
<td>51</td>
<td>1</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>≥1–3 serves/d including juice</td>
<td>90</td>
<td>24</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eat plenty of vegetables</td>
<td>≥2–4 serves/d excluding potatoes</td>
<td>2</td>
<td>0</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>≥2–4 serves/d including potatoes</td>
<td>14</td>
<td>5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eat plenty of cereals</td>
<td>≥3–4 serves/day</td>
<td>11</td>
<td>25</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Include lean meat, fish, poultry and/or alternatives</td>
<td>%&gt;EAR for protein, iron, zinc</td>
<td>99–100</td>
<td>93–100</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Include milks, yoghurts, cheese and/or alternatives</td>
<td>%&gt;Calcium EAR</td>
<td>55,31*</td>
<td>38</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Choose water as a drink</td>
<td>%&gt;fluid AI</td>
<td>57</td>
<td>46</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Limit saturated fat and moderate total fat intake</td>
<td>&lt;10% energy from saturated fat</td>
<td>19</td>
<td>22</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Choose foods low in salt</td>
<td>&gt;sodium AI</td>
<td>100</td>
<td>100</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Consume only moderate amounts of sugars</td>
<td>%&lt;20% energy from total sugars</td>
<td>33</td>
<td>39</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(Dietitians Association of Australia 2011a)

### Table 5.6  
**Proportion of children meeting the serve recommendations of the 2003 Australian Guide to Healthy Eating**

<table>
<thead>
<tr>
<th>Dietary Guideline</th>
<th>All children 9–13 years</th>
<th>All children 14–16 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Encourage, support breastfeeding</td>
<td>90</td>
<td>92</td>
</tr>
<tr>
<td>Eat plenty of fruits</td>
<td>51</td>
<td>1</td>
</tr>
<tr>
<td>Eat plenty of vegetables</td>
<td>2</td>
<td>0</td>
</tr>
<tr>
<td>Eat plenty of cereals</td>
<td>14</td>
<td>5</td>
</tr>
<tr>
<td>Include lean meat, fish, poultry and/or alternatives</td>
<td>99–100</td>
<td>93–100</td>
</tr>
<tr>
<td>Include milks, yoghurts, cheese and/or alternatives</td>
<td>55,31*</td>
<td>38</td>
</tr>
<tr>
<td>Choose water as a drink</td>
<td>57</td>
<td>46</td>
</tr>
<tr>
<td>Limit saturated fat and moderate total fat intake</td>
<td>19</td>
<td>22</td>
</tr>
<tr>
<td>Choose foods low in salt</td>
<td>100</td>
<td>100</td>
</tr>
<tr>
<td>Consume only moderate amounts of sugars</td>
<td>33</td>
<td>39</td>
</tr>
</tbody>
</table>

*Different EARs exist for children in this age range resulting in two figures for proportion meeting the EAR.*

(Adapted from Commonwealth of Australia 2008)
TEXT QUESTIONS

1. Are the nutrient needs of children and adults similar when compared:
   (a) per kilogram of body weight?
   (b) per unit of energy intake?

2. What are some of the approaches used to estimate energy and nutrient needs during childhood and adolescence?

3. It may be estimated that at the time of peak growth during adolescence 610 mg nitrogen and 400 mg calcium are retained daily in males. How do these amounts compare with maintenance requirements for these two elements?

4. What are the most common nutrition-related problems in childhood, and how do they arise?

5. What are the main nutrition-related problems encountered in adolescence, and what steps might be taken to reduce their prevalence?

6. What dietary factors in childhood can have important implications for health:
   (a) during childhood?
   (b) in later life?

7. Are school-aged children in general at risk of developing iron deficiency anaemia?

8. When interpreting the mean intake of iron what other factors will you need to consider.

9. Using tables 7 and 7 from Appendix 5 on DSO, construct a table of the average percent energy from total fat, saturated fat, carbohydrate, sugar protein and grams of fibre for 14- to 16-year-olds. Compare these average values with the NRVs (where available). What changes, if any are required in the average Australian diet to meet these guidelines? What practical strategies do you suggest to help achieve these changes?

References


Gafni, RI & Baron, J 2007, ‘Childhood bone mass acquisition and peak bone mass may not be important determinants of bone mass in late adulthood’, *Pediatrics*, vol. 119, suppl. 2, pp. S131–S36.


National Health and Medical Research Council 2000, Nutrition in Aboriginal and Torres Strait Islander Peoples– an information paper, AGPS, Canberra.


TOPIC 6

Factors influencing healthy eating behaviours

PREPARED BY THE UNIT TEAM

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Introduction

Healthy eating behaviours can be described as eating a healthy balanced diet as well as having a healthy attitude to eating. Developing healthy eating behaviours in our population is the responsibility of the individual, the family and the wider environment including: schools, social policy, government agencies and invested industries. In this topic we will explore factors associated with the establishment of healthy eating behaviours and factors that work to promote unhealthy eating behaviours in both the short and longer term.

Learning objectives

At the conclusion of this section you should have an understanding of and be able to discuss:

• what is meant by healthy eating behaviours
• what a positive food environment means
• the importance of the family mealtime
• the role of television and marketing in establishing eating behaviours
• strategies to promote healthy eating.

Learning resources

Print readings

The readings listed here are reproduced in the print readings for this unit.


Deakin Studies Online (DSO)

Access the unit site on DSO <http://www.deakin.edu.au/current-students/> for the appendices and other additional resources you can use to supplement this topic.

Basic concepts

Evidence gained from several decades of nutrition research provides us with an understanding of dietary factors associated with lowered risk of dietary related chronic diseases. Although by no means complete, this evidence provides us with the basis for developing both the *Nutrient reference values for Australia and New Zealand* and the *Australian Dietary Guidelines*. In addition, evidence collected from the most recent National Nutrition Survey provides us with information as to current dietary intake patterns within Australia. In order to achieve the goals of our dietary guidelines, there is a need to increase intakes of
fruits, vegetables, whole grains, and low-fat dairy products or alternatives and to lower intakes of added sugars, high fat foods, salty foods and ‘junk food’, especially soft drinks. Achieving and maintaining a healthy body weight and healthy physical activity patterns and reduced time spent in sedentary behaviours such as television viewing are also required. However, we have limited information to date on how best to achieve these changes. To be able to influence dietary habits it is first necessary to understand factors involved in establishing dietary habits.

Significant similarities exist between parental diet composition and child’s diet composition. Similarities between parents’ or carers’ dietary intake and their children’s are thought to exist due to the availability, accessibility and exposure of foods. Although there is also likely to be a genetic component to food liking and ultimately intake, taste preference is largely learnt as well, as shown by differences in food preferences across different populations. As described by Birch and colleagues: ‘Parents provide genetic predisposition but also provide environment in which these predispositions are expressed’ (Birch & Fisher 1998). Eating behaviours established in childhood track over early years and persist into adulthood; therefore, establishing healthy food habits early in life has the potential to significantly prevent the onset of many dietary related chronic diseases.

Healthy eating behaviours in infancy

Exposing an infant to either breast milk or infant formula has the potential to shape food preferences and acceptance of new foods in later life. The flavour of breast milk varies in response to maternal diet thereby exposing the breast fed infant to a range of flavours. Breast milk produced from mothers following a bland diet has been reported to be less well liked (i.e. the infant does not suck for the same duration) as breast milk produced from mothers following a diet enhanced with both vanilla and garlic flavours (Mennella & Beauchamp 1993; Mennella & Beauchamp 1996). Animal studies have also shown that in later life rat pups prefer the chow that their mothers consumed while nursing the pups. Long-term evidence of the effect of early frequent exposure to different flavours on dietary patterns is lacking; however, breastfeeding is associated with a greater acceptance of new foods and greater acceptance of a variety of foods during the early introduction of solid foods (Sullivan & Birch 1994; Cooke, Wardle et al. 2004).

Another significant difference between breastfeeding and formula feeding is the level of external control of intake that may be exerted by the parent or carer. With bottle feeding there is visual feedback as to the volume of milk ingested, there is less feedback with breast feeding. Bottle feeding has the potential to override the internal hunger cues of the infant if the feeding is continued or stopped based on the amount of fluid left remaining in the bottle. A breastfed infant may have greater control over the amount of milk consumed. US studies have indicated that formula fed infants grow more rapidly than do breast fed infants. One proposed hypothesis for this higher growth in formula fed infants may relate to the fact that mothers are controlling intake and may consistently over feed their infants.
Factors influencing food choice in the preschool and childhood years

The main features of this stage of the lifespan are the introduction of solid foods and the development and maturation of self-feeding skills. Introducing solid foods and new foods can often be a stressful and challenging time for parents and caregivers. It is also a time in which lifelong healthy eating behaviours can be developed.

Current research indicates that early repeated exposure to a variety of flavours can lead to greater acceptance of new foods in later life. We have an innate preference for sweet-tasting compounds and an aversion to bitter-tasting compounds, while preferences for fatty, salty and sour foods are likely to be learned. Introduction to a variety of textures in these early months also has the potential to enhance acceptance of varying textures. There is some evidence to suggest that the common practice of blending foods to a smooth consistency is not required if solid foods are delayed until approximately 6 months. Fork mashing of foods is sufficient for safety and may enhance acceptance of foods of varying textures. A preschooler is predisposed to be food neophobic—that is, to fear new foods, and has the ability to associate food textures and flavours with contexts and consequences of eating. As a child tends to eat what it likes and leave the rest, the shaping of early food exposures is important in determining healthful food preferences and such exposure is likely to be important determinants of food preferences.

Food neophobia

Food neophobia, a term used to describe the initial rejection of new foods, is common in children and peaks at around two to three years of age. The extent to which a child will reject or accept a new food varies significantly between children with some children reporting high levels of food neophobia. Highly food neophobic children report liking and trying fewer foods, and have less dietary variety than less food neophobic children. Although the extent of the research is limited, existing data suggest that children who are more food neophobic typically consume a diet with less fruits and vegetables and more fat than children who report less food neophobia (Cooke, Wardle et al. 2004; Cooke, Carnell et al. 2006). While food neophobia is an innate response to new foods it is possible to reduce the neophobic response to food and enhance liking of previously rejected foods. Repeat exposure to and opportunities to consume new foods is vital to increasing liking and acceptability of new foods, with 5–10 exposures often required. Work by Birch and Gunder has indicated that repeat exposure of a single food item not only increased the acceptance of that item, but also increased the liking of other, similar foods (Birch, Gunder et al. 1998). To further increase the acceptability of a new food, it should be introduced in a familiar environment as both animal and human studies have found that combining the introduction of a new food in a new situation increases reluctance to approach and eat a new food. Some evidence also suggests that the earlier the food is introduced to the child, the greater the child’s liking and intake of that food (Cooke, Wardle et al. 2004).
Conditioned preferences

Learned aversion or liking for food is well recognised in the literature and is distinct from food neophobia, which is an innate rather than a learned response to new foods. Learned aversion or liking refers to the propensity to associate either positive or negative post ingestive consequences of eating the food with the food itself. Learned associations may be conscious or unconscious and have been shown to be persistent. In general, acquiring liking for a food tends to take longer than acquiring disliking for a food, repeat positive exposures are required to enhance liking of a food, yet food aversions can develop after only one experience of a specific food (Hursti 1999). Even at a young age, an individual has the ability to learn to prefer foods due to physiologic consequences, for example consuming a high fat sugar rich food when hungry can enhance the liking of that food as the satiation effect becomes linked to the food.

Modifying food preferences

A child’s food preference can also be altered by placing the child in an eating environment with other children with different preferences. Several studies have shown that a child who does not like a vegetable will eat more of it and report they like it more if they are eating with children who like that vegetable (Birch & Fisher 1998). Positive peer modelling appears important in establishing healthy eating behaviours. This introduces interesting possibilities for establishing healthy eating behaviours within our current environment of a greater number of children in child care settings.

Parent/carer influence and the family food environment

The food environment the parent provides shapes children’s preferences and food acceptance patterns. Parents determine the feeding method (including the early choice of breast feeding or infant formula feeding), the foods they make available and accessible within the home, the opportunities to model eating, the form of interaction with the food environment and the extent of media exposure. These factors have been shown to be important in influencing a child’s food preference and intake and are discussed below.

Parental modelling

Although peer modelling may influence children’s eating, of greater importance is the role of the mother in determining liking or disliking of a food. Positive parental modelling of fruit and vegetable intake has been shown to increase the amount of fruits and vegetables consumed by children and traditional family mealtimes are associated with increased fruit and vegetable intake (Cooke, Wardle et al. 2004). In a large survey of children in the UK, Cooke and Wardle investigated predictors of fruit and vegetable intake in children aged approximately four years. Four factors were found to significantly predict fruit intake with higher intakes predicted by higher parental intake, early introduction of fruit, low levels of food neophobia, and breast feeding before the introduction of solids. Parental intake of vegetables, low recorded levels of food neophobia and greater enjoyment of food predicted the children’s vegetable intake. Early feeding method (i.e. breast feeding versus bottle feeding) and age of introduction of vegetables did not significantly influence vegetable intake. In a well controlled intervention trial, liking and consumption of a previously disliked vegetable was increased in 2- to 6-year-old children following
two weeks of exposure, encouragement to taste the vegetable and positive parental modelling of eating and liking the vegetable (Wardle, Cooke et al. 2003) Studies have also shown that children more readily put food into their mouth if they see their mother doing so compared with seeing a stranger eating it. The role of parental modelling, especially maternal modelling of liking and consumption of vegetables consistently appears in both cross-sectional and intervention studies.

**Restriction and parental control**

Restriction of foods and parental control over foods represent two key areas influencing children’s eating behaviours; however, these are difficult and challenging areas to research with often apparently conflicting outcomes. Parental control and restriction can be measured in a clinical or laboratory setting which removes the individuals from their normal home environment which may independently affect food behaviour. Alternatively, these parameters can be measured through observation in the individuals’ home setting or more frequently via a variety of questionnaires assessing feeding practices. In clinical or laboratory settings, studies have indicated that restricting access to food enhances liking for that food and increases consumption of the food item when it becomes available. For example, a study by Fisher and Birch on 31, 4-to 6-year-old children (Fisher & Birch 1999) exposed the children to two similar snack foods but restricted access to one of the foods; that is, the snack food was visible but not physically accessible. Despite the similar nature of the restricted item, the restricted food elicited more positive comments about it, more requests for it and more attempts to obtain it. This effect, however, seemed to diminish over time and no significant difference between the two food items was observed three weeks after the experiment.

These results would indicate that restricting food items may result in increased liking for that item. As restricted food items tend to be higher in fat and sugar (in an attempt to moderate intake), this practice may draw children’s attention to and focus their behaviour on foods that should be consumed in moderation (for a review see Ventura & Birch 2008). It is important, however, to be aware of some limitations to this research. The limited number of studies that have been conducted tend to investigate the effects of restricting access to visible food; they do not address whether similar outcomes may be observed if the food item is restricted by not having the food item visible (i.e. keeping it out of the home). In addition, the clinical environment in which this research has been carried out does not reflect the ‘at home’ environment. There is considerably less information available on the impact of food restriction in the home environment. The evidence currently available indicates that ‘prudent restriction’ appears to promote healthy eating behaviours, and thus availability is considered more important in establishing healthy or unhealthy eating patterns (Campbell et al 2010). This is discussed further at the end of this section.

**Pressure to eat**

An additional aspect to feeding behaviours is the use of pressure to eat. Pressure to eat has been associated with children with a lower fat mass or BMI and has been associated with a higher total energy intake, suggesting that this parental approach may be in response to a child that is perceived to be too small or not eat enough (Campbell, Crawford et al. 2002; Spruijt-Metz, Lindquist et al. 2002; Carnell &
Pressure to eat may also diminish the intake of the food item the child is pressured to consume more of (Galloway, Fiorito et al. 2006).

**Food as a reward**

The use of food as a reward has also been shown to positively increase liking for the reward food and decrease liking for the ‘means’ food. Eighty-seven 4- to 7-year-old children were asked to consume food A: before they were allowed to have a taste of food B. Prior to the reward experiment, both foods were equally well liked, post-test ratings , however, the results indicated that liking for food B had increased and liking for food A had diminished (Newman & Taylor 1992). Therefore, the common use of the phrases ‘you must eat your veggies’ or ‘you must finish your plate’ before dessert may diminish the liking for the food that parents are trying to increase intake of (Kröller & Warschburger 2008).

**Internal and external eating cues**

Young preschool children (under ~3 years) have the ability to eat to satisfy their own hunger. If an infant stops eating: they are full, if an infant continues eating: they are hungry. While the ability to self-regulate food intake diminishes and external cues become more powerful during childhood, it is possible to enhance or decrease a child’s ability to eat only to satisfy their hunger needs. In one of the early studies in this field, Birch and colleagues exposed 22 young children (~4-years-old) who were trained to focus either on external cues (i.e. the amount of food remaining on the plate) or internal cues of hunger and satiety. Only the children trained on focusing on internal cues were able to adjust the amount of food they consumed as the energy content of the meal varied. The children trained to focus on external cues increased *ad libitum* food intake as caloric content of the meals varied (Birch, McPhee et al. 1987). Observational data of 142 families of kinder-aged children found that 85% of families prompted children to eat more once they had finished eating. Once prompted, 83% of children ate more than they might otherwise have and 38% ate moderately to substantially more (Orrell-Valente, Hill et al. 2007). Excess parental control over the amount a child eats may start to decrease a child’s ability to self regulate.

Overall, the research described above indicates that greater parental control, in the form of restriction, pressure to eat, and the use of food as a reward may be counterproductive in enhancing healthy eating behaviours. However, research from cross-sectional studies of community groups indicate that some form of parental control does not necessarily promote obesity or unhealthy eating practices. Cross-sectional studies assessing children’s weight and parental control determined using questionnaires have shown no association between control and body mass index (BMI) in 3- to 6-year-olds (Faith et al. 2003) and 8- to 9-year-olds (Robinson, Kiernan et al. 2001). Furthermore, retrospective studies of adolescents have shown that children who had more healthful eating habits reported greater levels of parental control in preceding years. This evidence has coined the phrase ‘prudent restriction’; that is, a home environment in which unhealthy foods are not freely available but when available are not overly restricted. Thus, despite the clinical evidence that suggests parental restriction or control or using food as a reward increases liking for and intake of energy dense foods, the wider community evidence that this translates into an increased body weight and unhealthy eating behaviours in these children is lacking. The diversity of research methodologies employed in these studies, the range of questionnaires available to measure or
assess parental feeding behaviours, and potentially the ethnic and cultural variables affecting dietary intake and eating behaviours make it difficult to interpret the outcomes of these many studies. Potentially, longer term longitudinal studies are required to determine the long term effect of variances in parental feeding behaviours.

**Nutrition knowledge and Self-efficacy**

It is well recognised that increased nutrition knowledge does not necessarily translate into improved dietary intake (see Worsley 2002), however, having a lack of knowledge regarding nutrition may limit an individual’s ability to change dietary behaviour or identifying risk factors requiring change. Increased maternal nutrition knowledge has been associated with improved dietary intakes in young children. In addition, parental belief in their confidence to provide appropriate, healthy foods that their children will consume is referred to as self-efficacy. To date, self-efficacy has been under-researched in the literature regarding parental feeding strategies. In a recent study, mothers that recorded higher levels of self-efficacy for healthy eating reported higher intakes of vegetables and lower intakes of cordial and cakes in their children aged six to 20 months (Campbell et al. 2010)

**Mealtime environment**

Please now read Campbell et al. (2006), ‘Family food environment and dietary behaviours likely to promote fatness in 5–6 year-old children’.

Children learn to associate the situation or place where food is eaten with food and if placed in that situation, they are more likely to easily initiate eating even when they are not hungry. Therefore, these strong environmental cues are important in establishing healthy eating behaviours. It is important that meals are encouraged to be consumed at the table instead of in front of the television, or in the car for example. This sets up an environmental associated behaviour of wanting to eat when sitting down in front of the television even if they are not hungry. In addition to the physical placement of the meal, other factors such as who the food is eaten with, how eating is encouraged/discouraged, the level of stress/conflict during eating, and interruptions during eating (TV/telephone) have all been shown to influence children’s food habits and food intake (Hursti 1999; Campbell, Crawford et al. 2002). Parents of obese children (8- to 16-years-old) report greater psychological distress, greater mealtime conflict and less positive mealtime interactions than age and demographically matched parents of non-obese children (Zeller, Reiter-Purtill et al. 2007). The article by Campbell in your readings describes a study of the Australian family food environment, as you read through this article, note the family characteristics that were associated with an increased or decreased energy, vegetable and fruit intake.

While much of the literature in this area opens up many hypotheses for further investigation rather than providing non-conflicting data, what appears central to developing healthy eating behaviours is exposure in a positive environment to healthful foods. Children come to like what is familiar and what is familiar is what is present in the environment. A child cannot learn to like something they are not exposed to. What is yet to be explored fully is the causality of eating behaviours.
Many parents report buying foods that they know their children will like so as to avoid wastage. Therefore, although the child will come to like what is in the environment, their preferences also shape what is in the environment.

**Influence of television and food promotion**

The influence of television viewing and food promotion and marketing has attracted a great deal of attention internationally. Television viewing is recognised as a sedentary behaviour and is associated with unhealthy eating behaviours. Within Australia watching two or more hours of television daily is associated with significantly more unhealthy eating behaviours (greater intake of takeaway foods, high energy drinks, sweet and savoury snacks, total energy and less fruit and vegetable intake) and significantly less likelihood of engaging in organised physical activity (Campbell, Crawford et al. 2002; Salmon, Campbell et al. 2006). Repeat exposure to aggressive marketing of energy dense and fast-foods during viewing time is likely to contribute to the development of these unhealthy eating behaviours. In a study by Dibbs and Harris (1996), Australia was found to have the highest number of food advertisements per hour compared with other developed countries; however, a recent review of 11 countries indicated that Australia now ranks 4th in the world along with the US and UK (Kelly et al. 2010) (Figure 6.1). The majority of advertisements continue to be for low nutrient, high energy products (Dibbs & Harris 1996; Zuppa, Morton et al. 2003; Carter 2006, Kelly et al. 2010). With comparison to type of food advertisements during child and adult viewing times, there are a significantly higher rate of high fat/high sugar foods (3.5 advertisements per hour) during children’s viewing time, compared to adults viewing time (2.9 advertisements per hour). Overall, during children’s viewing time, high-fat and high-sugar food advertisements make up 49% of total food advertisements (Table 6.1). The most frequently advertised types of foods are fast-food restaurants (14.5%), confectionary (12.2%), dairy products (9.6%) and bread, cereals, rice and pasta (8.7%) (Figure 6.2 and Table 6.1).

![Figure 6.1 Food advertisements per hour during children's peak viewing time by country](image-url)
This advertising exists despite the Children’s Television Standards of the Australian Broadcasting Authority’s ban on advertising during broadcast of preschool classified programs and time limits on advertising during children’s classified programs. As the peak children’s television viewings times are between 7–8 pm with G-classified programs predominating, the current regulations may not effectively restrict advertising as intended. Younger children are particularly vulnerable to advertising as they have been shown to have high levels of trust in advertised messages and are ignorant of their purpose and intent. By the age of 12 years, children report being more cynical of advertisements and less requests are made for the advertised product (Carter 2006). The Australian Medical Association, the Royal Australasian College of General Practitioners, The Australian Consumers Association, Nutrition Australia and others have called for restrictions or outright bans on food advertising aimed at children.
Table 6.1 The frequency of food categories advertised during children's viewing hours and non-children's viewing hours in Sydney

<table>
<thead>
<tr>
<th>Food category</th>
<th>% of food advertisements</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Viewing band 1</td>
</tr>
<tr>
<td>Core foods</td>
<td>34.2</td>
</tr>
<tr>
<td>Dairy</td>
<td>9.6*</td>
</tr>
<tr>
<td>Fruit and vegetables</td>
<td>3.0</td>
</tr>
<tr>
<td>Meat, fish, poultry</td>
<td>4.4</td>
</tr>
<tr>
<td>Breakfast cereals, rice, pasta</td>
<td>8.7</td>
</tr>
<tr>
<td>Baby foods</td>
<td>1.1***</td>
</tr>
<tr>
<td>Core food combined</td>
<td>7.4</td>
</tr>
<tr>
<td>High-fat/high-sugar foods</td>
<td>48.6***</td>
</tr>
<tr>
<td>Confectionery</td>
<td>12.2</td>
</tr>
<tr>
<td>Fast-food restaurants</td>
<td>14.5***</td>
</tr>
<tr>
<td>Cakes, biscuits, museli bars</td>
<td>6.7</td>
</tr>
<tr>
<td>High-fat/high-sugar spreads</td>
<td>0.0</td>
</tr>
<tr>
<td>Breakfast cereals (&gt;20% sugar)</td>
<td>6.1</td>
</tr>
<tr>
<td>Sugared drinks</td>
<td>3.6***</td>
</tr>
<tr>
<td>Frozen/fried potato products</td>
<td>0.0</td>
</tr>
<tr>
<td>Juice</td>
<td>1.0***</td>
</tr>
<tr>
<td>Savoury crisps and pastries</td>
<td>3.1</td>
</tr>
<tr>
<td>Desserts</td>
<td>1.4</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>17.3***</td>
</tr>
<tr>
<td>Recipe helpers</td>
<td>6.0</td>
</tr>
<tr>
<td>Miscellaneous</td>
<td>10.7***</td>
</tr>
<tr>
<td>Alcohol</td>
<td>0.9</td>
</tr>
</tbody>
</table>

†Viewing band 1 includes Monday–Friday 07.00–08.00, 16.00–20.30; and Saturday–Sunday 07.00–20.30. || Includes breakfast cereals with <20% sugar. §Includes tea, coffee, alcohol, supermarkets, local restaurants/cages, supplements, vegemite and throat lozenges. *P < 0.05, ** P < 0.001.

While there is still debate regarding the role of marketing on the development of unhealthy eating practices, a joint report from the World Health Organization and the Food and Agriculture Organization concluded that our current environment of aggressive marketing is considered a ‘probable’ causal factor in weight gain and obesity (WHO 2003; Salmon, Campbell et al. 2006). Two key reviews of the effects of food advertising to children have been published (Hastings, Stead et al. 2004; McGinnis, Gootman et al. 2006). These reviews observe that food is promoted to children more than any other product, expect for toys during the Christmas period, television was the primary medium and the top five advertised products were sugared breakfast cereals, soft-drinks, confectionary, savoury snacks and fast foods (Hastings, Stead et al. 2004). The review found that this advertising had little or no effect on children’s perception of a healthy diet but it did influence children’s nutritional knowledge. For example, exposure to soft drink and breakfast cereal advertisements decreased children’s ability to determine correctly if a product contained real fruit. Advertising was found to influence food preferences and purchasing behaviours with children showing a greater liking for foods seen during advertisements. In addition to television advertising, in-school marketing, sponsorship, product placement, internet marketing and sales promotion on food packaging combine to promote an environment which makes healthy food choices difficult and less appealing. A recent survey of Sydney supermarkets found that television, movie celebrities and cartoon characters were the most common form of food promotion and that 82% of the promotions were for unhealthy foods (Chapman, Nicholas et al. 2006).
Urgent calls for changes to laws governing food advertising to children have been made around the world (Chapman, Nicholas et al. 2006; Nestle 2006). Despite the influence of such aggressive marketing, a recent study of parents of school aged children in Melbourne has shown that although the majority of parents recognise the strong influence of marketing on children’s request for food items, the manner in which parents responded to these requests varied (Campbell, Crawford et al. 2006). Some parents indulged the requests while others refused to purchase the items, suggesting that the influence of marketing is indirect with parents’ management being the vital step between advertising, purchase and eating.

Please now read Nestle (2006), ‘Food marketing and childhood obesity – a matter of policy’.

Adolescent eating patterns

The hallmark of adolescence is change. Important consequences of these changes include the assumption by adolescents of increasing control over their own eating patterns, diminishing family influence on food choice and increasing influence of their peer group. It is not unusual for these eating patterns to be based on food choices made on the basis of hedonic, social and status considerations rather than on nutritional content and possible longer term consequences for personal health.

Consumption of snack foods

Consumption of snacks both between and instead of meals is a common feature of adolescent diets. There is often concern that this kind of eating pattern cannot meet nutritional requirements. Clearly, the nutritional implications of such a pattern depend on the actual composition and combination of foods in the diet over a period of time.

An important fact that is often overlooked is that the adolescent actually has a higher total energy requirement than the young adult. Adolescent energy requirements are on average about 1000 kJ per day higher than those of adults, and therefore permit the consumption of at least some higher-energy density foods.

Although many snacks (chocolate bars, potato chips and crisps, cakes, pies, biscuits and soft drinks) may be high in fat, sugar and energy density, and correspondingly low in nutrient density, this is not an invariable feature of snacks. Fruit, cheese, sandwiches, breakfast cereals, egg, meat and fish can all be eaten in the form of snacks which have a relatively high nutrient density.

It is important, therefore, to distinguish between the question of eating snacks (as opposed to meals), which does not necessarily constitute a problem if a wide selection of foods is chosen, and the question of the reported consumption of only a limited number of snack foods, in particular those with high energy and low nutrient density, which may lead either to obesity or low nutrient intake or both. Table 6.2 shows the energy and nutrient density of some common snack foods.
### Table 6.2 Energy content and nutrient density of common snack foods

<table>
<thead>
<tr>
<th>Food</th>
<th>Energy (kJ)</th>
<th>Calcium (mg)</th>
<th>Iron (mg)</th>
<th>Vit. A (µg)</th>
<th>Vit. C (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hamburger with cheese &amp; salad</td>
<td>1820</td>
<td>1552</td>
<td>2.8</td>
<td>178</td>
<td>3.90</td>
</tr>
<tr>
<td>Hot dog</td>
<td>1365</td>
<td>165</td>
<td>1.9</td>
<td>8</td>
<td>0</td>
</tr>
<tr>
<td>Meat pie</td>
<td>1535</td>
<td>21</td>
<td>2.2</td>
<td>14</td>
<td>0</td>
</tr>
<tr>
<td>Small spring roll</td>
<td>610</td>
<td>17</td>
<td>1.3</td>
<td>0</td>
<td>0.6</td>
</tr>
<tr>
<td>Sandwich:</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cheese</td>
<td>1080</td>
<td>230</td>
<td>1.0</td>
<td>78</td>
<td>0.8</td>
</tr>
<tr>
<td>Ham</td>
<td>890</td>
<td>36</td>
<td>1.1</td>
<td>77</td>
<td>0</td>
</tr>
<tr>
<td>Egg &amp; lettuce</td>
<td>875</td>
<td>48</td>
<td>1.3</td>
<td>127</td>
<td>1.0</td>
</tr>
<tr>
<td>French fries (medium serve 100g)</td>
<td>1390</td>
<td>10</td>
<td>1.1</td>
<td>0</td>
<td>3.1</td>
</tr>
<tr>
<td>Corn chips (small packet 20 g)</td>
<td>405</td>
<td>22</td>
<td>0.4</td>
<td>2.6</td>
<td>0</td>
</tr>
<tr>
<td>Peanuts (30 g)</td>
<td>770</td>
<td>15</td>
<td>0.7</td>
<td>0.3</td>
<td>0</td>
</tr>
<tr>
<td>Sweet biscuits (2)</td>
<td>500</td>
<td>13</td>
<td>0.4</td>
<td>4.5</td>
<td>0</td>
</tr>
<tr>
<td>Ice-cream (2 scoops)</td>
<td>390</td>
<td>43</td>
<td>0.02</td>
<td>90</td>
<td>0</td>
</tr>
<tr>
<td>Fruit bun</td>
<td>925</td>
<td>39</td>
<td>2.0</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Mars bar</td>
<td>900</td>
<td>63</td>
<td>0.7</td>
<td>21</td>
<td>0</td>
</tr>
<tr>
<td>Milk chocolate (30 g)</td>
<td>275</td>
<td>26</td>
<td>0.07</td>
<td>50</td>
<td>0</td>
</tr>
<tr>
<td>Flavoured milk (300 mL)</td>
<td>1000</td>
<td>303</td>
<td>0.3</td>
<td>115</td>
<td>3</td>
</tr>
<tr>
<td>Orange juice (300 mL)</td>
<td>430</td>
<td>28</td>
<td>0.3</td>
<td>44</td>
<td>110</td>
</tr>
<tr>
<td>Cordial (300 mL)</td>
<td>200</td>
<td>1</td>
<td>0</td>
<td>0.3</td>
<td>1.5</td>
</tr>
<tr>
<td>Apple</td>
<td>310</td>
<td>7</td>
<td>0.3</td>
<td>1.6</td>
<td>8</td>
</tr>
<tr>
<td>Banana</td>
<td>335</td>
<td>5</td>
<td>0.3</td>
<td>14</td>
<td>10</td>
</tr>
<tr>
<td>Orange</td>
<td>200</td>
<td>33</td>
<td>0.5</td>
<td>18</td>
<td>69</td>
</tr>
</tbody>
</table>

(FoodWorks 2010)

Preceding sections have discussed the importance of family meal times on healthy eating behaviours. Eating together as a family is associated with a wide variety of healthful habits independent of dietary intake, including greater levels of socialisation, positive role modelling, healthful habits (i.e. a reduction in risk taking behaviours), and greater sense of family unity and connectedness. Regular family meals are associated with higher fruit and vegetable intake and lower intake of fried foods and soft drinks in adolescents. Adolescence is, however, a time characterised by decreasing family meal times with more families reporting dining with preteens than teenagers. The reasons for this appear to vary between families, but scheduling difficulties are frequently cited as reasons for not being able to eat together. Although family mealtimes decrease during this period in the
lifespan, both parents and adolescents report positive experiences eating with the family. A recent US study has shown that the majority of adolescents (63%), and their parents (98%) considered it important to eat a meal together; both groups reported mealtimes were enjoyable and had a positive atmosphere. Family characteristics associated with more meals together include decreased television viewing and a greater degree of reporting that mealtimes were a family priority (Fulkerson, Neumark-Sztainer et al. 2006). Table 6.3 summaries the differences and similarities between parents and adolescents perception of meal times.

### Table 6.3 Perception of mealtimes

<table>
<thead>
<tr>
<th></th>
<th>Adolescent</th>
<th>Parent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Important that our family eat a meal together</td>
<td>Strongly agree</td>
<td>23%, 83%</td>
</tr>
<tr>
<td>Dinner is more than food, we talk</td>
<td>Strongly agree</td>
<td>23</td>
</tr>
<tr>
<td>Brings people together in enjoyable way</td>
<td>Strongly agree</td>
<td>17</td>
</tr>
<tr>
<td>Expected to follow rules</td>
<td>Strongly agree</td>
<td>12</td>
</tr>
<tr>
<td>Manners are important</td>
<td>Strongly agree</td>
<td>30</td>
</tr>
</tbody>
</table>

(Fulkerson, Neumark-Sztainer et al. 2006)

Given the importance of family meal times on establishing healthy eating behaviours, it is important to continue to encourage time to eat together as a family. Given that parents and adolescents see this time together as important, emphasising pleasure and enjoyment during mealtimes, removing distractions and leaving discipline, money, and other stressors for another environment are effective strategies for making these experiences positive. Involving family members in meal preparation and planning, scheduling and planning in advance and being creative with the time of day or location are also effective.

### Across the life span

There is much research that is still needed in order to gather a greater understanding of the factors influencing eating behaviours. Many of the studies conducted to date are cross-sectional and do not allow us to determine causality of eating behaviours. There are many hypotheses that still require investigation; however, it is still possible to summarise some strategies that can positively or negatively influence eating behaviours. Table 6.4 outlines a ‘division of responsibility’ strategy which is often utilised in family settings to enhance positive eating behaviours.
Table 6.4  Division of responsibility

<table>
<thead>
<tr>
<th>Parents’ responsibility</th>
<th>Child’s responsibility</th>
</tr>
</thead>
<tbody>
<tr>
<td>What food is served</td>
<td>How much to eat</td>
</tr>
<tr>
<td>When it is served</td>
<td>Whether they eat any at all</td>
</tr>
<tr>
<td>Where it is served</td>
<td></td>
</tr>
</tbody>
</table>

(Adapted from Satter 1986)

It is important to recognise individual characteristics of the child. Each child is different and will like or dislike different foods, have different degrees of acceptance of variety and different degrees of enjoyment of food.

**Strategies for encouraging healthy eating behaviours**

- Children’s eating modified by:
  - Exposure and accessibility.
  - Modelling behaviour of peers, siblings and parents with the best evidence for parental modelling.
- Conditioned preferences:
  - Children prefer what is eaten when hungry, in social environments, and food used as rewards.
  - Child-feeding practices such as force feeding and restriction promotes preference for that food.
- Liking for fat, sugar salty foods enhanced by:
  - Environments where food is present.
  - Seeing role models eating it.
  - Having the food overly restricted.
- Parental directives to encourage or restrict consumption may be counter-productive:
  - May discourage child’s ability to respond to hunger and satiety cues.
  - The more restricted the more they like it, the more it is forced the less they like it.

**TEXT QUESTIONS**

1. Describe the terms food neophobia, conditioned preferences, pressure to eat and restriction and parental control.

2. A child care centre has contacted you and asked you for advice on how to encourage healthy eating in their centre. The centre provides meals for fifty children from the age of six months to five years. What strategies would you recommend for enhancing healthy eating in this age group?
3 ‘Missed meals’ and ‘snacking’ are typical features of adolescent eating patterns. What strategies could be employed to increase intake of nutrient dense snack foods and decrease the intake of energy dense nutrient poor foods for this age group?

4 What health benefits could be expected from decreasing television viewing hours during preschool and childhood years and during adolescence?

References


TOPIC 7

The adult years

PREPARED BY THE UNIT TEAM

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Introduction

The adult years commence at the age of 21 years and conclude when an individual turns 65. The priority of nutrition is to maintain body homeostasis, optimise general wellbeing and prevent the onset of chronic diseases of affluence, namely cardiovascular disease, obesity and diabetes.

Learning objectives

At the end of this section you should be familiar with, and have an understanding of the following aspects of adult males and adult females:

• the physiology of the adult
• nutrient requirements
• dietary intakes
• common chronic diseases in which diet has a role in the aetiology
• main nutritional concerns in adult females (pre and post menopausal) and adult males
• the main differences between males and females.

Learning resources

Print reading

The reading listed here is reproduced in the print readings for this unit.


Online reading

Access the reading listed here using your internet browser.


Deakin Studies Online (DSO)

Access the unit site on DSO <http://www.deakin.edu.au/current-students/> for the appendices and other additional resources you can use to supplement this topic.

Physiology of the adult

Physiological maturity

The adult years are characterised by the completion of sexual and reproductive development, and attainment of both full genetic size and a state of tissue
maintenance. Body tissues are in a state of flux, with some tissues undergoing a greater rate of turnover. In the younger years protein synthesis exceeds protein breakdown, whereas in adults protein breakdown eventually exceeds protein synthesis and one observes a gradual decline of body tissues.

**Body composition**

Body composition varies both within and between males and females, and is influenced by age, genetic predisposition, level of physical activity and nutrient intake. Water is by far the largest component of the human body and the percentage of water is generally higher in males than in females because of the proportionally greater muscle (75–80% water) mass in men and smaller amount of adipose tissue (8–10% water). Percentage body fat can be determined non-invasively by measuring skin fold thickness at various points around the body (e.g. tricep, suprailiac, pectoral and thigh; Gibson 2005). Data collected as part of the USA’s National Health and Nutrition Examination Survey (NHANES) indicates the change in fat mass in males and females with age (Figure 7.1). Adult females typically have a greater proportion of their body that is fat mass when compared to males. Elderly women (65 years) have a higher percentage of body fat when compared to younger women (20 years), 43% and 35% respectively (Figure 7.1) (Kelly et al. 2009). Similar data from a nationally representative sample of Australian adults is not available.

![Figure 7.1](image-url) % fat mass in Caucasian males and females

(Adapted from Kelly et al. 2009)

**Relationship between diet and health**

Dietary recommendations were originally developed to prevent the development of deficiency diseases, such as scurvy, which results from a deficiency of vitamin C. In Westernised countries like Australia, where food is in abundance for the majority of the population, we are seeing an escalation of diseases associated with overconsumption of particular food nutrients rather than insufficient intakes. These
diseases include coronary heart disease (CHD), non-insulin dependent diabetes (NIDDM), cerebrovascular disease, hypertension and cancer. Dietary factors are known to have a role in the aetiology of all of these diseases. However, it also known that genetic predisposition and other environmental factors (e.g. stress, physical activity and smoking) also play a role in disease development, and the interactions between these factors are complex. The Australian Dietary Guidelines were developed to try and reduce the prevalence of these diseases of affluence (NHMRC 2011). The following discusses selected nutrients and their relationship to the development of diseases common in developed countries generally resulting from overconsumption, and also common deficiency diseases such as iron deficiency anaemia. It will firstly look at nutritional issues common in adult females (pre and post-menopausal) and adult males. Even though we have chosen to discuss these issues separately, it is important to note that nutritional issues common in pre menopausal women, for example iron deficiency anaemia, can also be a problem in post-menopausal women.

**Adult women pre-menopause**

The pre-menopausal woman has been defined as a woman who falls in the age range of 19 to 45–55 years and has the ability to reproduce. Although adolescent females also have the ability to reproduce, their nutritional issues have already been discussed in the section on children and adolescents.

**Iron – additional requirements for women**

The obligatory daily iron losses due to desquamation of surface cells, in secretions and by small quantities of physiological blood loss amount to 0.9–1.0 mg. Pre-menopausal women will have additional losses of iron via menstruation. The median loss of iron during a menstrual cycle is 0.45 mg/day, with losses of 1.35 mg/day at the 90th percentile. The 2006 nutrient reference values (NRV) set the estimated average requirements (EAR) for adult by modelling the components of iron, estimating the requirements for absorbed iron at the fiftieth percentile with use of an upper limit of 18% iron absorption. The recommended daily intake (RDI) was set by modelling the components of iron requirements, estimating the requirement for absorbed iron at the 97.5 percentile, with use of an upper limit of 18% iron absorption. For the EAR and RDI for women, it was assumed that women over 50 years do not menstruate. Accordingly the 2006 RDI for women 18–50 years is 18 mg/day which falls to 8 mg/day over the age of 50 years. Absorption is higher from a mixed western diet including animal foods (about 18%) than a vegetarian diet (about 10%), so vegetarians will need intakes about 1.8 times higher.

The 1995 National Nutrition Survey provides useful information on the eating patterns of adult females (see Appendix 4 on DSO). The mean daily intake of iron was well below the RDI at every age range for pre-menopausal women. Women aged 19–24 years were consuming 10.4 and 13.8 mg of iron at the 25th and 50th centile, well below the RDI for iron (ABS 1998). The main source of iron for women aged 19 years and over was cereal and cereal products, at 29% of total dietary intake. Meat and meat products contributed 16.9% of iron. This percentage of iron from meat and meat product intake has reduced when compared to data collected during the 1983 National Nutrition Survey. The 1983 survey ascertained
that 27% of dietary iron was derived from meat and meat products. At the same time, mean daily meat and meat product intake decreased from 144 g/day during 1983 to 116 g/day during the most recent dietary survey of 1995. From these data, the majority of dietary iron is derived from non-haem iron sources, which contain a form of iron that is less efficiently absorbed when compared to haem iron.

**Zinc**

Zinc intakes also tend to be low in women and parallel the situation with iron, although the 2006 RDIs are lower than the 1991 RDIs and are 8 mg/day compared to previous level of 12 mg/day. The recommended intake was estimated by summing the estimated non-intestinal zinc losses (urinary, integument, semen for men) and the intestinal losses to derive total endogenous losses. The EAR was then estimated assuming an absorption of 24% for men, 31% for women, and with a further safety factor applied. Absorption is higher from animal foods than plants sources, so vegetarians, particularly strict vegetarians, will need intakes about 50% higher.

**ONLINE READING**

Please now read section ‘2.4 Lean meat and poultry, fish, eggs, nuts and seeds, and legumes/beans’ (pp. 51–61) in the draft *Australian Dietary Guidelines*.

These foods are emphasised as a source of dietary iron, zinc and B₁₂ as well as protein.

**Diets and underweight**

Please now read section ‘4. Achieve and maintain a healthy weight’ (pp. 102–30) in the draft *Australian Dietary Guidelines*.

Modest weight losses, 5–10% of total body weight, can reduce the risk of developing chronic diseases such as non insulin dependent diabetes and coronary heart disease. However, excessive weight loss can lead to ill health. The weight loss industry in Australia is a multimillion dollar industry, mainly attracting women. Women in Westernised societies are vulnerable to mass media messages that say a thin body is a more beautiful body and this equates with being a more successful and well-liked individual. This can ultimately lead to poor self esteem, disordered eating and poor dieting practices. Rapid weight loss resulting from poor dietary practices is generally followed by weight gain in the form of adipose tissue, usually a greater amount of weight than what was initially lost, and this is commonly called ‘weight-cycling’. This type of dieting behaviour and consequently weight gain in females may predispose them to an increased risk of developing NIDDM, CHD, hypertension and hyperlipidaemia, conditions associated with overweight and obesity.
The 1995 National Nutrition Survey questioned respondents about the type of diet they were currently following. 7.6% of women aged 19–24 years were on a weight reduction diet, compared to 1.0% of men. This increased to 8.7% of women aged 25–44 years, 2.5% in men of the same age range (ABS 1998).

Being underweight, a body mass index (BMI) <18.5, is also associated with health risks, resulting from the development of amenorrhoea, hypokalaemia and psychological disturbances. 5.4% of women aged 19–24 years of age and 2.2% of women aged 25–44 years were underweight (ABS 1998).

**Alcohol**

Please now read section ‘3.4 Alcoholic drinks’ (pp. 92–101) in the draft *Australian Dietary Guidelines*.

The following discussion will look at the issue of alcohol intake in both men and women. Excessive alcohol intake can lead to overweight and obesity, increase blood pressure and is a risk factor for the development of cancer. Alcohol can also cause brain, pancreatic and liver damage. The Australian Dietary Guidelines recommend that you ‘limit your alcohol if you choose to drink’. The NHMRC recommends for healthy men and women, drinking no more than two standard drinks on any day reduces the lifetime risk of harm from alcohol-related disease or injury over a lifetime. Drinking no more than four standard drinks on a single occasion reduces the risk of alcohol-related injury arising from that occasion. (NHMRC 2009).

**Table 7.1**

<table>
<thead>
<tr>
<th>Common alcoholic beverages containing 10 g of alcohol (one standard drink)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Alcoholic beverage</strong></td>
</tr>
<tr>
<td>Beer</td>
</tr>
<tr>
<td>low alcohol (2–3%)</td>
</tr>
<tr>
<td>standard (4–5%)</td>
</tr>
<tr>
<td>Table wine</td>
</tr>
<tr>
<td>(approx. 10%)</td>
</tr>
<tr>
<td>Sparkling wine</td>
</tr>
<tr>
<td>(approx. 12%)</td>
</tr>
<tr>
<td>Fortified wines</td>
</tr>
<tr>
<td>(sherry/port, approx 20%)</td>
</tr>
<tr>
<td>Spirits, liqueurs</td>
</tr>
<tr>
<td>(approx. 40%)</td>
</tr>
</tbody>
</table>

(NHMRC 2009, p. 143)

The mean daily intake of alcohol was 6.6 g for women aged 19–24 years (0.6 of a standard drink) and 15.2 g for men of the same age range (1.5 standard drinks), and these intakes increased as age increased (see Appendix 4 on DSO). These data would seem to suggest that both men and women consume alcohol in safe levels.
However, it is known that some individuals consume in excess of these amounts. The NHMRC reports that approximately half of the adult Australian population drink at levels that place them at high risk of short-term alcohol-related harm. Approximately one-quarter of Australian adults drink at levels that place them at high risk of long-term alcohol-related harm (NHMRC 2009). The lifetime risk of death from alcohol-related diseases increases with increasing volume intake, the risk more than triples when consumption increases from two to three standard drinks per day. Women are at a greater risk of alcohol-related disease deaths than males (NHMRC 2009). Figure 7.2 displays the lifetime risk of alcohol-related disease deaths per 100 people who drink at each level (i.e. number of standard drinks/day). For those who regularly drink two standard drinks per day, the lifetime risk of death from an alcohol-related disease is about 0.4 in 100 people with that drinking pattern. For each additional standard drink consumed per day the risk increases, and is above 1 in 100 at three drinks per day. There is a greater increase in risk for women (NHMRC 2009).

**Figure 7.2**

*Lifetime risk of death from alcohol-related disease per 100 drinkers, by number of standard drinks per occasion, Australia 2002*

Breast cancer

The development of breast cancer in women involves a complex interplay of genetic, environmental and hormonal factors. It is known that 5–10% of women who develop breast cancer have a family history of this disease. Dietary factors linked to breast cancer include overweight, excess alcohol intake and potentially total fat intake. In their review of the literature, The World Cancer Research Fund (2008) concluded that there is limited evidence suggesting the consumption of total fat is a cause of postmenopausal breast cancer. However, because the role of excess body fatness is a well-established risk factor in breast, (as well as bowel and some other cancers), and high fat diets are known to contribute to excess body fatness, maintaining a healthy body weight through appropriate fat intakes is recommended to reduce the overall risk of breast cancer.
The oral contraceptive pill

The oral contraceptive pill has the potential to interact directly with vitamins and minerals or their metabolites and may influence the status of some vitamins and minerals. Oestrogen interferes with the binding of pyridoxal-5-phosphate, the functional form of vitamin B6, to kynurenine aminotransferase and kynureninase potentially leading to a secondary functional deficiency of vitamin B6. Some studies have shown a significant decrease in serum B12 and B6 levels but no change in folate or homocystine levels. Progesterone can increase appetite, alter carbohydrate metabolism and cause weight gain, while oestrogen can increase subcutaneous fat and cause fluid retention. However, a recent Cochrane review of oral contraceptive use and body weight found no evidence of a causal association between oral contraceptive use and weight gain. They did note however the limited available data (Gallo et al. 2006). Oral contraceptive agents may negatively alter serum lipid levels by raising total cholesterol and triglyceride levels, increasing the risk of developing CHD. Oral contraceptive use reduces menstrual blood loss by about 50% and can therefore favourably impact iron status.

Adult women post menopause

Menopause occurs between the ages of 45–55 years and signals the end of a female’s reproductive ability. Depletion of ovarian follicles results in a decline in ovarian oestrogen production. Adipose tissue, the liver and the adrenal cortex will still produce small amounts of oestrogen. Nevertheless insufficient oestrogen leads to a range of physical and emotional changes. The most obvious change is the cessation of ovarian and menstrual cycles. Beyond the reproductive system, lack of oestrogen has an impact on two major body systems, the skeleton and cardiovascular systems. The postmenopausal reduction in oestrogen leads to increased activity of the bone dissolving osteoclasts and diminished activity of the bone building osteoblasts. The end result is a decrease in bone density and a greater incidence of bone fractures. Postmenopausal oestrogen deficiency leads to a decrease in production of HDL-cholesterol and an increase in LDL-cholesterol, both of which increase the risk of developing atherosclerosis, a risk factor for having a heart attack.

Calcium and osteoporosis

The majority of calcium (99%) in the body is found in the skeleton. The daily accumulation of calcium in skeleton that commenced in infancy and peaked during adolescence continues until the age of 30–35 years. Calcium requirements in adults are based on the amount of calcium to maintain calcium balance and ultimately to optimise skeletal calcium levels. Failure to consume adequate levels of calcium from infancy to 30–35 years of age can lead to a state of negative calcium balance, where losses of calcium via urine, faeces or skin exceed calcium intake. In this situation calcium skeletal reserves will be utilised to maintain plasma calcium levels which will compromise the ability to attain optimum peak bone density. Osteoporosis can result; a condition characterised by a reduction in bone density, usually manifesting in the elderly years, particularly in postmenopausal women.
There is an increase in requirement for calcium after menopause (1300 mg/day). Postmenopausal women are at greater risk of being in a state of negative calcium balance which may be due to a decrease in intestinal calcium absorption and/or increase in urinary calcium excretion. Reduction in oestrogen, a feature of menopause, leads to increased activity of bone-dissolving osteoclasts and diminished activity of the bone-building osteoblasts, placing a post menopausal woman at greater risk of having a bone fracture. Please read the relevant section from your prescribed text regarding the role of calcium intake in the development of osteoporosis. Note any studies that have particularly looked at post menopausal women.

The mean intake for calcium falls below the RDI at every age range, particularly the 45- to 64-year-old age group where the requirements of some women would be 1300 mg/day. The percentile distributions of adjusted nutrient intakes describe the range of nutrient intake between the 10th and 90th percentiles. Women aged 45–64 years were consuming 419 and 543 mg of calcium at the 10th and 25th centile, well below the RDI (1991) for calcium (ABS 1998).

It is important to mention vitamin D and the role this fat soluble vitamin has in calcium homeostasis. Calcitriol synthesised in the kidney is considered the active form of vitamin D and functions like a steroid hormone. Calcitriol induces production of proteins involved in calcium absorption in the small intestine and has been proposed to be involved in the parathyroid hormone stimulation of calcium and phosphorus reabsorption in the distal renal tubule. Vitamin D deficiency in the adult years is generally thought to be rare, except for those individuals who may spend extensive periods of time indoors. Although those with darker skin, particularly veiled women, have been found to have high rates of vitamin D deficiency, and a recent study in Geelong found low levels of serum vitamin D in 22% of younger women at the end of winter.

Other non-dietary factors can influence the development of osteoporosis. Regular weight bearing exercise, such as walking, is known to be protective against the development of osteoporosis. Smoking has a negative effect by reducing oestrogen production and consequently calcium absorption.

**Adult women postmenopause and adult men**

Obesity and coronary heart disease are a concern in both post menopausal women and adult men.

**Energy expenditure, energy intake, exercise and the development of overweight and obesity**

Please now read section ‘4. Achieve and maintain a healthy weight’ (pp. 102-130) in the draft *Australian Dietary Guidelines*. 
Energy expenditure and energy intake

The components of energy expenditure in adults are basal metabolic rate (BMR), physical activity, the thermic effect of food and facultative thermogenesis. Total energy expenditure can be estimated by calculating BMR using prediction equations and multiplying this BMR by the appropriate level of activity.

e.g. Male, 25 years old, 75 kg, very sedentary

\[
\text{BMR} = 0.063 \times 75 + 2.896 = 7.6 \text{ MJ/day}
\]

\[
\text{BMR} \times 1.3 \text{ (activity factor)} = 9.9 \text{ MJ/day}
\]

Note that theoretical energy requirements estimated using prediction equations decrease with age, 6.5–12.0 MJ/day for a woman 31–50 years, 160 cm in height and 56.3 kg in weight, compared to 6.0–10.9 MJ/day for a woman of the same body proportions 51 years or over (see Appendix 2 on DSO). The doubly-labelled water technique can measure total energy expenditure in free-living adults over an extended period of time, 1–3 weeks. This technique can provide a more accurate estimate of energy expenditure when compared to prediction equations. The disadvantages of this technique are that it is expensive, the isotope is not readily available, and a mass spectrometer is required for analysis.

The decline in expenditure can be attributed to a decrease in BMR and physical activity. Poelhman (1993) has reviewed factors regulating energy expenditure in ageing humans. BMR, which constitutes approximately 60–75% of daily energy expenditure, is known to decline with age in healthy women, and is most pronounced between the ages of 51–81 years. This equates to a 0.6% decline per decade in women aged 18–50 years and this increased to a 4% decline per decade in women aged 51–81 years of age.

Men also exhibit a decline in BMR, but this is more pronounced at an earlier age (40 years) when compared to women. In addition, BMR declines at a greater rate in men after the age of forty. Physical activity is the second component of energy expenditure that can vary among adult males and females.

The 2007–2008 National Health Survey (ABS 2009) is the fourth in a series of cross-sectional surveys designed to obtain national benchmarks on a range of health issues, including physical activity levels. The sample included approximately 20, 800 people across all states and territories. Almost half (48%) of participants stated that they had walked for exercise in the two weeks preceding the survey, 36% engaged in some form of moderate exercise and 15% engaged in vigorous exercise (ABS 2009). Females were more likely to report walking for exercise (51%) than males (44%). Whereas males were more likely than females to do moderate exercise (38% vs 33%) and vigorous exercise (19% vs 11%) (ABS 2009). The percentage engaged in vigorous exercise during leisure time by age group is shown in Figure 7.3. Three or more sessions of vigorous exercise per week at an average of 20 minutes are considered sufficient with regards to reducing the risk of developing CVD.
The mean daily energy intake decreases with age for both adult males and females (see Appendix 4 on DSO).

**Overweight and obesity**

Overweight and obesity result from a chronic state of positive energy balance. When energy intake exceeds energy expenditure, excess energy is generally stored as adipose tissue.

The body mass index \([\text{weight (kg)} / \text{height (m)}^2]\) indicates the degree of overweight. An individual who is overweight or obese is at greater risk of developing non-insulin dependent diabetes mellitus, coronary heart disease, hypertension, gall bladder disease and some types of cancer (gall bladder, endometrial, ovarian and breast cancer). A BMI of 20–25 inclusive is defined as the acceptable or healthy weight range as a number of prospective studies have indicated this BMI is associated with the lowest death rate.

<table>
<thead>
<tr>
<th>Classification of BMI</th>
<th>Body mass index (kg/m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>underweight</td>
<td>&lt; 18.5</td>
</tr>
<tr>
<td>normal range</td>
<td>18.5–24.9</td>
</tr>
<tr>
<td>overweight</td>
<td>&gt; 25.0–29.9</td>
</tr>
<tr>
<td>obese</td>
<td>above 30</td>
</tr>
</tbody>
</table>

(WHO classification; AIHW 2010, p. 113)
The Australian Diabetes, Obesity and Lifestyle Study (AusDiab) provides recent data on the prevalence of overweight and obesity in Australia. Data from 2001 for the preceding 20 years indicates that the proportion of all adults aged 25–64 who are overweight or obese is rising steadily (Figure 7.4). This data is confined to people from capital cities and therefore may not be reflective of overweight and obesity in rural Australia. Indigenous people are much more likely to be obese than non-Indigenous people in Australia. In 1995, 24% of Aboriginal and Torres Strait Islanders 18 years and over were obese compared with 12% of other Australians. In 2001, 31% of Indigenous people were considered obese compared with 16% of other Australians (ABS 2002).

Figure 7.4  
Trends in the age-standardised prevalence (%) of overweight and obesity: 1980–2000

![Figure 7.4](image)

Figure 7.5  
Trends in the age-standardised prevalence (%) of obesity

![Figure 7.5](image)

*(Age standardised to 1991 Australian population*.

*(BMI > 30 kg/m²)*.

(Dunstan et al. 2001, p. 15).

(Adapted from Dunstan et al. 2001)
For males, the prevalence of being at least overweight increased by 17.5% between 1980 and 2000, while the prevalence of obesity increased by 10%. For females, the same figures were 18% and 12% respectively. For men this has been a more than doubling in obesity in that 20 year period and for women this represents a near tripling.

The mean weight of males and females increases with age and men weigh more than women at every age range. Figures 7.6 and 7.7 show that rates of overweight and obesity increase with age, peaking in the 55–74 year age group. When overweight and obesity are grouped together there are more overweight and obese men than women in every age group. However, there are more obese women (BMI >30) than men in every age bracket except for the 25- to 34-year-old group. Overall, 19% of all males were obese in 2000 compared to 22% of all females.

**Figure 7.6** Prevalence of overweight and obesity (BMI ≥ 25 kg/m²) of males and females

(Adapted from Dunstan et al. 2001)

**Figure 7.7** Prevalence of obesity (BMI ≥30kg/m²) of males and females

(Adapted from Dunstan et al. 2001)
Please read Stubbs and Lee (2004), ‘The obesity epidemic: both energy intake and physical activity contribute’. This paper discusses the relative contributions of increased total energy intake and reduced physical activity to the worldwide energy crisis. Emerging data published after 2004 suggests that obesity levels might be rising in populations where physical activity remains unchanged or has actually increased (Swinburn et al. 2009).

Summarise the main contributors to obesity in Australia.

**Fat and coronary heart disease**

Please now read section ‘3.1 Limiting intake of foods and drinks containing saturated and trans fat’ (pp. 76–81) in the draft *Australian Dietary Guidelines*.

High fat diets (usually high in saturated fat) are more energy density and are associated with weight gain and eventually obesity. Saturated fatty acids can increase both total and LDL-cholesterol, which both increase the risk of developing coronary heart disease.

The 2006 NRVs recommend a total dietary fat intake between 20–35% of energy. The lower end of the range is determined by the amount required to sustain body weight and to allow for intakes of estimated average requirements of micronutrients. Some communities, notably some Asian groups do have average fat intakes below this level but they are often smaller in stature and their overall nutrient status is not always known. The upper level was set in relation to risk of obesity and cardiovascular disease, bearing in mind that high fat diets are often high in saturated fat, a known risk factor for heart disease, and are also often energy dense increasing a propensity to over consumption of energy. Saturated fats should be limited to no more than 10% energy. The 2006 NRVs have recommended intake of linoleic acid (n-6 fats) which equates to 4–5% dietary energy. The recommended intake of α-linolenic acid (n3 fat) equates to 0.4–0.5% dietary energy of 90th centile of population intake. The recommendations for n-3 and n-6 fats are based on intakes to help optimise chronic disease risk, notably coronary heart disease.

The National Nutrition Survey ascertained that the mean per cent contribution of fat to total energy intake was 31–33% for both males and females. Appendix 4 on DSO highlights that intakes of saturated fat were above optimal levels, and that the ratio for the three different types of fats was very similar for both males and females across all age groups. The main food group contributing to fat intake for both males and females was the meat/meat equivalent group, 23.7% for men and 19.8% for females.

**Waist to hip ratio**

The waist to hip ratio (WHR) is an indicator of the distribution of body fat. A WHR $> 0.8$ in women and $> 0.9$ in men is indicative of a body fat distribution around the abdomen (android obesity) and is associated with an increased risk of developing NIDDM, CHD, hypertension, hyper-triglyceridemia and low HDL-cholesterol levels. Gynoid obesity is characterised by a weight distribution around the hips and...
is associated with a lower risk of chronic diseases such as CHD. Men typically present with android obesity whereas women are more likely to develop gynoid obesity, and this may explain the gender differences in disease prevalence. The percentage of males and females with WHR above 0.9 and 0.8 respectively is known to increase with age (Table 7.3).

Table 7.3  Waist to hip ratio, National Nutrition Survey, 1995

<table>
<thead>
<tr>
<th>WHR</th>
<th>19–24 years</th>
<th>25–44 years</th>
<th>45–65 years</th>
</tr>
</thead>
<tbody>
<tr>
<td>Females, &gt; 0.8</td>
<td>11.7 %</td>
<td>22.3 %</td>
<td>48 %</td>
</tr>
<tr>
<td>Males, &gt; 0.9</td>
<td>12.6 %</td>
<td>45.6 %</td>
<td>77 %</td>
</tr>
</tbody>
</table>

(Adapted from ABS 1998, pp. 111–12)

Sodium and hypertension

Please now read section ‘3.2 Limit intake of foods and drinks containing added salt’ (pp. 82–6) in the draft Australian Dietary Guidelines.

Cardiovascular disease was the most costly disease in Australia in 1993–1994, costing more than $3.7 billion or 12% of the total health bill. Hypertension is a major risk factor for cardiovascular disease, increasing the risk by two to four times. Blood pressure is known to rise with age in developed countries on high and relatively high sodium intakes. It has been estimated that 2.2 million adult Australians (17%) are hypertensive. Certain groups in the population are more sensitive to changes in dietary salt. They include older people, selected subgroups of patients with hypertension, those with a family history of hypertension, black American and individuals that have diabetes. There is evidence from the recent DASH sodium study that reducing dietary sodium even within the context of a low fat, high calcium high fruit and vegetable diet containing less red meat and increased legumes does effectively reduce blood pressure (Sacks et al 2001) supporting the case for reducing dietary sodium on a ‘healthy diet’.

The NRV recommendations advocate a general reduction in salt intake to less than 1600mg sodium per day (approximately 2/3 teaspoon of salt) for the general population. This is a significant reduction in recommended Sodium levels from the previous 1991 RDI. Sodium intakes were not measured in the 1995 National Nutrition Survey. The NHMRC working party on sodium estimated that the intake of sodium varied between 130–200 mmol/day, with some individuals consuming in excess of 400 mmol/day (NHMRC 1991). Major dietary contributors of sodium in the Australian diet include bread, cheese, butter, margarine, processed meats, take-away food and snack foods.
Dietary fibre

Please now read section ‘2.2 Plenty of vegetables, including different types and colours, and legumes/beans, and eat fruit’ (pp. 32–44) in the draft Australian Dietary Guidelines.

Diets high in dietary fibre can reduce the incidence of constipation, diverticular disease and colon cancer. In addition, high fibre foods are high in bulk and have a low energy density, both of which can minimise weight gain. The 2006 NRVs for dietary fibre recommend 25 g/day for women and 30 g/day for men. However the upper recommended level of 38g/day for men and 28g/day for women is recommended for reduction in coronary heart disease.

In the National Dietary survey women were consuming 70% of this recommendation; men had higher intakes but were still below this recommendation (see Appendix 4 on DSO).

The main food group contributing to fibre intake was the cereal and cereal products group, 34.9% for males (19 years and over) and 33.6% for females (19 years and over) (ABS 1998).

Indigenous health

Please now read section ‘A7.4 Aboriginal and Torres Strait Islander peoples’ (pp. 185–7) in the draft Australian Dietary Guidelines.

Significant differences exist between urban and remote aboriginal communities with respect to dietary intake and health status. The limited available data indicate that urban Aborigines add more salt to their diet and consume more takeaway meals, whereas in remote communities the consumption of sugar-sweetened beverages, sugar and white flour were significantly higher than non-indigenous consumption patterns. Nutrients of particular concern within remote communities are calcium, zinc, folic acid, vitamin E, beta-carotene and energy (generally high but in some remote communities very low energy intake levels have been documented). The current dietary guidelines are pertinent to Indigenous Australians; however, two additional recommendations are also made:

• choose store foods that are most like traditional bush foods
• enjoy traditional bush foods whenever possible.

Food requirements

The National Health and Medical Research Council have outlined recommendations for the amounts of food that, if consumed, would provide the estimated energy requirements and meet the EAR of all nutrients for men and non-pregnant women aged 19–50 years, of average height and with sedentary to
moderate activity levels (Dietitians Association of Australia 2011, NHMRC 2011a, NHMRC 2011b). These recommended food patterns are provided in Table 7.4. Men and women who are taller and more active should choose extra serves to meet their extra energy requirements.

**Table 7.4** Recommended dietary patterns for men and women aged 19–50 years of average height and with sedentary to moderate activity levels

<table>
<thead>
<tr>
<th>Food group</th>
<th>Example serving sizes</th>
<th>Recommended number of serves per day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vegetables and legumes / beans</td>
<td>1/2 cup cooked vegetables (75 g) 1 cup salad vegetables 1 small potato ½ cup cooked dried or canned beans, chickpeas or lentils, no added salt</td>
<td>Men</td>
</tr>
<tr>
<td>Fruits</td>
<td>1 medium piece of fresh fruit (150 g) 30 g dried fruit (e.g. 4 dried apricot halves) 1 cup canned fruit (150 g)</td>
<td></td>
</tr>
<tr>
<td>Grain (cereal) foods</td>
<td>1 slice of bread or ½ a medium roll or flat bread (about 40 g) ½ cup cooked rice, pasta, noodles ½ cup cooked porridge or polenta, 2/3 cup breakfast cereal flakes (30 g) or ½ cup muesli</td>
<td></td>
</tr>
<tr>
<td>Lean meat and poultry, fish, eggs, nuts and seeds, and legumes / beans</td>
<td>65 g cooked lean red meats or ½ cup of lean mince, 2 small chops 80 g cooked poultry 100 g cooked fish fillet or 1 small can of fish 2 large eggs 170 g tofu 30 g nuts or seeds</td>
<td></td>
</tr>
<tr>
<td>Milk, yoghurt, cheese and/or alternatives</td>
<td>250 mL cup of milk 200 g tub of yoghurt 40 g or 2 slices of cheese 120 g ricotta cheese</td>
<td></td>
</tr>
<tr>
<td>Additional serves from the five food groups or discretionary choices for those who are taller or more active</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

(NHMRC 2011)
1. Why are pre-menopausal women at risk of developing iron deficiency anaemia?

2. Why do females develop higher blood alcohol levels than males even when they consume equal volumes of alcohol?

3. How does menopause contribute to the development of osteoporosis? Can adequate calcium consumption prevent or slow down the progression of osteoporosis in the post menopausal years?

4. What factors contribute to the increase in BMI in adult females?

5. Compare the RDIs for adult males with adult females for the following nutrients: zinc, iron, calcium, folate and energy. Explain why there are differences or similarities with the RDIs.

6. Why is physical activity important for adult females and males?

7. Summarise in a table the main nutritional concerns for pre-menopausal women, post-menopausal women and adult men.

8. Summarise the main issues that contribute to the development of obesity in males and females. It may be useful to put this summary into a flow diagram.

9. Why do adult men and post menopausal women have the same iron requirements?

10. What are the limitations of using equations to estimate energy expenditure?

---

**Activity: Background**

11. Michael is a 21-year-old university student. He has just had a nutritional assessment, the details of which are outlined below.

**Family history**

- Father – recent heart attack, overweight, apple shaped.

**Anthropometric/biochemical**

- Height 170 cm, weight 77 kg, waist 82 cm, maximal gluteal 78 cm
- Weight 12/12 ago 71 kg.
- Cholesterol elevated.

**Dietary intake (refer to dietary analysis)**

- Breakfast
  - Misses four days out of seven
  - On other days:
    - Nutrigrain (large bowl) with full-cream milk
    - 2 slices of toast (thick white) butter and vegemite.
- Mid morning
  - Nil or sports drink e.g. Gatorade 600 mL
  - Sometimes a muesli bar.
• Lunch
  - Buys lunch at university
  - 1 medium pizza
  - or stir fried beef and fried rice
  - or meat pie
  - or schnitzel in white roll and bucket of chips
  - plus milkshake or another sports drink.

• Afternoon
  - Usually hungry
  - Dry biscuits and cheese or potato crisps or Twisties.

• Evening meal
  - At home
  - Flatmate cooks rice/vegetables or noodles and vegetables.
    (Michael does not like the food, but does not know what else to cook)
  - No dessert.

• At work
  - Steak/chips or fish/chips or lasagne and salad.

• Supper
  - Usually feels hungry in the evening
  - Another bowl of Nutrigrain
  - or bread/toast/crumpets with butter and vegemite
  - Occasionally a bar of chocolate.

• Other foods/drinks
  - Eats fruit when he remembers, but his flatmate Veronica eats more of the fruit.
  - Finds it difficult to budget for food. They shop once a fortnight at the supermarket, but run out of food before the next big shop and have to buy from the local 7–11 or do without until the next shopping day.
  - They use different milks; Michael hates soy milk.
  - Michael could cook steak and vegetables or a roast meal (his mother taught him) but has no ideas for any vegetable-based dishes that his flatmate would also enjoy.
  - Twice a week he has 4–6 stubbies of beer in the evening.

ACTIVITY

• Michael spends most of his week attending lectures and tutorials, and studying in the library because he wants to finish top of his year.
ACTIVITY: QUESTIONS

(a) What is Michael's BMI? Is he underweight or overweight?
(b) What is Michael's waist to hip ratio?
(c) Comment on Michael's family history and biochemistry.
(d) Calculate an estimated energy expenditure for Michael using the Nutrient Reference Values Estimated Energy Requirements (refer to Appendix 2).

From Michael's dietary analysis:
(e) What foods are the major sources of the following nutrients in his diet? (Refer to Figure 7.8 and Figure 7.9 on the next page)

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Major food sources</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy</td>
<td></td>
</tr>
<tr>
<td>Fat</td>
<td></td>
</tr>
<tr>
<td>Protein</td>
<td></td>
</tr>
<tr>
<td>Carbohydrate</td>
<td></td>
</tr>
</tbody>
</table>

(f) Do you think he is consuming an excessive intake of energy?
(g) What would be the long-term effect of this level of energy intake?
(h) Is he at risk of consuming inadequate amounts of any nutrients?
(i) How does his per cent energy from protein, fat and carbohydrate differ from the current recommendations?
(j) Reflect on your responses to questions one to nine; comment on the presence of any risk factors which may place Michael at risk of developing any lifestyle disease.
Figure 7.8  Activity: nutrient intake

Name: Michael

Details:
Male, 21 yrs, 77 kgs
Sedentary activity

Body Mass Index (BMI):

<table>
<thead>
<tr>
<th>Nutrients (mean all days):</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight: 2525.5 g</td>
<td>Retinol: 2009.49 ug</td>
</tr>
<tr>
<td>Energy: 15393.26 Kj</td>
<td>B-Carotene Eq.: 5613.75 ug</td>
</tr>
<tr>
<td>Protein: 111.13 g</td>
<td>Total A Eq.: 2945.6 ug</td>
</tr>
<tr>
<td>Total Fat: 165.31 g</td>
<td>Vitamin C: 148.65 mg</td>
</tr>
<tr>
<td>Carbohydrate: 424.15 g</td>
<td>Thiamin: 5.75 mg</td>
</tr>
<tr>
<td>Alcohol: 0.0 g</td>
<td>Riboflavin: 6.48 mg</td>
</tr>
<tr>
<td>Dietary Fibre: 28.2 g</td>
<td>Niacin: 43.97 mg</td>
</tr>
<tr>
<td>Total Sugars: 147.34 g</td>
<td>Niacin Eq.: 66.16 mg</td>
</tr>
<tr>
<td>Starch: 275.85 g</td>
<td>Sodium: 5133.07 mg</td>
</tr>
<tr>
<td>Water: 1758.36 g</td>
<td>Potassium: 3845.75 mg</td>
</tr>
<tr>
<td>Cholesterol: 309.63 mg</td>
<td>Magnesium: 501.42 mg</td>
</tr>
<tr>
<td>Sat. Fat: 71.19 g</td>
<td>Calcium: 1361.57 mg</td>
</tr>
<tr>
<td>Mono. Fat: 54.34 g</td>
<td>Phosphorus: 2161.09 mg</td>
</tr>
<tr>
<td>Poly. Fat: 27.46 g</td>
<td>Iron: 21.08 mg</td>
</tr>
<tr>
<td></td>
<td>Zinc: 13.18 mg</td>
</tr>
</tbody>
</table>

(Lynn Riddell 2007, Deakin University)
<table>
<thead>
<tr>
<th>Food/recipe</th>
<th>Amount</th>
<th>Weight g</th>
<th>Energy KJ</th>
<th>Protein g</th>
<th>Fat g</th>
<th>Carb g</th>
<th>EtOH g</th>
<th>SatFat g</th>
<th>PolyFat g</th>
<th>Mono g</th>
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<tr>
<td><strong>Breakfast</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Kellogg's Nutri-grain</td>
<td>2 cup</td>
<td>60.00</td>
<td>939.00</td>
<td>10.68</td>
<td>1.56</td>
<td>41.82</td>
<td>0.00</td>
<td>0.24</td>
<td>0.60</td>
<td>0.30</td>
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<tr>
<td>Milk, Fluid, Whole</td>
<td>1 cup</td>
<td>257.50</td>
<td>700.40</td>
<td>8.50</td>
<td>9.79</td>
<td>12.10</td>
<td>0.00</td>
<td>6.44</td>
<td>0.26</td>
<td>2.58</td>
</tr>
<tr>
<td>Bread, White, toasted</td>
<td>2 regular slice</td>
<td>80.00</td>
<td>817.60</td>
<td>6.72</td>
<td>2.08</td>
<td>35.84</td>
<td>0.00</td>
<td>0.32</td>
<td>0.80</td>
<td>0.40</td>
</tr>
<tr>
<td>Vegemite</td>
<td>2 tsp</td>
<td>12.00</td>
<td>71.16</td>
<td>2.86</td>
<td>0.12</td>
<td>1.07</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>Butter, Regular</td>
<td>1 tb</td>
<td>19.20</td>
<td>585.60</td>
<td>0.12</td>
<td>15.74</td>
<td>0.13</td>
<td>0.00</td>
<td>10.37</td>
<td>0.40</td>
<td>4.17</td>
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<tr>
<td><strong>Mid Morning</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gatorade</td>
<td>1 bottle (500ml)</td>
<td>509.00</td>
<td>636.25</td>
<td>0.00</td>
<td>0.00</td>
<td>34.10</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
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<tr>
<td>Muesli Bar, Chocolate-</td>
<td>1 bar</td>
<td>32.00</td>
<td>552.32</td>
<td>2.08</td>
<td>6.37</td>
<td>16.38</td>
<td>0.00</td>
<td>3.49</td>
<td>0.51</td>
<td>2.05</td>
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<td></td>
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<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>Veal Steak, Crumbed,</td>
<td>150g</td>
<td>150.00</td>
<td>2881.50</td>
<td>23.70</td>
<td>59.70</td>
<td>15.15</td>
<td>0.00</td>
<td>18.15</td>
<td>13.50</td>
<td>23.55</td>
</tr>
<tr>
<td>Bread Roll, White</td>
<td>1 roll (&gt;10cm)</td>
<td>74.00</td>
<td>903.54</td>
<td>6.66</td>
<td>2.81</td>
<td>39.22</td>
<td>0.00</td>
<td>0.37</td>
<td>0.96</td>
<td>0.59</td>
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<tr>
<td>Lettuce, Common, Raw</td>
<td>1 large leaf</td>
<td>15.00</td>
<td>6.15</td>
<td>0.14</td>
<td>0.02</td>
<td>0.06</td>
<td>0.00</td>
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<td>Tomato, Raw</td>
<td>20g</td>
<td>20.00</td>
<td>13.20</td>
<td>0.20</td>
<td>0.02</td>
<td>0.38</td>
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<td>0.00</td>
<td>0.00</td>
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<td>Mayonnaise, Regular</td>
<td>15g</td>
<td>15.00</td>
<td>233.10</td>
<td>0.14</td>
<td>4.85</td>
<td>2.99</td>
<td>0.00</td>
<td>0.54</td>
<td>2.88</td>
<td>1.19</td>
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<td>Milk, Whole, Chocolate</td>
<td>1 carton</td>
<td>310.00</td>
<td>1029.20</td>
<td>10.23</td>
<td>11.16</td>
<td>27.59</td>
<td>0.00</td>
<td>7.44</td>
<td>0.31</td>
<td>3.10</td>
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<td><strong>Afternoon</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Crisps, Potato,</td>
<td>1 packet (30g)</td>
<td>30.00</td>
<td>679.50</td>
<td>2.13</td>
<td>10.02</td>
<td>14.40</td>
<td>0.00</td>
<td>4.29</td>
<td>1.14</td>
<td>4.14</td>
</tr>
<tr>
<td><strong>Dinner</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rice, Brown, Boiled</td>
<td>2 cup (cooked)</td>
<td>360.00</td>
<td>2311.20</td>
<td>11.52</td>
<td>3.60</td>
<td>114.48</td>
<td>0.00</td>
<td>0.72</td>
<td>1.44</td>
<td>1.08</td>
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<tr>
<td>Oil, Peanut</td>
<td>0.5 lb</td>
<td>9.10</td>
<td>336.70</td>
<td>0.00</td>
<td>9.10</td>
<td>0.00</td>
<td>0.00</td>
<td>1.65</td>
<td>3.06</td>
<td>3.99</td>
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<tr>
<td>Broccoli, Cooked</td>
<td>1 cup</td>
<td>102.00</td>
<td>140.76</td>
<td>4.79</td>
<td>0.31</td>
<td>0.61</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
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<tr>
<td>Carrot, Cooked</td>
<td>50g</td>
<td>50.00</td>
<td>70.50</td>
<td>0.45</td>
<td>0.05</td>
<td>2.90</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>Onion, Mature, Cooked</td>
<td>50g</td>
<td>50.00</td>
<td>76.00</td>
<td>0.90</td>
<td>0.05</td>
<td>3.10</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
<td>0.00</td>
</tr>
<tr>
<td>Bread, White</td>
<td>2 regular slice</td>
<td>64.00</td>
<td>654.08</td>
<td>5.38</td>
<td>1.66</td>
<td>28.67</td>
<td>0.00</td>
<td>0.26</td>
<td>0.64</td>
<td>0.32</td>
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<tr>
<td>Butter, Regular</td>
<td>1 tb</td>
<td>19.20</td>
<td>585.60</td>
<td>0.12</td>
<td>15.74</td>
<td>0.13</td>
<td>0.00</td>
<td>10.37</td>
<td>0.40</td>
<td>4.17</td>
</tr>
<tr>
<td><strong>Supper</strong></td>
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<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kellogg's Nutri-grain</td>
<td>1 cup</td>
<td>30.00</td>
<td>469.50</td>
<td>5.34</td>
<td>0.78</td>
<td>20.91</td>
<td>0.00</td>
<td>0.12</td>
<td>0.30</td>
<td>0.15</td>
</tr>
<tr>
<td>Milk, Fluid, Whole</td>
<td>1 cup</td>
<td>257.50</td>
<td>700.40</td>
<td>8.50</td>
<td>9.79</td>
<td>12.10</td>
<td>0.00</td>
<td>6.44</td>
<td>0.26</td>
<td>2.58</td>
</tr>
</tbody>
</table>
References


Australian Bureau of Statistics 2009, National health survey: Summary of results, 2007–2008 (Reissue), Table 14: Type of exercise undertaken in the previous two weeks, ABS, Canberra.

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Lifespan Nutrition


# Nutrition during pregnancy

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Introduction

Topic 2 focused on the nutritional requirements of the foetus during pregnancy. In this topic we examine the physiological changes and nutritional requirements during the period of gestation from the mother’s point of view. On completion of this section, it would be useful to briefly review topic 2 in order to have a complete understanding of what happens during pregnancy.

During pregnancy significant maternal physiological adjustments occur directed towards the transfer and storage of nutrients in the foetus. These adjustments result in altered nutrient metabolism, nutrient storage and tissue fluid concentrations of nutrients. As a consequence, indicators of nutritional status need to be interpreted differently in the pregnant woman. In general, the alterations in maternal metabolism result in improved absorption and utilisation of nutrients and mean that nutrient requirements are in effect less than would be expected.

Learning objectives

The aim of this topic is to give you an understanding of the factors that determine nutritional requirements during pregnancy and the influence of nutrition on the outcome of pregnancy. At the end of this section you should be familiar with the following aspects of nutrition in pregnancy:

• maternal physiological adjustments during pregnancy
• nutritional requirements during pregnancy
• the influence of maternal nutritional status and diet on the outcome of pregnancy
• common nutritional concerns
• considerations for assessing nutritional status.

Learning resources

Print readings

The readings listed here are reproduced in the print readings for this unit.


Deakin Studies Online (DSO)

Access the unit site on DSO <http://www.deakin.edu.au/current-students/> for the appendices and other additional resources you can use to supplement this topic.
The physiology of pregnancy

The components of maternal weight gain

The growth of the foetus and maternal tissues is usually assessed by monitoring maternal weight gain (Figure 8.1). Weight gain is relatively small during the first trimester (13 weeks) with a total gain of only 1–2 kg and increases in the second and third trimesters with an average gain of around 0.4 kg per week.

Figure 8.1 Pattern and components of maternal weight gain during pregnancy

(Pitkin 1976; reprinted in IOM & NRV 2009, p. 91)

In the first half of pregnancy about 80% of weight gain is due to maternal tissues. In the final weeks of pregnancy, from 40 weeks, the foetus accounts for about a quarter of the maternal weight gain (Table 8.1). The remainder is largely due to an increase in maternal extracellular fluid. Overall water accounts for well over half of the maternal weight gained during a normal pregnancy.
**Table 8.1** The components of weight gain during pregnancy

<table>
<thead>
<tr>
<th>Body component</th>
<th>Increase in weight (kg) at 40 weeks</th>
<th>Percentage (% of total weight gain)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Products of conception</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fetus</td>
<td>3.40</td>
<td>27.2</td>
</tr>
<tr>
<td>Placenta</td>
<td>0.65</td>
<td>5.2</td>
</tr>
<tr>
<td>Amniotic fluid</td>
<td>0.80</td>
<td>6.4</td>
</tr>
<tr>
<td>Maternal tissues</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Uterus</td>
<td>0.97</td>
<td>7.8</td>
</tr>
<tr>
<td>Mammary gland</td>
<td>0.41</td>
<td>3.3</td>
</tr>
<tr>
<td>Blood</td>
<td>1.25</td>
<td>10.0</td>
</tr>
<tr>
<td>Extracellular, extravascular fluid</td>
<td>1.68</td>
<td>13.4</td>
</tr>
<tr>
<td>Total weight gain</td>
<td>12.50</td>
<td>100.0</td>
</tr>
<tr>
<td>Assumed fat deposition</td>
<td>3.35</td>
<td>26.8</td>
</tr>
</tbody>
</table>

(British Nutrition Foundation 2006, p. 32)

The average total weight gain during pregnancy has traditionally been around 10–13 kg; however, new data supports current recommendations for weight gain during pregnancy which are dependent on pre-pregnancy BMI (Table 8.2). These figures differ for women having twins or triplets.

**Table 8.2** Recommendations for weight gain during pregnancy

<table>
<thead>
<tr>
<th>Pre-pregnancy BMI</th>
<th>Recommended weight gain</th>
</tr>
</thead>
<tbody>
<tr>
<td>Less than 18.5 kg/m²</td>
<td>12½ to 18kg</td>
</tr>
<tr>
<td>18.5 to 24.9 kg/m²</td>
<td>11½ to 16kg</td>
</tr>
<tr>
<td>25 to 29.9 kg/m²</td>
<td>7 to 11½ kg</td>
</tr>
<tr>
<td>Above 30 kg/m²</td>
<td>5 to 9kg</td>
</tr>
</tbody>
</table>

(Adapted from the Institute of Medicine 2005)

*Adipose tissue*

The increase in maternal adipose tissue mass during pregnancy varies with the maternal pre-pregnancy weight status and with the maternal dietary energy supply. The additional fat accumulates, subcutaneously at least, over the abdomen, back and upper thighs rather than on the upper and lower limbs. Normally this fat is mobilised in the post-partum period and provides an energy reserve for lactation. However, fat that is retained with increased parity (births) is deposited in the intraabdominal cavity which is a concern for women’s later health (Nohr 2008). Fat accumulation is greatest during the early part of pregnancy, when the demands of foetal metabolism are low, and reduces in late pregnancy when they are high, and thus has a smoothing effect on the energy demands of pregnancy. This fat is utilised as a source of fuel for the mother in order to conserve glucose for the foetus. Measurements of body composition in pregnancy (Pipe et al. 1979) indicate
that a considerable amount of adipose tissue is deposited during the first half of pregnancy. Kopp-Hoolihan et al. (1999) used a four compartment model to measure the amount of body fat gained in nine pregnant women. The gains in fat mass were varied and ranged from –0.6 to 10.6 kg, and this variability was not related to pre-pregnancy body composition or energy intake during pregnancy.

**Underweight women**

Women with a low pre-pregnancy weight have a higher risk of delivering small for gestational age (SGA) infants (Nohr 2008). The risk can be decreased by gaining more weight during pregnancy (Figure 8.2); however, high gestational weight gain during pregnancy carry other risks of complications including excess postpartum weight retention (Nohr 2008). Perinatal mortality is lowest in underweight women who gain the most weight (about 14 kg) during gestation (Naeye 1979) and a gain of this order is associated with a birth weight comparable to that of normal weight women. If the energy cost of tissue deposition is taken as 5 kJ/g, the energy requirement of underweight women would be about 150 kJ (36 kcal)/day higher than that of normal weight women throughout pregnancy.

**Overweight women**

In developed countries, like Australia, obesity is more likely to pose a problem. A woman who is more than 120% of expected weight for height is at risk for numerous complications during pregnancy, for example, hypertension, gestational diabetes, postpartum haemorrhage, caesarean section and a large for gestational age (LGA) infant who weighs more than 4000 g.

Neural tube defects are also more frequent in infants of obese mothers (Shaw et al. 1996). These LGA infants are at risk of injury during delivery and for higher neonatal mortality and morbidity.

The amount of weight obese women should gain during pregnancy has recently been determined from large, population-based, cohort studies. A study of over 298,000 women in Sweden in 2007 comparing pre-pregnancy BMI and amount of gestational weight gain to twelve obstetric and neonatal outcomes found that weight gain of 6 kg or less was safest for women with a BMI of 30 kg/m² or more (Cedergren 2007). Research of more than 120,000 obese women in the US found that limited or no weight gain in obese pregnant women has favourable pregnancy outcomes (Kiel et al. 2007).
Maternal tissue changes and physiological adjustments

Placenta

Although not a major component of maternal weight gain (~650 g), the placenta plays two major roles. Firstly, it is the source of hormones that regulate both foetal and maternal metabolism during pregnancy and, secondly, as we have already seen in Topic 3, it provides the means whereby oxygen and nutrients are delivered to the foetus and waste products are removed.

Early in pregnancy the cells of the trophoblast and subsequently the placenta manufacture a number of hormones. Because a great variety of regulatory hormones are synthesised in the placenta, the metabolic control exercised by the placenta during pregnancy is similar to that exercised by the pituitary gland throughout life. During pregnancy these hormones are responsible for ‘re-setting’ a number of homeostatic mechanisms with consequent changes in the retention, utilisation and excretion of nutrients in order to provide an uninterrupted supply of nutrients to the foetus.

Progesterone and oestrogen are two placental hormones that have major effects on maternal physiology during pregnancy. The chief action of progesterone is to cause relaxation of the smooth muscles of the uterus so that it can expand as the foetus grows, but it also has a relaxing effect on other smooth muscles in the body.

Relaxation of the muscle of the gastrointestinal tract reduces motility in the gut, allowing more time for nutrients to be absorbed. The slower movement is also a cause of constipation commonly experienced by pregnant women. More general metabolic effects of progesterone are to induce maternal fat deposition, reduce alveolar and arterial pCO₂ (to facilitate exchange of lung gases during respiration) and to increase renal sodium excretion.
During the early months of pregnancy the secretion of oestrogen is lower than that of progesterone but it rises sharply near term. Its role is to promote the growth and control the function of the uterus. However, there are also generalised effects as with progesterone. One of these is an alteration in the structure of connective tissue mucopolysaccharides which increases the affinity of the tissues for water and also has the effect of making the tissues more flexible. Together the hydroscopic effect of oestrogen and the sodium-losing effect of progesterone produce a confusing clinical picture of a pregnant woman’s fluid and electrolyte balance. Many pregnant women complain of excess fluid retention in the skin and mild oedema appears to be an accompaniment of normal pregnancy.

**Blood volume and composition**

During pregnancy the plasma volume may increase by as much as 50% above the usual non-pregnant volume of 2600 mL. The increase in plasma volume follows a sigmoid pattern with little change during the first 10 weeks and a linear increase during the second and early third trimester until a plateau is reached at 30–34 weeks.

Erythrocyte volume also expands, but to a lesser extent, and unlike the plasma volume it continues to expand until term. In a healthy woman not receiving iron supplements, the erythrocyte volume increases by about 20%. The early expansion of the red blood cell (RBC) mass probably protects the foetus from hypoxia. The stimulus for RBC expansion is unknown but increased levels of erythropoietin have been detected in the maternal serum as early as the eighth week of pregnancy and continue to increase until term, even in the absence of evidence for a reduced total oxygen-carrying capacity of the blood. Because the increase in RBC mass is less than the increase in blood volume, there is an associated fall in Hb level of around 1–3% and in haematocrit of around 5%. The amount of Hb in the red cells does not change and the cells are of normal size (normochromic and normocytic). Non-pregnant Hb values of 13–14 g/100 mL can drop to 10–11g/100 mL. A diagnosis of anaemia in pregnancy is usually not made unless Hb is below 11 g/100 mL.

Considerable variation is found between women, but in general, blood volume increases more in those with a small initial blood volume, in multigravidae and in cases of multiple births. If the iron supply is limited, the degree of expansion is reduced. For example, in one study, the total Hb iron increased by 570 mg in supplemented women but only by about half this amount in un-supplemented women. Serum levels of other constituents also change, and values for the pregnant woman must be interpreted in the light of the physiological effects of pregnancy. Changes in the rates of protein synthesis and expansion of the plasma volume cause the total serum protein to fall within the first three months of pregnancy. A plateau is reached in mid-pregnancy and most of the reduction is the result of a decline in the albumin fraction. The reduction in serum albumin changes the colloidal osmotic pressure of the blood and is another factor responsible for the tendency of pregnant women to accumulate extracellular fluid.

In contrast to the water-soluble nutrients, the fat-soluble nutrients generally show increases with pregnancy, for example triglycerides, cholesterol and free fatty acids (Figure 8.3).
Most of the increased cholesterol is in VLDL with lesser increases in LDL and HDL. Changes in serum calcium, iron and zinc tend to reflect changes in their transport proteins.

**Cardiovascular and renal function**

During pregnancy cardiac output increases by as much as one litre per minute because of peripheral vessel dilatation and the additional arterio-venous shunt provided by the placenta. Blood flow through the kidneys is also increased by about 80%, and the glomerular filtration rate (GFR) increases by 50% to facilitate the clearance of creatinine, urea and other waste products of foetal and maternal metabolism. The change in GFR is partially caused by the lower osmotic pressure of the plasma, which results from the fall in plasma albumin. As a consequence of these changes, substantial amounts of glucose, amino acids and water-soluble
vitamins appear in the urine during pregnancy. The most satisfactory explanation, at present, is that the high GFR presents the renal tubules with larger amounts of nutrients than can be reabsorbed.

**Respiration**

Oxygen requirements in the mother increase as a result of the increase in metabolic rate and tissue mass in the uterus and breasts. It is also noted that there is a more efficient exchange of gases in the alveoli of the lungs with a resultant increase in the oxygen-carrying capacity of the blood.

**Gastrointestinal function**

Progesterone causes a decrease in tone and motility of smooth muscles of the gastrointestinal tract. This can result in delayed gastric emptying and reverse peristalsis, which can eventually lead to gastro-oesophageal reflux. Another outcome is that absorption of nutrients from the small intestine is increased. Water absorption from the colon is also increased which can lead to constipation, and this is further exacerbated by the enlarging uterus on the gastrointestinal tract also making elimination difficult.

Other physiological changes include an increase in appetite, nausea and vomiting and alterations in the sense of taste.

**Metabolic adjustments**

Changes in the metabolism of carbohydrate, fat and protein metabolism occur during gestation so that fat becomes the major fuel source for the mother in order to spare glucose for the foetus. Glucose contributes 50–70% of energy requirements to the foetus during the last trimester. The rapid uptake of glucose by the foetus results in fasting blood glucose levels lower than what is observed in the non pregnant woman. Placental oestrogen and progesterone cause an increase in the storage of body fat in the mother during the second trimester. Lipolysis increases during the third trimester when foetal demands for glucose cause maternal plasma glucose levels to fall.

**Nutrient requirements in pregnancy**

The low plasma values of some nutrients and the tendency for the kidneys to excrete considerable amounts of other nutrients, together with the increased absorption of nutrients during pregnancy, means that the establishment of nutrient requirements during pregnancy cannot be based on the same criteria as apply in the non-pregnant state. For example, although plasma concentrations of some nutrients are decreased, the total circulating levels are in fact higher than before pregnancy. Since the reduced levels are characteristic of normal pregnancy in healthy, well-nourished women, it is unlikely that the reduced levels reflect maternal nutrient deficiency. It is much more likely that the altered circulating levels simply help to facilitate the transfer of nutrients from the mother to the foetus and to protect the foetus against fluctuations in the maternal nutrient supply.

**Recommended dietary intakes (RDI)**

The RDIs for almost all nutrients are increased to some extent during pregnancy. The maternal physiological adaptations that occur during pregnancy will mean the
requirements are relatively evenly spread throughout the course of pregnancy rather than being concentrated in the last trimester when foetal growth rate is at its maximum.

**Energy**

Changes in physical activity and an increase in basal metabolism influence energy requirements in the pregnant woman. Basal metabolic rate (BMR) usually rises by the fourth month of gestation and exceeds non-pregnant levels by 15–20% towards the end of gestation. The increase in BMR reflects the increase in maternal cardiac output dictated by the increase in oxygen demands by the foetus and the additional energy necessary to support the growth of foetal and maternal tissue.

**PRINT READING**

Please read Butte et al. (2004). Summarise the key information from this study. What are the advantages of using doubly-labelled water to assess total energy expenditure?

Studies that have used doubly-labelled water to assess total energy expenditure have provided further insights into the behavioural and metabolic adjustments which occur during pregnancy and lactation. One of the most important findings to emerge from these studies is that inter-individual variability in the energy cost of pregnancy is very large even within the same population. Figure 8.4 shows the variation observed, in the changes in BMR in pregnancy in six countries. From these studies, an increase in energy costs (BMR) is seen to vary across countries, and the greatest increase is observed in the final trimester of pregnancy.

![Cumulative increases in BMR in pregnant women](image_url)

(Adapted from Butte & King 2005)

Increases in total energy expenditure (TEE) and resting metabolic rate (RMR) are most pronounced in the second half of pregnancy. TEE and RMR were measured in
healthy Swedish women by the method of doubly-labelled water (Forsum et al. 1992). RMR showed a slight but non-significant increase in gestational weeks 16–18 and a larger and significant increase in gestational week 30. Pooled data shows that in healthy, well-nourished women, BMR increases from pre-pregnancy levels by 4.5, 10.8 and 24% for the first, second and third trimesters, respectively. In six longitudinal studies examining energy expenditure of pregnant women, TEE is seen to increase throughout pregnancy in proportion to an increase in body weight. TEE increased by 1, 6 & 19% and weight increased by 2, 8 & 18% over baseline in the first, second and third trimesters respectively. The increments in TEE in the first, second and third trimesters were 0.1, 0.4 and 1.5MJ/day, but it is important to note that this included studies conducted in developing countries (Butte 2005).

The energy cost of pregnancy varies with the amount and the composition of the maternal and foetal tissues deposited. On average it is estimated to be 1175 kJ per day in women with a gestational weight gain of around 12kg (Butte 2005). In practice, however, many women tend to report energy intakes which are considerably less than this. This is likely to be due in part to a reduction in physical activity during pregnancy, to varying levels of fat deposition and to lower increases in metabolic rate than previously estimated.

Usually no allowance is made for the extra energy cost associated with moving a heavier body during pregnancy. The 2006 nutrient reference values (NRV) recommend an additional 1.4 MJ/day during the second trimester and 1.9 MJ/day during the third trimester. This level of additional energy can easily be provided by a piece of fruit and a small sandwich. These items would also provide additional protein and therefore pregnancy does not require a marked increase in overall food intake.

**Protein**

The need for total nitrogen (N) during pregnancy can be estimated from the amount of protein deposited in the maternal and foetal tissues (925 g) which requires the retention of 148 g N. Since foetal and maternal lean tissue synthesis occurs primarily in the last half of pregnancy the N need is greatest at this time and has been estimated to be about 0.9 g/day. The need for essential amino acids has not been quantified in pregnancy. Increased N retention is achieved in pregnancy by a reduction in urinary N excretion primarily due to a decrease in urea N probably because a larger proportion of the available amino N is used for tissue synthesis rather than for deamination.

The 2006 NRV for protein during pregnancy recommend an additional 0.2 g/kg/day during the last two trimesters making the estimated average requirement (EAR) stage of 0.8 g/kg/day. The RDI is estimated using a CV (coefficient of variation) of 12% giving an RDI in the second and third trimesters of 1.00–1.02 g/kg/day or 60 g/day. Those aged 14–18 years having the higher requirement. The increased requirement for protein is based on estimates of the increase in nitrogen content of the maternal and foetal tissues but may not in fact be necessary for women who normally consume liberal amounts of protein in the diet. In Australia, protein deficiency during pregnancy is unlikely to occur except in association with energy deficiency. Excess protein intake during pregnancy is non-beneficial to the foetus and can in fact be harmful (Kramer & Kakuma 2003).
A review of all evidence in this area updated in 2010 shows that high protein supplementation in pregnancy is associated with an increased risk of babies which are small for gestational age and to a lesser extent with neonatal death.

**Micronutrient requirements**

The RDIs for most nutrients are increased in pregnancy. Exceptions are potassium, sodium, calcium and vitamin D. Requirements of some nutrients are linked to the increased energy metabolism and larger increases in other nutrients, for example iron, ascorbic acid and folate are recommended either to increase the margin of safety or to meet the unusually high needs thought to be imposed by the foetus especially during the last trimester. It is quite likely that, as with energy, the increased needs for these nutrients are actually spread throughout the period of pregnancy and are largely met by changes in the efficiency of absorption and utilisation of nutrients during pregnancy.

**Vitamins**

The RDIs for thiamin and riboflavin are slightly higher to allow for an additional energy allowance of around 10% during pregnancy and for the amount incorporated into maternal and foetal tissues. An apparent biochemical deficiency of riboflavin occurs in pregnancy as evidenced by a slightly elevated erythrocyte glutathione reductase (EGR) coefficient. However, this elevation is not associated with a decrease in the basal activity of EGR as in clinical riboflavin deficiency. Iron-deficiency anaemia is associated with an increase in EGR activity, and could be the mechanism for the observed increase in EGR during pregnancy. The activation coefficient, however, does not rise progressively with the period of pregnancy.

Niacin metabolism is also altered during pregnancy, as evidenced by an increased urinary excretion of N\(^1\)-methylnicotinamide. The change in excretion is hormonally mediated and may be due to an enhanced capacity for the biosynthesis of nicotinate ribonucleotide from tryptophan during pregnancy, or to increased methylation of niacin in pregnant women resulting in an increased niacin requirement.

Tryptophan metabolism may also be enhanced during pregnancy and it is possible that the tryptophan-niacin conversion may be increased. However, while there are still many questions to be answered about niacin metabolism during pregnancy, the niacin requirement is considered to increase during gestation. The 2006 RDI is 18 mg/day. There is no direct evidence to suggest a change in requirements in pregnancy, but an additional 3 mg/day would be needed to cover increased energy utilisation and growth.

The 2006 RDI for vitamin B\(_{12}\) during pregnancy is 2.6 µg per day, which is 0.2 µg per day higher than the recommendation for adult females. The extra vitamin B\(_{12}\) is required for the foetus, and an increased metabolic demand in the mother. Foetal storage of vitamin B\(_{12}\) increases during pregnancy and reaches a plateau at 38 weeks of gestation. An intake of 2.6 µg B\(_{12}\) per day is easily achieved as long as the diet contains some animal products. Pregnant women who consume a vegan diet must supplement their diet with vitamin B\(_{12}\).
**Folate**

Biochemical and haematological signs of folate deficiency (macrocytic anaemia) are common during pregnancy partly due to increased cell turnover and partly due to interference with normal folate metabolism by the high levels of oestrogen and progesterone. It is likely that many changes in indicators of folate status in pregnancy are hormone related and not clinically significant. Expansion of plasma and red cell volume may also contribute to changes in blood folate levels. An abrupt increase in serum folate is seen postpartum without any folic acid therapy, suggesting that low serum folate in pregnancy is secondary to shunting folate to the placenta. Both serum and red-cell folate are higher in cord blood than in maternal blood reflecting active transport across the placenta. Pregnant women with folate deficiency and even megaloblastic anaemia have infants with normal Hb and higher serum and red-cell folate levels than adults. Because of the crucial role that folate plays in cell division in DNA and RNA synthesis, and due to the paucity of information on the availability of folate from foods, the 2006 NRVs for Australians have set and EAR of 520 µg and an RDI of 600 µg dietary folate equivalents per day to cover the increased dietary requirement for folate, however, they state that ‘this recommendation does not include consideration of additional needs to prevent neural tube defects (NTD) as the neural tube is formed before most women know they are pregnant’. The Draft Dietary Guidelines recommend a woman planning pregnancy and during the first three months of pregnancy requires a daily 400ug (0.4mg) folic acid supplement in addition to foods naturally rich in folate.

**Vitamin B₆**

Vitamin B₆ status in pregnancy, as judged by a number of biochemical measures, in particular tryptophan metabolism, appears to be low. However, supplementation with B₆ results in only small increases in cord blood despite marked increases in maternal pyridoxal-5-phosphate (PLP) levels, suggesting that the observed changes are a consequence of foetal uptake rather than a functional B₆ deficiency. Low vitamin B₆ status in pregnancy is not generally associated with adverse birth outcomes. The extra allowance for B₆ in pregnancy is from 1.3 mg/day to 1.9 mg/day (2006 RDI).

**Minerals and trace elements**

**Iron**

During the second and third trimesters of pregnancy the 1991 NHMRC RDIs were 22.0–36.0 mg. The 2006 RDI recommend 27 mg/day during pregnancy. This is an amount which cannot generally be provided by diet alone, and supplements would be required to reach this level of intake. The rationale for the RDI is that this level is appropriate for the third trimester to build iron stores during the first trimester of pregnancy. The EAR of 22 mg/day was set by modelling the components of iron requirements for absorbed iron for the 50th percentile and the RDI by modelling the 97.5 percentile, and using an upper limit of 25% iron absorption and rounding. Absorption is higher from a mixed western diet including animal foods (about 18%) than a vegetarian diet (about 10%), so vegetarians will need intakes about 1.8 times higher.

Why have the Australian iron requirements in pregnancy been set at a level that appears to require iron supplementation? Firstly, this has been done because anaemia during pregnancy may compromise delivery of oxygen to the foetus and
because an anaemic woman is less well able to tolerate obstetric complications during childbirth. Secondly, the net additional amount of iron required during pregnancy (Table 8.3) is greater than the iron stores of many women, which seldom exceed 500 mg and are less than 100 mg in a considerable proportion. The extra iron needed for a normal pregnancy therefore has to be provided mainly from external sources.

Table 8.3 Iron requirements in pregnancy

<table>
<thead>
<tr>
<th>Component</th>
<th>Amount of iron (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>IRON REQUIREMENTS DURING PREGNANCY</td>
<td>300</td>
</tr>
<tr>
<td>Foetus Placenta</td>
<td>50</td>
</tr>
<tr>
<td>Expansion of maternal red cell mass</td>
<td>440</td>
</tr>
<tr>
<td>Basal iron losses</td>
<td>240</td>
</tr>
<tr>
<td>Total iron requirement</td>
<td>1040</td>
</tr>
<tr>
<td>NET IRON BALANCE AFTER DELIVERY</td>
<td></td>
</tr>
<tr>
<td>Contraction of maternal erythrocyte mass</td>
<td>+450</td>
</tr>
<tr>
<td>Maternal blood loss</td>
<td>-250</td>
</tr>
<tr>
<td>Net iron balance</td>
<td>+200</td>
</tr>
<tr>
<td>Net iron requirements for pregnancy if sufficient maternal iron stores are present (1040 – 200 = 840)</td>
<td>840</td>
</tr>
</tbody>
</table>

(FAO/WHO 2002)

Thirdly, the NHMRC committee assumed a maximum absorption of dietary iron of 25%. Under this assumption a woman entering pregnancy with minimal iron stores would need to consume an additional 10 mg of dietary iron daily throughout pregnancy. This is essentially impossible without an increase in energy intake much greater than that required to meet the additional energy cost of pregnancy, given an iron concentration in the average Australian diet of only 1.0 to 1.5 mg per 1000 kJ.

However, data exists that indicates that an intake of 27 mg per day may not be required to maintain iron stores during pregnancy. When iron absorption is measured throughout pregnancy using stable isotopes, the increase in the absorption of iron from food is shown to be a physiological consequence of normal pregnancy and not simply the result of developing anaemia during pregnancy (Barrett et al. 1994). Moreover the increase is large enough to meet the increased requirements of pregnancy provided that dietary intake is of the order of 12–16 mg per day. Iron absorption increases from an average of 7% at 12 weeks to 36% at 24 weeks and 66% at 36 weeks of gestation in subjects with normal iron status at the outset of pregnancy. In addition, the mother saves a total of 120 mg of iron due to an absence of menstruation.

An increase in iron absorption of this magnitude means that while, on average, only about 1 mg of an intake of 15 mg of dietary iron is absorbed in the first trimester, towards the end of the last trimester of pregnancy as much as 10 mg may be absorbed daily; and in consequence a normal dietary intake of 12–16 mg of iron...
is likely to be sufficient to meet the iron requirements of pregnancy in women with normal iron status, even without significant stores of iron at the outset of pregnancy.

Calcium
There 2006 NRVs do not make any additional allowance for pregnancy. This is in contrast to the previous 1991 RDI for calcium during pregnancy which was an extra 300 mg per day. The 2006 EAR and RDI for pregnancy were based on the needs of the mother plus any additional allowance for the foetus and products of conception. The foetus retains about 25–30 g mostly in the third trimester of pregnancy but there is evidence that pregnancy is associated with increased calcium absorption. Significant increases in maternal calcium accretion, bone turnover and intestinal absorption early in pregnancy prior to foetal bone mineralisation have also been shown. These adaptations provide the minerals necessary for foetal growth without requiring an increase in maternal dietary intake or compromising long term maternal bone health. Dietary calcium intake does not appear to influence changes in maternal bone mass in pregnancy and there is no relationship between the number of previous pregnancies and bone mineral density or fracture risk; however, targeted supplementation has been recommended as a precautionary measure for women with very low intakes of calcium or who are at risk of vitamin D deficiency (Prentice 2003). Indeed some studies show a positive correlation between number of children born and radial bone mineral density of total body calcium as well as reduction in hip fracture risk. These findings support the concept that the maternal skeleton is not used for foetal calcium needs. The available information does not support the need for additional dietary intake in pregnancy as maternal adaptive mechanisms including enhanced efficiency of absorption more than account for the additional needs in the last trimester. This, of course, assumes that the usual maternal intake dietary calcium intake is sufficient for maximising bone. Human placental chorionic somatomamnotropin increases the rate of bone turnover progressively through pregnancy. Oestrogen (also largely from the placenta) inhibits bone resorption and provides a compensatory release of parathyroid hormone which maintains the serum calcium concentration while enhancing intestinal calcium absorption and apparently decreasing its urinary excretion.

Zinc
The 2006 NRVs for pregnant women increase the RDI from 8 mg/day (adult female) to 11 mg/day. The EAR was established by estimating the additional needs for the additional maternal and foetal tissues and adding this to the equivalent non-pregnant EAR. The figure used was based on late pregnancy estimates of zinc accumulation (the period of greatest need) to give a single recommendation throughout pregnancy. Zinc accumulation at this time averages 0.73 mg/day. Absorption in pregnancy is thought to be similar to that of non-pregnant women so an absorption rate of 31% was used to estimate the additional requirement of 2.35 mg/day. Absorption is higher from animal foods than plants sources, so vegetarians, particularly strict vegetarians, will need intakes about 50% higher. NB: For women taking high levels of iron supplements during pregnancy and lactation, the proposed EAR and thus RDI may not be adequate. There is some evidence that high levels of iron supplements prescribed to pregnant and lactating women may decrease zinc absorption.
Iodine
Iodine requirements are higher during pregnancy than in the non-pregnant state because the production of thyroid hormones (which requires iodine) is increased in pregnancy by 50%. The developing foetus is at greatest risk of iodine deficiency. Mild to moderate iodine deficiency can cause learning difficulties and affect physical development and hearing. The EAR and RDI for iodine during pregnancy are 160µg and 220 µg/day respectively. The National Health and Medical Research Council (NHMRC) recommends that all women who are pregnant, breastfeeding or considering pregnancy, take an iodine supplement of 150 micrograms (µg) each day to meet these requirements.

Magnesium
For magnesium, the NRVs recommend an extra 80 mg per day during pregnancy based on an estimated requirement of 18 mg per day for foetal metabolism and growth.

Electrolytes
During pregnancy, hormonal changes result in a markedly increased glomerular filtration rate in order to maintain homeostasis in the increased maternal blood volume. This results in an additional filtered electrolyte load. Compensatory mechanisms, however, continue to maintain fluid and electrolyte balance and there is no additional requirement for dietary sodium or potassium during pregnancy.

**Nutrition in adolescent pregnancy**

Pregnancy during adolescence (especially if the young woman is less than 15 years of age), is associated with a number of medical complications including abnormal weight gain, elevated blood pressure and higher risks of iron deficiency anaemia. Low birth weights are more common and birth weights are around 150–200 g lighter in younger mothers, despite young women gaining more weight than their older counterparts. This is indicative of competing maternal and foetal demands for nutrients. Deliveries before 37 weeks are significantly more common in adolescent women (Lenders et al. 2000). Current evidence indicates that there is little change in dietary behaviours between pregnant and non-pregnant adolescents. Therefore the low dietary intakes of calcium, iron, zinc and folate and the overconsumption of salt, saturated fat and added sugar observed in the 2007 Children’s Nutrition and Physical Activity Survey (CNPAS) would indicate less than ideal dietary practices for young pregnant women.

**Food requirements during pregnancy**

Table 8.4 summarises the recommended dietary patterns for pregnant women aged 19–50 years and pregnant adolescents of average height and with sedentary to moderate activity levels. These recommended food patterns do not meet the EAR for iron in pregnancy for either age group (14–18 years: 23 mg/day; 19–50 years: 22 mg/day) (Dietitians Association of Australia 2011, NHMRC 2006).
### Recommended dietary patterns for pregnant women aged 19-50 years and pregnant adolescents of average height and with sedentary to moderate activity levels

<table>
<thead>
<tr>
<th>Food group</th>
<th>Example serving sizes</th>
<th>Recommended number of serves per day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vegetables and legumes / beans</td>
<td>1/2 cup cooked vegetables (75 g)</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>1 cup salad vegetables</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1 small potato</td>
<td></td>
</tr>
<tr>
<td></td>
<td>½ cup cooked dried or canned beans, chickpeas or lentils, no added salt</td>
<td>5</td>
</tr>
<tr>
<td>Fruits</td>
<td>1 medium piece of fresh fruit (150 g)</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>30 g dried fruit (e.g. 4 dried apricot halves)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1 cup canned fruit (150 g)</td>
<td>2</td>
</tr>
<tr>
<td>Grain (cereal) foods</td>
<td>1 slice of bread or ½ a medium roll or flat bread (about 40 g)</td>
<td>8 ½</td>
</tr>
<tr>
<td></td>
<td>½ cup cooked rice, pasta, noodles</td>
<td></td>
</tr>
<tr>
<td></td>
<td>½ cup cooked porridge or polenta, 2/3 cup breakfast cereal flakes (30 g) or ½ cup muesli</td>
<td>8</td>
</tr>
<tr>
<td>Lean meat and poultry, fish, eggs, nuts and seeds, and legumes / beans</td>
<td>65 g cooked lean red meats or ½ cup of lean mince, 2 small chops</td>
<td>3 ½</td>
</tr>
<tr>
<td></td>
<td>80 g cooked poultry</td>
<td></td>
</tr>
<tr>
<td></td>
<td>100 g cooked fish fillet or 1 small can of fish</td>
<td>3 ½</td>
</tr>
<tr>
<td></td>
<td>2 large eggs</td>
<td></td>
</tr>
<tr>
<td></td>
<td>170 g tofu</td>
<td></td>
</tr>
<tr>
<td></td>
<td>30 g nuts or seeds</td>
<td></td>
</tr>
<tr>
<td>Milk, yoghurt, cheese and/or alternatives</td>
<td>250 mL cup of milk</td>
<td>2 ½</td>
</tr>
<tr>
<td></td>
<td>200 g tub of yoghurt</td>
<td></td>
</tr>
<tr>
<td></td>
<td>40 g or 2 slices of cheese</td>
<td>3 ½</td>
</tr>
<tr>
<td></td>
<td>120 g ricotta cheese</td>
<td></td>
</tr>
<tr>
<td>Additional serves from the five food groups or discretionary choices for those who are taller or more active</td>
<td>0–2 ½</td>
<td>0–3</td>
</tr>
</tbody>
</table>

(DNHMRC 2011)

### Diet and pregnancy outcome

On an individual basis it is difficult to relate dietary intake to the outcome of pregnancy, but at the population level associations between diet and pregnancy outcome have been observed under the abnormal, and often extreme, conditions of war and natural disasters.

For example, observations in Europe during WWI and WWII indicate that when dietary restriction was both severe and prolonged, as it was during the siege of Leningrad, not only did foetal mortality and prematurity increase but fertility was also reduced (Antonov 1947). When the period of dietary restriction was severe but relatively short (six months), the average birth weight fell by about 200 g and was
lowest in infants whose mothers were exposed to dietary restriction throughout the second half of pregnancy. However, in mothers exposed to famine conditions only during the first half of pregnancy, the birth weight of the infants was little affected (Smith 1947).

The effects of severe food restriction on stillbirths and congenital malformations followed a different pattern. The rates were lowest for infants conceived before the famine and highest for those conceived during it. These findings are in line with what might be expected from knowledge of the timing of different aspects of foetal growth and development.

In prolonged and severe undernutrition, infertility appears to provide a protective mechanism against further maternal physiological stress. However, if conception occurs and is followed by acute dietary restriction, the effects on foetal growth and development depend on the timing of the restriction.

The effects of lesser degrees of food restriction during pregnancy, such as might be found in more affluent societies are difficult to measure. Godfrey and co-workers (1997) have looked at the relative effect of diet and maternal birth weight on the infant’s birth weight. While in early pregnancy they found a small inverse association between energy intake and infant birth weight, they found no association in later pregnancy. These results suggest that in well-nourished societies the effect of differences in maternal energy intake during pregnancy is small relative to the influence of maternal physiological factors.

Kramer and Kakuma (2003) have reviewed the evidence from controlled clinical trials of energy and protein intake during pregnancy on the outcome of gestational weight gain and the outcomes of pregnancy. Trials of nutritional advice have been successful in increasing protein and energy intake but on balance this did not result in beneficial pregnancy outcomes. The use of balanced energy and protein supplements showed only modest gains in maternal weight gain and foetal growth even in undernourished women, whereas high protein supplements inferred no benefit and may actually be harmful. Energy and protein restriction, during pregnancy, in overweight women showed little benefit and could be harmful to the foetus.

Kramer and Victoria (2001) suggest blanket administration of balanced energy and protein supplementation to pregnant women in populations where there is a high prevalence of maternal undernutrition in order to prevent impairment of foetal growth.
Factors influencing maternal nutrition

Figure 8.5 Influences on and outcomes of maternal nutritional status

Figure 8.5 shows the many influences on maternal nutritional status during pregnancy and how these factors interrelate to affect the outcome for both mother and child. Socioeconomic factors and biological factors probably play equally important roles in determining pregnancy outcome. As the figure shows, pregnancy outcome for the child is judged by birth weight and subsequent health and development. For the mother, pregnancy can have both short-term and long-term effects. In the short term, her lactational performance and fertility are affected; and in the longer term her nutritional status during pregnancy can influence her morbidity, social interactions and life expectancy.

Ultimately, the socioeconomic and biological influences on pregnancy are mediated largely through maternal food intake and nutritional status. Because of the importance of maternal nutritional status to pregnancy outcome, an understanding of nutritional requirements during pregnancy is essential. However, it is also important to appreciate that our knowledge of nutritional requirements during pregnancy is far from complete.
Common nutritional concerns

Anaemia

The primary purpose of maintaining an adequate maternal iron status during pregnancy is to prevent problems which can arise as a consequence of anaemia in pregnancy. While the current Australian RDI for iron during the second and third trimester of pregnancy recommends a level of iron intake which can only be achieved by supplementation of the diet with iron, this does not mean that iron supplementation is necessary in all pregnant women. Iron deficiency anaemia in pregnancy is much more common in aboriginal women than in non-aboriginal women (Markey et al. 1998). Routine supplementation is not necessary in individuals in whom haematological status can be monitored and is found to be adequate. Walker (1998) has reviewed the evidence for the effectiveness of iron supplementation in pregnant women and concludes that under most circumstances, particularly in the developed world, iron supplementation during pregnancy appears to be unwarranted and that other approaches to increasing iron intake may be more appropriate.

Guidelines for assessing and evaluating iron status in pregnancy are shown in Figure 8.6.

Listeria

Listeria infection can be caused by eating foods contaminated with the bacteria called Listeria monocytogenes. Pregnant women are at risk of Listeria infection, which can cause miscarriage, still birth, premature birth or a very ill infant at birth. Eight to 24 cases of Listeria infection are reported to the Victorian Government Department of Human Services each year. However, not all of these cases involve pregnant women. High risk foods for Listeria infection include:

- smoked fish, smoked mussels, oysters and raw seafood
- prepared salad, including coleslaw
- pre-cooked meat products eaten without further cooking, e.g. pate, sliced deli meat
- unpasteurised milk
- soft serve ice-cream
- soft cheeses, e.g. brie, camembert, ricotta.

It is recommended that pregnant women avoid these high-risk foods and ensure safe handling practices when preparing food.
Figure 8.6 Recommended guidelines for the assessment and evaluation of iron status in pregnancy

Iron and Pregnancy

RECOMMENDED GUIDELINES

FIRST VISIT

**History**
- High risk factors
  - Blood donation - current or recent history
  - Previous iron deficiency

Other risk factors
- Poor socio-economic status, recent immigrant
- Vegetarian diet

Past history
- Post-partum haemorrhage
- Multiparity
- Short gap between pregnancies
- Heavy periods

Routine Hb
- MCV, Hb, Hct

**Assessment**

1. Anemia
   - Criteria:
     - Hb < 10.5 gm%
   - Action:
     - Management as per established protocols for investigation of anemia in pregnancy (including serum ferritin assay).

2. High risk of developing ID
   - Criteria:
     - Hb < 10.5-11.5 gm% and
     - Presence of one major or
     - Two or more of any risk factors.
   - Action:
     - Specific dietary advice (see below)
     - Iron supplementation: at least 30 mg/day of elemental iron
     - Reassess at 28 weeks.

3. Possible risk of iron deficiency
   - Criteria:
     - Hb < 11.5 gm% or
     - Of the practice of at least one of the above risk factors.
   - Action:
     - Specific dietary advice: (see below)
     - Iron supplementation is not required.
     - Reassess at 28 weeks.

4. Low risk of iron deficiency
   - Criteria:
     - Hb > 11.5 gm% and no risk factors present.
   - Action:
     - General preventative dietary advice
     - Iron supplementation is not required.

**Subsequent Early Antenatal Care**

Assessment:
- Diet
- Presence of nausea, vomiting
- Compliance of those women prescribed iron supplementation

28 WEEK VISIT

Assessment: Serum ferritin for all patients in groups 1 (genetic) or 3 (possible risk of ID), as well as a repeat Hb.

Action: If serum ferritin < 16 pg/mL, iron supplementation is advisable, elemental iron.

**Specific Dietary Advice**
- Include foods rich in absorbable iron (red meat, chicken, pork, fish).
- Maximize the absorption of iron (vegetables and cereals at the main iron-containing meal):
  - Avoid consuming absorption inhibitors (tea, coffee, phytate)
  - Use absorption promoters (meat, vitamin C-rich foods).

**Iron Supplementation**
- Appropriate dose is at least 30 mg elemental iron/day or 40 mg/day in sustained release form, such as combination iron/folate preparation.
- Greater doses may be necessary in cases of established ID. Avoid taking with tea or coffee.
- Be careful to prevent accidental iron poisoning in children.

(Australian Iron Status Advisory Panel 1997)
QUESTIONS

1 What is the basis for theoretical estimates of the additional energy needed during pregnancy?

2 What are the theoretical energy requirements during pregnancy? How much additional energy do women actually consume during a typical pregnancy and what amount of food does this equate to? What can explain this difference?

3 At the end of the second trimester of pregnancy the foetus is still quite small but maternal weight has increased by about 8 kg on average. What are the major components of weight gain up to this time?

4 How is the optimum weight gain during pregnancy influenced by weight-for-height status (BMI) prior to pregnancy?

5 What is the role of vitamin and mineral supplements during pregnancy and why?

6 Comment on the relative importance, in relation to the outcome of pregnancy, of:
   (a) nutritional status before pregnancy and
   (b) nutrient intake during pregnancy.

7 What are the major maternal physiological adaptations which occur during pregnancy, and how do they influence:
   (a) measures of maternal nutritional status?
   (b) maternal nutritional requirements?

8 The concentration of some nutrients in the maternal blood falls during pregnancy. How should this fall be interpreted?

9 The RDI for iron during pregnancy is 27 mg/day. Are pregnant women likely to consume this much iron on a daily basis? If a woman does not consume this much iron through her diet is she likely to become anaemic? What is the role of oral iron supplements during pregnancy?

10 Where would a foetus derive calcium from if the mother consumed minimal amounts of dietary calcium during her pregnancy?

11 Draw up a comparison table of RDIs for folate, B_{6}, B_{12}, iodine, and vitamin C for adult women and pregnant women. What factors are responsible for changes in recommended amounts and what nutrients would be challenging to achieve from diet alone?

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TOPIC 9

The role of maternal nutrition in lactation

PREPARED BY THE UNIT TEAM

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**Introduction**

A woman’s breasts are prepared for lactation, by hormones secreted during pregnancy, irrespective of whether she decides to breastfeed her infant or not.

The process of breastfeeding is successfully initiated by almost all women who attempt to do so. When it fails the causes of failure are more often psycho-social or related to management of the breastfeeding process rather than to inadequate maternal nutrition.

In the section of infancy we discussed the benefits of breast milk for the infant, whereas we will discuss the physiology of lactation, benefits and nutritional requirements from the point of view of the mother.

**Learning objectives**

The aim of this section is to give you an understanding of the nutritional requirements for successful lactation. At the end of this section you should be familiar with, and have an understanding of the following aspects of human lactation:

- the physiological process of lactation
- the nutrient composition of human milk at different stages of lactation
- the nutritional requirements during lactation
- the effect of maternal diet on milk composition and volume
- factors influencing breastfeeding initiation and cessation
- the advantages and contraindications of breastfeeding.

**Learning resources**

**Print reading**

The reading listed here is reproduced in the print readings for this unit.


**Deakin Studies Online (DSO)**

Access the unit site on DSO <http://www.deakin.edu.au/current-students/> for the appendices and other additional resources you can use to supplement this topic.
Physiology of lactation

The requirements for successful lactation are that the infant can suck with sufficient strength, that the breast structure allows sucking to occur, that the sucking process stimulates release of pituitary hormones in the mother to maintain lactation, and that emotional responses in the mother do not inhibit this process.

Development of the mammary glands

The development of the mammary glands begins early in foetal life but is not complete until after the birth of the female’s first offspring. The precise hormonal regulation of this process is not fully understood but oestrogen, progesterone and human placental lactogen are all involved.

At birth the mammary glands are sufficiently developed to appear as distinct organs with a discernible lobular structure connected by ducts that are capable of producing a milk-like secretion. After birth there is some involution of the mammary glands and no further development occurs until puberty.

With the onset of puberty and the accompanying maturation of the ovaries there is an increased output of oestrogen. This stimulates both elongation of the mammary ducts and proliferation of the ends of the mammary tubules. At the same time there is also an increase in fibrous and fatty tissue between the milk ducts.

As ovulation becomes established the regular production of progesterone promotes the development of the lobules and of the glandular secreting cells. The differentiation into a lobular gland is complete about one to two years after the onset of menstruation but some further development of the secretory structures continues, under the influence of oestrogen and progesterone, during each menstrual cycle but in particular during pregnancy. During pregnancy there is some increase in size and weight such that by term the breast may weigh between 400 and 600 g and during lactation this weight increases to between 600 and 800 g.

As illustrated in Figure 9.1, the human breast is an exocrine gland composed of milk-secreting alveoli that drain into the lactiferous sinuses through milk ducts. From the sinuses the milk flows to the nipple. These milk-secreting structures are supported by fat and connective tissue and endowed with rich nerve and blood supplies.

Until pregnancy occurs the breast is composed largely of fat and connective tissue, and the milk-secreting structures are relatively undeveloped. During pregnancy, important hormonal changes occur. Under the influence of many hormones, including oestrogen, progesterone, corticosteroids and hypophyseal and placental lactogens, the ducts and alveoli develop in the breast tissue. Nipple length and protractility, which facilitate the infant drawing the nipple into its mouth, also increase. The milk-producing system is fully developed for lactation late in pregnancy. Initiation and maintenance of lactation constitute a complex process involving both nerves and hormones. Lactation involves the sensory nerves in the nipples, the spinal cord, the hypothalamus and the pituitary gland with its various hormones.
The process of lactation occurs in four stages:

- initiation and maintenance of milk secretion
- milk production
- milk ejection
- ingestion of milk.

**Structure of the mammary gland**

(Worthington-Roberts & Williams 1989, p. 247)
Initiation and maintenance of lactation

Initiation of milk secretion, or lactogenesis, begins in the last trimester of pregnancy and results in small amounts of colostrum being produced. This occurs as a result of stimulation of the breast alveolar cells by placental lactogen (a prolactin-like substance). Full lactation, however, does not occur immediately and over the first two to three days after birth only a small amount of colostrum is secreted. In subsequent days, a rapid increase in milk secretion occurs, and in usual cases lactation is reasonably well established by the end of the first week. In first-time mothers, however, the establishment of lactation may take up to three weeks or even longer.

As soon as the placenta is delivered at birth, the concentrations of progesterone and oestrogen fall, which until then exert an inhibitory effect on prolactin, the key hormone in initiation and maintenance of milk production (Figure 9.2).

![Hormone levels during pregnancy and lactation](Riordan 2005, p. 76)

Maintenance of milk secretion requires the continued sucking of the infant, which stimulates the production of prolactin. The newborn infant’s rooting and sucking reflexes are strongest 20–30 minutes after birth. By putting the baby to the breast soon after delivery lactogenesis is greatly facilitated.
**Milk production**

Prolactin exerts its effect on the milk-producing alveoli by enhancing specific enzymes associated with the synthesis of fat, protein and lactose.

Each milk-producing alveolar cell proceeds through a secretory process, which is preceded and followed by a resting stage. The process of milk synthesis is most active (more cells are in a secretory phase) while the infant is suckling but continues at a lower level at other times.

Several secretory processes are involved. These include exocytosis, fat synthesis and secretion, secretion of ions and water and immunoglobulin transfer from the extracellular space. Proteins, lactose and some ions are secreted by exocytosis. Most of the proteins present in human milk are specific to human milk and not found elsewhere. Inclusion of proteins derived from maternal plasma occurs mainly in colostrum.

The three main milk proteins: casein, \( \alpha \)-lactalbumin and \( \beta \)-lactalbumin are all synthesised within the mammary gland from amino acids. Lactose, which is the predominant carbohydrate in milk, is synthesised from glucose and galactose.

Short-chain fatty acids are synthesised by the alveolar cells predominantly from acetate while long-chain fatty acids and triglycerides are derived from maternal plasma. Some triglyceride is also synthesised from intracellular carbohydrate. The triglycerides, synthesised in the alveolar cells, coalesce into large droplets which then make their way to the top of the alveolar cell and are enveloped by the apical membrane before separating from the cell.

Sodium, potassium and water move freely across the apical membrane. Water moves across the membrane in response to the osmotic gradient set up by the
secretion of lactose and the electrolytes follow in response to the movement of water across the cell membrane.

Immunoglobulin A and possibly other proteins attach to receptors on the basal membrane of the cell from where they enter the cell by endocytosis and are transported to the apical membrane for subsequent release into milk.

Under normal circumstances substances do not pass directly from the maternal capillaries into the milk. During mastitis and involution of the glands, however, the junctions between the alveolar cells become ‘leaky’ and allow plasma constituents to pass directly into milk. When this happens the milk tends to be high in sodium and chloride and lower in lactose and potassium.

Since prolactin is produced in response to nipple stimulation (Figure 9.3), the importance of ‘demand feeding’ rather than 3-hourly to 4-hourly scheduled feeding is clear. A baby sucks most vigorously when it is hungry. Therefore a baby fed on demand will provide optimal nipple stimulation. Demand feeding results in more frequent, intense nipple stimulation and an increased prolactin production which assures an adequate milk supply.

An additional advantage of demand feeding throughout the day and night is that it stimulates prolactin secretion, which acts as a natural contraceptive. Whether it is the prolactin itself that inhibits ovulation, or as some other substance for which prolactin is a ‘marker’, is not known. The custom in most Western countries of encouraging babies to sleep through the night, without breastfeeding, as soon as possible after birth, results in an early return of fertility and the need for contraceptives. In societies where the infant is frequently fed both day and night, lactational infertility is prolonged. This would appear to be a protective measure for both mother and child, by delaying the arrival of another infant.

**Milk ejection**

Movement of the milk from the alveoli to the lactiferous sinuses is an active process within the breast which occurs in response to a neurohormonal mechanism referred to as the ‘let-down’ reflex. Nipple stimulation by the infant (neuro) results in the secretion of oxytocin by the posterior pituitary (hormonal). The principal action of this hormone is to cause the alveoli to eject or ‘let down’ the milk they contain.

In a relaxed woman the ‘let-down’ of milk occurs in response to the infant’s cry or simply thoughts about feeding the infant. Negative emotions such as anxiety and uncertainty, or just lack of interest can, however, inhibit the reflex. A poor ‘let-down’ reflex sets up the cycle whereby the hungry baby sucks harder, causing pain and cracked nipples, which may become infected. Nipple infection leads to even greater discomfort for the mother, and this further reduces the likelihood of satisfactory milk ejection, and ultimately mastitis or a breast abscess may occur and lactation fails completely.

Early failure of lactation is accelerated by complementary bottle feeds not only because demand is reduced but also because the baby tends to suck less strongly as bottle teats require much less active sucking.
Milk ingestion

Milk ingestion requires both an adequate let down reflex and functional rooting, suckling and swallowing reflexes in the baby. All four stages of lactation are dependent upon the health and nutritional status of both mother and child.

Factors affecting breast milk composition and volume

Table 9.1 illustrates how difficult it is to state ‘absolute’ figures for breast milk composition because of the variation that normally occurs both within and between women.

Stage of lactation

Milk composition varies considerably with the stage of lactation. The most dramatic change occurs during the first two weeks of lactation, in the transition from colostrum to mature milk.

Colostrum is very high in protein (4–5%) but contains less fat and lactose than mature milk. It is slightly lower in energy than mature milk ~240 KJ v 280 KJ/100 mL. Some of the extra protein comes from the anti-infective agents that are particularly high in colostrum, such as the immunoglobulins. Colostrum is lower in most of the B vitamins than mature milk, but higher in the fat-soluble vitamins A, D, E and minerals.

Once the transition from colostrum to mature milk has occurred (usually within the first month of lactation), changes in composition with time are less dramatic. Both riboflavin and folic acid have been reported to decrease during lactation, and zinc decreases quite markedly during the first six months of breastfeeding.

By about six months the amount of several of the nutrients in breast milk are limiting, in particular energy, iron and zinc, and there is a need for the addition of other foods to the diet.

Stage of a feed

It is well known that milk composition varies significantly within each feed. The fat content increases up to fourfold from the beginning to the end of a feed. Some of the trace elements also vary in concentration within a feed and vitamin A increases with the increase in fat content.

Time of day

At the early-morning feed, the volume of milk produced is usually greater than during the remainder of the day and its nutrient concentration, especially fat, is lowest. The fat concentration increases during the day, as does the vitamin C content.

Individual variation

Breast milk composition varies between women: no two women produce milk of exactly the same composition. It also varies from day to day, week to week, and month to month, and even varies between the left and right breast.
TABLE 9.1 Variation in human milk composition between women, with stage of lactation and with nutritional status

<table>
<thead>
<tr>
<th>Nutrient content per litre</th>
<th>Between women</th>
<th>With lactation</th>
<th>With nutritional status</th>
</tr>
</thead>
<tbody>
<tr>
<td>Energy (kcal)</td>
<td>630–790</td>
<td>650–700</td>
<td>550–770</td>
</tr>
<tr>
<td>Protein (g)</td>
<td>10–14</td>
<td>16–12</td>
<td>9–13</td>
</tr>
<tr>
<td>Fat (g)</td>
<td>35–46</td>
<td>35–40</td>
<td>24–48</td>
</tr>
<tr>
<td>Lactose (g)</td>
<td>64–76</td>
<td>66–70</td>
<td>65–77</td>
</tr>
<tr>
<td>Calcium (mg)</td>
<td>250–410</td>
<td>400–310</td>
<td>210–340</td>
</tr>
<tr>
<td>Iron (mg)</td>
<td>0.5–1.6</td>
<td>0.4–0.8</td>
<td>–</td>
</tr>
<tr>
<td>Zinc (mg)</td>
<td>1.2–3.9</td>
<td>3.8–2.2</td>
<td>–</td>
</tr>
<tr>
<td>Vitamin A (µg)</td>
<td>400–800</td>
<td>880–540</td>
<td>360–650</td>
</tr>
<tr>
<td>Thiamin (mg)</td>
<td>0.1–0.2</td>
<td>0.2–0.15</td>
<td>–</td>
</tr>
<tr>
<td>Riboflavin (mg)</td>
<td>0.3–0.4</td>
<td>0.4</td>
<td>–</td>
</tr>
<tr>
<td>Vitamin B₁₂ (µg)</td>
<td>0.3–1.0</td>
<td>0.4–0.5</td>
<td>–</td>
</tr>
<tr>
<td>Vitamin C (mg)</td>
<td>5–55</td>
<td>55–44</td>
<td>–</td>
</tr>
</tbody>
</table>

*The first value in each case is for transitional milk.


Milk composition

Energy

The energy in milk is provided mainly from fat and carbohydrate (lactose). In well-nourished women, dietary effects on the energy supplied by breast milk are minimal. The most significant influence on the energy content of breast milk is the increase in milk fat that occurs towards the end of a feed. In malnourished women the energy content of the milk does not alter markedly unless severe malnutrition occurs, when the milk fat content is reduced. The total amount of energy supplied by the milk may, however, be lower if the volume is decreased.

Protein

The amount of protein, predominantly casein and whey, in breast milk also does not seem to be much influenced by the maternal diet, even in malnourished women. The concentration of protein in breast milk is 1.27g/100 mL. There appears, however, to be some effect of maternal diet on the synthesis of specific milk proteins. The amino acid profile adequately meets the needs of the infant. Breast milk contains the amino acid taurine, which is associated with bile acid production. Taurine may also have a role as a neurotransmitter or neuromodulator in the brain and retina. Cow’s milk contains little taurine when compared to breast milk.

Lactose

The lactose content of breast milk is largely independent of dietary factors.
Fat
The majority of fat is present in the form of triglycerides, with smaller amounts of phospholipids, cholesterol, diglycerides, glycolipids, sterol esters and free fatty acids. Breast milk is also known to contain the essential fatty acids linoleic and alpha-linolenic acid, and eicosapentanoic acid (EPA) and docoshexanoic acid (DHA). The amount of fat in human milk is not affected by the maternal diet, except in cases of severe undernutrition. However, the type of fat found in breast milk varies with the mother’s diet and it is possible to increase or decrease the polyunsaturated fatty acid content of breast milk by changing the mother’s diet. When the mother’s energy intake is adequate (i.e. she is maintaining her body weight), the fatty-acid pattern of milk resembles that of her diet, whereas if the mother is losing weight, the milk fatty acid pattern resembles that of her adipose tissue. Fat digestion is aided by the presence of lipases in breast milk.

Vitamins
The concentration of water-soluble vitamins usually reflects the dietary intake of the mother. Whereas the effect of day-to-day fluctuations in dietary intake may not be apparent, the long-term effects of low or high intakes (or vitamin supplements) can be clearly observed. For example, in areas of the world where fresh fruit and vegetable availability is seasonal, the vitamin C content of breast milk is also subject to seasonal variation.

In the short term, the effect of the maternal dietary intake of the fat-soluble vitamins A and D is both less pronounced and less variable than for the water-soluble vitamins.

Minerals
Breast milk is known to contain potassium, calcium, phosphorus, chlorine, sodium, iron, copper, manganese, zinc, magnesium, aluminium, iodine, chromium, selenium and fluoride. Much work remains to be done on the effect of maternal intake of minerals on breast milk composition. Studies have demonstrated that the iron, copper, fluoride and zinc content of breast milk are not altered by the administration of these elements to lactating women. In contrast to this, there is some evidence to show that the level of zinc in mature milk is higher in women with a higher zinc intake during the last trimester of pregnancy (Ortega et al. 1997) and that the fractional absorption of zinc is significantly increased during lactation (25%) as compared with 14% before pregnancy (Fung et al. 1997).

The calcium content of human milk, although quite variable, does not respond to dietary fluctuations in calcium intake or to supplementation with calcium during lactation. Supplementation during lactation also appears to have no effect on the decrease in bone density, which occurs during lactation. Overall the evidence suggests that optimal lactation and maternal bone health do not depend on calcium intake by the mother and that breastfeeding women need not consume extra calcium (Prentice 1997). As Prentice points out, however, this does not imply that an adequate calcium intake is not important during lactation.
Milk volume

Larger volumes of milk are produced in infants that feed more frequently and have a stronger suck. A study of lactating women conducted in Cambridge (England) and in The Gambia found that the level of milk production is established very early in lactation (Rowland, Paul & Whitehead 1981). Initially, high milk outputs are found in mothers with bigger babies. However, these peak sooner, and then progressively fall off more rapidly than those of mothers with smaller babies. Possibly the higher early output exhausts the mother’s supply sooner. Milk volume is likely to be reduced in premature infants that may have weaker sucking.

It is known that increased intake of water or fluids has no physiological effect on the volume of milk produced, although it is important for lactating women to maintain an adequate fluid intake.

There is no evidence that maternal energy stores as assessed by body mass index (BMI) have a detectable relationship with the volume of milk produced both between and within populations. Inter-country analysis also fails to show any detectable association between BMI and the energy content of breast milk (Prentice, Goldberg & Prentice 1994).

Stress can reduce milk volume by interfering with the let down reflex. Alcohol consumption and cigarette smoking can also reduce milk volume and let down in a negative dose dependent manner.

Other components in breast milk

There are more than twenty enzymes present in breast milk including proteases, lipases and amylases. Bifidus factor has a protective effect against enteropathic organisms, while immunoglobulins confer resistance against bacteria and viruses that infect the gastrointestinal tract.

Exercise

Relatively little information is available on the effects of exercise on milk volume and composition. Dewey et al. (1994) conducted a randomised controlled trial of the effects of regular aerobic exercise involving the expenditure of around 1.7 MJ on five days per week for a period of 12 weeks. No significant differences were observed between the two groups of women in the volume or composition of the milk or in the weight gain of their infants. There were also no differences in the amount of body weight or fat loss, however, cardiovascular fitness was significantly improved in the exercise group.

Nutrient requirements during lactation

Maternal nutrient requirements during lactation are not easy to define and are at best estimates (see Appendix 2 on DSO). Nutrient needs are dependent on the composition and volume of milk produced.
Energy

Theoretically, lactation requires approximately an extra 3000 kJ/day. This figure is based on an average daily milk yield of 850 mL, with an energy content of 277–323 kJ (66–77 kcal) per 100 mL and an efficiency of conversion of maternal to milk energy of around 80–90%.

Energy requirements will vary as daily milk yield varies significantly between women and from day to day in the same woman. In poorly nourished mothers it has been estimated that milk output is around 500–700 mL per day in the first six months of lactation, 400–600 mL per day in the second six months and 300–500 mL per day in the second year. In contrast, some well-nourished and highly motivated mothers are able to produce 1 to 1.5 litres of milk per day. Such high yields are probably the exception even in Australia, but demonstrate the variability in milk production.

The amount of body fat laid down during pregnancy is another possible source of variation in the additional energy required for lactation. Most women store 2.0–4.0 kg of body fat which can be mobilised to provide energy for the first three months. A recent study of total energy metabolism during lactation in well-nourished women, however, has found that body fat utilisation contributed very little to the energy requirements of lactation (Goldberg 1994). In this study, on average, just over half of the additional energy cost of lactation was met by an increased intake of energy and the remainder by a reduction in physical activity relative to pre-pregnancy level, although these proportions varied considerably in individual women. Overall energy balance in this study demonstrated very good agreement between reported energy intake and total energy expenditure, including that lost as breast milk.

Many women can expect a gradual weight loss during lactation, even with an increased energy intake at this time, but there are some who do not lose the extra weight gained during pregnancy until after they stop breastfeeding.

Weight loss in overweight women

Retention of weight gained during pregnancy may contribute to the development of obesity. Lovelady et al. (2000) conducted a randomised, controlled trial to determine if weight loss in overweight women during lactation will affect the growth of infants. Forty overweight breastfeeding women, four weeks postpartum, participated in the 10-week study. The experimental group reduced their daily energy intake by 2050 kJ and exercised for 45 minutes per day for four days per week. For the control group, the diet was unchanged and their exercise was limited to one session per week. The diet and exercise group lost between 1.7–8.3 kg (mean 4.8 kg), whereas the control group had a gain of 4.6 kg to a loss of 4.6 kg (mean loss of 0.8 kg). There were no significant differences between the groups in the infants’ gain in weight or length. The authors have recommended that weight loss of 2 kg per month in the early postpartum period is unlikely to affect milk production adversely.
Protein
The concentration of protein in breast milk is 1.27 g/100 mL. Based on a milk yield of 850 mL per day, the actual protein loss (N × 6.25) from the maternal body to milk is only around 9 g per day. The recommended daily intake (RDI) allows both for a higher milk yield of 1000 mL per day and an NPU of 70% for dietary protein. The Australian dietary recommendation is 1.1 g/kg or 67 g/day.

Vitamins

Thiamin
The RDI for thiamin for lactating women is based on the amount of thiamin incorporated into the milk (about 0.16 mg thiamin per day) and an additional 0.1 mg/day is also needed to cover the metabolic need for thiamin incurred by the energy requirement for milk production. The total additional intake recommended during lactation is 0.26 mg per day. As discussed previously, breast milk composition is not static; it varies within and between women. Likewise, energy requirements are extremely variable, amongst the general population as well as lactating women. Consequently, thiamin requirements cannot be set as a single figure for all women. It is important to be aware of the safety margin in the RDI and of the variability that exists between women in their thiamin intakes and requirements.

Riboflavin
Recommendations for riboflavin intake are based on transfer of 0.3 mg/day into milk. Use of riboflavin for milk production is estimated at 70%, meaning that 0.4 mg is needed. This amount is added to the estimated average requirement (EAR) for non-pregnant women and takes the total RDI to 1.6 mg/day.

Niacin
During lactation the Australian EAR for niacin is an additional 2.4 mg niacin equivalent per day based on an estimated requirement for niacin of 1.0 mg niacin equivalents per 1000 KJ, plus 1.4 mg secreted in 850 mL milk.

Vitamin B₆
Similarly the RDI for vitamin B₆ for lactating women increases to 2.0 mg/day and is based on the additional protein requirement for lactation plus 0.25 mg per day to provide for the amount secreted in the milk.

Vitamin B₁₂
The Australian RDI for vitamin B₁₂ for lactating women has been set at 0.4 µg higher than for the non-lactating woman. This figure includes 0.3 µg for the daily output of vitamin B₁₂ in breast milk. Vitamin B₁₂ deficiency can arise in infants exclusively breastfed by mothers with a very low vitamin B₁₂ intake, for example vegans.

Folic acid
As discussed in the section on pregnancy, folic acid deficiency during and shortly after pregnancy is not uncommon due to the demands of increased cell turnover in the foetus and placenta, and the secretion of folate in the milk. The average amount of folate secreted in milk is only about 75 µg/day, but because of the high demand
for folate during pregnancy, and the lack of information about the availability of folate from the diet, a large margin of safety is incorporated into the RDI, which increases to 500 µg/day.

**Vitamin C**

The Australian RDI for vitamin C for lactating women is 85 mg/day. On average 50 mg per day is secreted daily in the milk, although this varies considerably between women. The minimum amount of vitamin C that will prevent scurvy is 10 mg per day. The RDI for vitamin C in lactating women, therefore, does not have a high margin for safety.

When milk vitamin C concentration is low, small increases can be achieved with maternal vitamin C supplements. However, once a critical milk vitamin C concentration is reached (5–11 mg/100 mL), any extra vitamin C ingested by the mother is excreted in the urine. Vitamin C is actually concentrated in breast milk, the milk concentration being significantly higher than the plasma concentration. If a mother who is not saturated with the vitamin takes extra vitamin C, her milk vitamin C concentration increases before any excess is excreted in the urine. If vitamin C is then withdrawn from the diet, the urinary excretion decreases before the milk vitamin C concentration does. The vitamin C concentration in breast milk can therefore give some idea of maternal vitamin C status. What governs the critical milk vitamin C concentration for each woman is not known. It does not appear to be the plasma vitamin C threshold.

**Vitamin A**

The average vitamin A content of breast milk is 31 µg/100 mL and a daily milk yield of 850 mL therefore results in ~260 µg vitamin A being transferred from mother to child. The Australian RDI for vitamin A for lactating women is of the same order, 1100 µg RE/day.

The concentration of vitamin A in milk usually reflects the long-term adequacy of the maternal diet during pregnancy and lactation, and is frequently lower in developing countries than in most Western countries. In women with depleted vitamin A stores, supplements appear to satisfy the maternal requirements before altering the milk vitamin A concentration.

As with fat content, the vitamin A content of breast milk increases during a feed and varies between women. The ‘average’ milk vitamin A concentration is therefore correspondingly difficult to estimate.

**Minerals**

In general, the Australian RDIs for minerals and trace elements for lactating women consist of the basic requirement for adult females plus a safety margin and an allowance for the amount secreted daily in the milk. There are, however, some points about the RDI for individual minerals and trace elements that warrant further discussion.

**Iron**

The 1991 RDI for iron for lactating women was the same as that for non-lactating women, whereas the 2006 RDI is lower for iron during lactation (9 mg/day) versus 18 mg/day for women (18–50 years). This is because, although during lactation there is a small maternal iron loss each day in the milk this is offset by the lack of
menstrual loss during much of the period of breastfeeding. The total daily iron loss during lactation generally does not exceed 1.05–1.25 mg per day. An increase in maternal iron intake is not reflected in a change in the milk iron concentration, even when iron supplements are taken.

**Calcium**
Calcium, at a concentration of around 330 mg per litre, is the major mineral of breast milk. Maternal calcium loss during lactation is about 250 mg per day in the milk and 100 mg in the urine, a total of 350 mg daily. If calcium absorption is maximal at this time (approx. 40%) the necessary calcium intake is only 800 mg. The 2006 nutrient reference values (NRVs) have no additional allowance for lactation.

**Zinc**
The 2006 RDI for zinc during lactation is an additional 4 mg per day (from 8 mg/day to 12 mg/day). This figure is based on an average milk volume of 850 mL per day. The lactation recommendation was based on consideration of the additional needs for milk production together with estimates of zinc released for use because of decreasing maternal blood volume. This averages about 30 mg/zinc that can be reused Absorption is somewhat higher in lactation at 42% giving an additional dietary zinc requirement of 3.2g. Absorption is higher from animal foods than plants sources, so vegetarians, particularly strict vegetarians, will need intakes about 50% higher.

**Iodine**
The 2006 EAR for lactation was based on the adult female needs (100 µg/day), together with replacement needs for iodine secreted in breast milk (90 µg/day). The RDI was set assuming a CV of 20% and is set at 270 µg/day.

**Vitamin D**
The body stores of nutrients accumulated during pregnancy and during lactation affect the offspring’s stores and can lead to deficiencies in both mothers and infants. In 47 Melbourne women who were identified as Vitamin D deficient during their pregnancy 40% of their infants had vitamin D deficiency at 4–10 months (Thomson et al. 2004). Supplements are available for both maternal and infant needs and in some countries, routine supplementation of breastfeed infants with vitamin D drops is part of routine care. There are some groups in Australia who are susceptible to vitamin D deficiency, dark skinned women, housebound women and women who are heavily veiled.

**Food requirements during lactation**
Table 9.2 shows the diet patterns recommended by the NHMRC for lactating women aged 19–50 years and lactating adolescents (NHMRC 2011). The eating patterns of certain groups within Australia will differ from that reflected by the sample menu in Table 9.2. Such groups include vegetarians, those who are socioeconomically disadvantaged, migrants, and those with very high or low activity levels.

In the case of women with very low activity levels weight gain must be carefully monitored throughout pregnancy and lactation, and food intake modified accordingly. However, only very rarely will a woman require less than the
minimum number of recommended serves from each food group. In such women there may be a need for dietary supplements of vitamins and minerals.

Table 9.2 Recommended dietary patterns for lactating women aged 19–50 years and lactating adolescents of average height and with sedentary to moderate activity levels

<table>
<thead>
<tr>
<th>Food group</th>
<th>Example serving sizes</th>
<th>Recommended number of serves per day</th>
<th>19–50 years and lactating</th>
<th>Adolescent and lactating</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Vegetables and legumes / beans</strong></td>
<td>½ cup cooked vegetables (75 g)</td>
<td></td>
<td>7½</td>
<td>5½</td>
</tr>
<tr>
<td><strong>Fruits</strong></td>
<td>1 medium piece of fresh fruit (150 g)</td>
<td></td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td><strong>Grain (cereal) foods</strong></td>
<td>1 slice of bread or ½ a medium roll or flat bread (about 40 g)</td>
<td></td>
<td>9</td>
<td>9</td>
</tr>
<tr>
<td><strong>Lean meat and poultry, fish, eggs, nuts and seeds, and legumes / beans</strong></td>
<td>65 g cooked lean red meats or ½ cup of lean mince, 2 small chops 80 g cooked poultry 100 g cooked fish fillet or 1 small can of fish 2 large eggs 170 g tofu 30 g nuts or seeds</td>
<td></td>
<td>2½</td>
<td>2½</td>
</tr>
<tr>
<td><strong>Milk, yoghurt, cheese and/or alternatives</strong></td>
<td>250 mL cup of milk 200 g tub of yoghurt 40 g or 2 slices of cheese 120 g ricotta cheese</td>
<td></td>
<td>2½</td>
<td>4</td>
</tr>
<tr>
<td><strong>Additional serves from the five food groups or discretionary choices for those who are taller or more active</strong></td>
<td></td>
<td></td>
<td>0–2½</td>
<td>0–3</td>
</tr>
</tbody>
</table>

(NHMRC 2011)

Those with very high energy requirements, such as athletes, can meet these requirements by increasing the number of serves from all of the food groups and from cereal foods in particular.

Migrants often consume different foods from those commonly eaten in the Australian diet, but these still need to be chosen from the same basic food groups.
An understanding of the types of foods commonly used must always be obtained before recommendations concerning the appropriate amounts of different foods can be made.

**Advantages of breastfeeding – the mother’s perspective**

Breastfeeding promotes physiological recovery from pregnancy by promoting uterine involution and decreasing risk of postpartum haemorrhage. Breastfeeding also acts as a natural contraceptive by increasing the period of postpartum anovulation. Breastfeeding promotes the psychological attachment between mother and child, saves time and money spent on the preparation of infant formula and sterilisation of equipment. Despite the recognised benefits of breastfeeding, rates of initiation and length of duration fall short of national targets. The article by Scott et al. in your readings details factors associated with the initiation and duration of breastfeeding. Read through this article and make notes on the factors positively and negatively associated with breastfeeding. Further insights into the reasons for early cessation of breastfeeding can be found in the Melbourne INFANT study (Melbourne Infant Feeding Activity and Nutrition Trial). Within this study 58% of the mother’s continued to breastfeed their infants at 6 months. As observed previously, the mothers that continued to breastfeed their infants were more likely to be living in areas of high socioeconomic position, have a lower BMI and less likely to be working. The most common reason given by mothers for discontinuing breastfeeding prior to 6 months was ‘I felt I wasn’t making enough milk’. Such comments may suggest a lack of confidence in breastfeeding that may be modifiable via a peer support. Indeed mothers who were participants of a ‘mother’s group’ containing a high proportion of women continuing to breastfeed their infants, were 2.1 times more likely to continue to breastfeed at 6 months (Cameron et al. 2010). These data suggest the benefits of positive peer influence on the continuation of breastfeeding and provides some practical insights into strategies that can help to further promote the continuation of breastfeeding.

---

**PRINT READING**

*Please read Scott et al. (2006), ‘Predictors of breastfeeding duration: evidence from a cohort study’.*

**Contraindications to breastfeeding**

The genetic disorder galactosemia requires that the infant receives a feed free of lactose; failure to do so will jeopardise the health of the infant. Mothers who are HIV positive should probably not breastfeed, nor should mothers who test positive for active tuberculosis. Breastfeeding is not advised if the mother is receiving chemotherapy or radiotherapy for breast cancer.

The concentration of ethanol in breast milk is known to reach levels equivalent to those found in maternal blood. The infant may develop pseudo-Cushing syndrome if the mother consumes large amounts of alcohol during lactation. Nicotine enters breast milk and can cause nicotine poisoning of the infant. Alcohol consumption and cigarette smoking are not advised during lactation.
Caffeine can pass from the maternal bloodstream into breast milk and can accumulate over time in the infant. This may manifest in hyperactive infants, particularly when a mother consumes 6–8 cups of caffeine-containing beverages in a day.

**Common nutritional concerns**

Women following a vegan diet present a serious nutritional problem in Australia. Without supplements of vitamin B12, their infants can become so deficient in vitamin B12 as to incur irreversible neurological damage. Because vegetarian diets tend to be bulky, a lactating woman with a small appetite may have difficulty eating enough food to meet her nutritional requirements. Vegans need dietetic counselling during pregnancy and lactation.

**TEXT QUESTIONS**

1. What are the essential requirements for successful lactation?
2. How is milk secretion initiated after birth?
3. What is the role of the infant in maintaining lactation?
4. What is the ‘let-down’ reflex, and why is failure of this reflex associated with failure of lactation?
5. The composition of human milk is influenced by numerous factors. What are these factors, and how do they influence the composition of the milk?
6. What is the usual basis for determining the additional energy and nutrient requirements associated with lactation?
7. Which nutrients in the maternal diet have an immediate (short-term) influence on milk composition and which affect it over longer periods?
8. What are the recommended number of serves of each food group for adult, pregnant and breastfeeding women? Explain why the food groups increase or stay the same during pregnancy and lactation?
9. Which groups within the Australian population are most likely to be at risk of nutrient deficiencies during pregnancy and/or lactation, and how can any associated risks to the foetus and the mother be minimised?
10. Prepare a table with the following headings: nutrient; RDI – women aged 19–50 years; RDI – Pregnancy; RDI – lactation. For energy, protein, folate, iron, iodine, calcium, vitamin C note the RDI, note any increases for pregnancy and lactation (for some nutrients there may be no increase). Also comment on whether women in this country can meet these requirements.
11. Why is an infant who is exclusively breastfed to the age of nine months likely to develop iron deficiency anaemia?

**References**


# TOPIC 10
## Nutrition for successful ageing

**PREPARED BY THE UNIT TEAM**

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Introduction

Adequate nutrition in later life is an important component of successful ageing. The ageing process starts to present many challenges for accessing, preparing and consuming appropriate food. Decreased appetite and ability to taste, smell and appreciate food flavours, combined with increasing social isolation, drug-nutrient interactions and mobility, can start to compromise the ability of older people to meet their nutrient needs. Changes in energy expenditure, absorption and synthesis of nutrients result in specific nutrient needs for this life stage. Ensuring adequate nutritional intake in later years can significantly improve the quality of life of this growing proportion of the Australian population.

Learning objectives

After completing this section, students should have an understanding of the:

- changing demographics of the ageing population
- physiological changes associated with ageing
- influence of ageing on body composition
- nutritional requirements of ageing
- common nutritional concerns for the ageing.

Learning resources

Print readings

The readings listed here are reproduced in the print readings for this unit.


Deakin Studies Online (DSO)

Access the unit site on DSO <http://www.deakin.edu.au/current-students/> for the appendices and other additional resources you can use to supplement this topic.

Demographics of ageing

Life expectancy

The number of people surviving beyond maturity and into old age has increased and is continuing to increase throughout the world. One of the most commonly cited and useful indicators of long-term trends in health is life expectancy. It is the average number of years of life remaining to a person at a specified age if current
age-specific mortality experience does not change during the person’s lifetime. At the beginning of the 20th century, the life expectancy for Australian males was around 55 years and close to 59 years for Australian females (Figure 10.1). In the early 1920s, life expectancy at birth had risen to 59 years for males and 63 years for females. Although there was a plateau in the 1960s, due largely to the increasing burden of cardiovascular disease at this time, the trend for an increase in life expectancy has since continued in both males and females. From 1901–1910 to 2005–2007 male life expectancy at birth increased by 24 years, similarly female life expectancy at birth increased by 25 years (Figure 10.1). An Australian male born in 2005 could expect to live 79.0 years, and a female could expect to live 83.7 years (ABS 2008).

Figure 10.1  Life expectancy trends at birth, 1907–2007

Proportion of older people in the population

Another important indicator of trends in the health of Australians during the 20th century is the proportion of older people in the population. In the first quarter of the century, less than 5% of the population were aged 65 and over, and the proportion of those aged 80 years and over was below 1%. These proportions increased consistently throughout the century, with more than 13.3% of the population aged 65 years and over in 2009, compared with 8.3% in 1971 (Figure 10.2) (AIHW 2010). The proportion of those aged 85 and over also crossed the 2% level in the 1990s. Growth in this age group has continued to steadily rise, with the number of people increasing more than five-fold from the early 1970s to 2009 (Figure 10.2) (AIHW 2010). There has also been a marked increase in the number of Australians aged 100 years and over, increasing from 200 in 1971 to more than 3700 in 2009 (AIHW 2010).
More females than males survive to older age. In 1999, the sex ratio among those aged 65 years and over was 78 males to every 100 females. For those aged 80 years and over, the ratio was 54 males to 100 females. Age, particularly advanced age, is a significant predictor of poor health and disability. Chronic diseases and conditions such as arthritis, heart disease, cancer and dementia are highly prevalent in the older population.

**Causes of death**

The four major causes of death in Australia are diseases of the circulatory system such as heart attack or stroke; neoplasms (cancers) such as lung, colorectal, breast and prostate cancer; diseases of the respiratory system such as asthma, emphysema and bronchitis; and injury and poisoning including motor vehicle accidents, falls and suicide. Diseases of the circulatory system and neoplasms are responsible for approximately 68% of all deaths in Australia. Although the proportion of deaths due to diseases of the circulatory system has been declining, deaths from neoplasms have increased slightly. The proportion of deaths due to neoplasms and respiratory diseases increased slightly over the past decade (Figure 10.3) (de Looper & Bhatia 2001).

In 2008 the four leading causes of death were ischaemic heart diseases (16%), cerebrovascular disease (8.3%), dementia and Alzheimer’s disease and trachea and lung cancers (5.5%) (ABS 2008) (Figure 10.3). In comparison to 1999, the proportion of deaths due to ischaemic heart disease has fallen by 6%, similarly the proportion of deaths due to cerebrovascular disease has also decreased (1.3%). In contrast the number of deaths due to dementia and Alzheimer’s disease has seen a marked increase (138%). Similarly the proportion of deaths due to trachea and lung cancers has risen 0.2% from 1999 to 2008 (ABS 2008). The major causes of death by gender are depicted in Figure 10.4.
The fact that people now live longer is an indication that overall health experience is better now than it was in the past. However, while it is now possible to prevent many early deaths and to prolong the life expectancy of those in middle age, the problems associated with ageing have not been solved. Older adults experience considerably more ill health than younger adults and the proportion of older people...
affected by ill health increases markedly with age. In fact, it has been suggested that most of the recent gains in life expectancy are associated with increased years of disability and not with increased years of active life. However, whether or not this is actually occurring is difficult to determine. The most recent report from the AIHW (AIHW 2006) indicates that age-specific prevalence rates of disability appear relatively stable, however, due to the ageing of the Australian population and the greater longevity of individuals there are more people, especially those at older ages, with a disability and a severe or profound core activity limitation.

**Self-reported prevalence of disability**

Disabilities and core activity restrictions are long-term consequences of a health condition, impairment, disease or accident that can have a severe impact on the quality of life of the affected person. The 1998 ABS Survey of Disability, Ageing and Carers defines ‘disability’ as the presence of one or more of 17 ‘limitations, restrictions, or impairments’ identified by survey respondents. According to this definition, more than 3.6 million people in Australia reported a disability in 1998. Disability is strongly related to age and sex. The rates are higher among males and increase rapidly after the age of 45. More than one out of two persons aged 65 years and over reported at least one disabling condition in 1998. Arthritis and musculoskeletal disorders are the most commonly reported disabling conditions. Many other chronic diseases such as coronary heart disease, stroke and diabetes are also significant contributors to disability. The proportion of people with a disability appears to be rising. However, the majority of the apparent increase in disability rates is a result of increased identification of people with disabilities, rather than a substantial increase in the prevalence of people with disabilities (de Looper & Bhatia 2001).

The relative contributions of nutrition and other lifestyle factors, both in the ageing process and in relation to the development of ill health and disability in old age, are important issues to be considered.

**Physiological aspects of ageing**

Ageing is a normal phenomenon and has been defined as ‘regression of physiological function accompanied by advancement of age’. In effect ageing begins at conception, but during the first 20 to 25 years of life, the genetically determined features of ageing are growth of body tissues and development of physiological functions. Before maturity, signs of regression are clearly evident only in the immunological system.
The ageing process, while genetically determined, is clearly also influenced by environmental factors. The marked increases in life expectancy during the twentieth century are a clear indication of the role of environmental factors in the ageing process. However, as average life expectancy increases and approaches the maximum attainable life span, it is inevitable that the gains in life expectancy occur at ages when the ageing process is well advanced, and older persons are at risk for nonfatal and often disabling conditions such as dementia, osteoporosis, sensory impairments and musculoskeletal disorders.

**Physiological changes with age**

Even during growth and development there is a constant process of cell destruction in body tissues. What is different after maturity is that the cells themselves and the processes associated with the growth and replacement of damaged cells begin to function less efficiently. The rate and degree of failure varies from system to system. Figure 10.5 illustrates the deterioration observed in various biological measurements, expressed as a percentage of performance at age 30 years. What is notable is that every single measurement shows a decline throughout the age range.

**Major organ systems**

As already indicated, ageing affects all of the major organ systems of the body, including the central nervous, endocrine, cardiovascular, renal, pulmonary and digestive systems.

**Central nervous system**

The changes in mental function associated with normal ageing are not well defined. Neurones lose their capacity to divide after cell differentiation, while glial cells continue to divide throughout life. The functional significance of the changing ratio of these two types of cells is not known. At one time it was thought that normal ageing was characterised by brain atrophy and a significant loss of neurones.
Recent work suggests that some degree of neuronal loss occurs after age 50, but major loss of neurones only occurs in individuals suffering from brain diseases. The ageing brain also seems to be capable of some degree of regeneration and the ability to form additional dendrites. This makes new connections possible and may compensate to some extent for the loss of nerve cells. In normal older people neither brain size nor cell number is related to mental function. In the elderly there is an increase in the incidence of brain disorders such as Parkinson’s disease and dementia.

Endocrine system
Many components of the endocrine system undergo changes but the changes are not uniform in direction. Essentially the changes are the result of structural and metabolic changes in endocrine-related target tissues. For example, the reduction in lean body mass is thought to be the basis for the age-related decline in thyroxin levels. Changes in the synthesis of carrier proteins also influence not only blood levels, but also rates of conversion to active forms. Altered endocrine function may contribute to an impaired ability to adapt to changes in nutrient intake.

Cardiovascular system
Cardiac structure and function in older individuals are the result of age-related changes complicated by lifestyle factors and disease. In healthy older men and women heart weight does not decrease in relation to body size, but modest increases in the thickness of the left ventricular wall occur even in normotensive individuals. In the major arteries changes related to ageing occur independent of atherosclerotic disease. The aorta increases in diameter and length and the arterial walls stiffen and lose their elasticity due to structural changes in their elastin and collagen and to the deposition of calcium.

For most healthy older people, cardiac function is sufficient to meet physiologic needs at low or moderate levels of activity. The decrease in maximum oxygen consumption observed in healthy older people may be due to changes in the peripheral tissues rather than to changes in cardiovascular function. A loss of lean body mass reduces the maximum possible work rate and maximal oxygen consumption. If cardiac function is markedly impaired, the reduced cardiac output will compromise the function of other organ systems. In individuals with cardiovascular disease cerebral blood flow may be reduced by 20% and renal blood flow by as much as 60%.

Renal function
Kidney function can decrease with age both as a result of the loss of nephrons and to changes in blood flow. Kidney mass may decrease by as much as 30% by 90 years of age and the accompanying structural changes contribute to the age-related decline in the glomerular filtration rate (GFR) of around 1 mL per minute per year after the age of 30 years.

The older kidney is also less able to increase the rate of urine flow and to increase urine osmolality. These changes limit the capacity to excrete waste products and may become critical in older individuals with low fluid intakes and high intakes of protein and electrolytes. Megadoses of vitamins, taken on the assumption that what is not needed can be readily excreted, may accumulate at high levels and drug dosages may also need to be adjusted in individuals with significant loss of kidney function.
Pulmonary system
Changes associated with ageing reduce both the ventilation of the alveoli and the exchange of oxygen and carbon dioxide across the alveolar membrane. Structural changes in the lung tissues cause a decrease in elasticity and loss of alveolar surface area. The alveolar membranes also weaken and stretch and the air sacs become larger with the collapse of some small airways. The surface area of gas exchange can decline by as much as 30% in advanced age.

Gas exchange is also less efficient because of thickening and reduced permeability of alveolar membranes and a reduced blood flow through the alveolar capillaries. Oxyhaemoglobin saturation in older people is thus only about 90% as compared with 96% in younger people.

Because the chest wall stiffens and is less easily expanded, the work of breathing is increased. Total lung capacity does not change significantly, but the proportion of alveolar space that is ventilated with each breath decreases and the residual volume increases. As a consequence, even healthy older people who are free of lung disease can still be vulnerable to complications associated with infections, such as pneumonia, that lead to reduced oxygen levels.

Gastrointestinal function
While impaired digestive or absorptive capacity is frequently reported in older hospitalised or institutionalised people, these observations are more likely to reflect the effect of disease states or the use of medications rather than the effect of ageing. In general, most gastrointestinal functions are well preserved in older people probably because the intestine, pancreas and liver all have a large reserve of functional capacity. Clinically important changes in gastrointestinal function with ageing include decreased taste threshold, hypochlorhydria due to atrophic gastritis and decreased liver blood flow and size.

Taste acuity does decline with age (Schiffmann 1994) and can affect both the amounts and types of food consumed. In addition, some medications and disease states also impair taste perception. A loss of teeth and/or problems associated with ill-fitting dentures can also affect food intake.

Atrophic gastritis increases in prevalence with ageing and appears to be linked with the presence of Helicobacter pylori organisms in the upper gastrointestinal tract. As a result of atrophic gastritis, there is decreased secretion of acid and intrinsic factor and this in turn leads to impaired absorption of vitamin B12. In the small intestine, atrophic gastritis causes a rise in pH which reduces the bioavailability of calcium, iron and folate (Russell 1997).

Pancreatic secretion decreases somewhat with age and the sensitivity of the gall-bladder to cholecystokinin is also reduced. The effects of these changes, however, are evident only when intakes of fat and protein are above levels of greater than 100 g fat/day and 1.5 g protein/kg/day and thus not of practical importance except when high energy diets are given to older people as part of diet therapy. Mixed meals containing up to 200 g carbohydrate have also been reported to not be well absorbed in a large proportion of elderly subjects, but not in younger subjects, but it is not clear whether this was due to maldigestion and/or absorption of the carbohydrate in the small intestine or to a different bacterial flora present in the small intestine of the older subjects.
Both liver size and portal blood flow decrease with age and these decreases alone may account for the decrease in drug elimination rates observed in the elderly.

The mucosal surface of the small intestine has been reported to be slightly less than in younger subjects but the evidence for a decrease in absorptive function in older as compared with younger individuals is limited to absorption of zinc, calcium and possibly carbohydrate.

There is some degree of mucosal atrophy and a weakening of the smooth muscle layer of the colon with ageing but this does not impair the most important functions of the colon which are the absorption of water, electrolytes and bile acids and the fermentation of non-starch polysaccharides. The major structural change in the colon is the development of diverticula and the major functional change is the reduction in motility that can lead to constipation.

**Malnutrition**

Refusal to eat is common in the elderly in both community and institutional settings. There is some controversy concerning the nature of anorexia with respect to ageing. Older patients with anorexia have been found to have lower levels of some hormones involved in appetite e.g. lower plasma β-endorphin and increased concentrations of cholecystokinin.

**Physiological changes associated with ageing that affect energy balance**

- Energy intake ↓
- Basal metabolic rate (BMR) ↓
- Lean body mass ↓
- Appetite ↓
- Responsiveness to opioid-induced feeding drive ↓
- Neuropeptide Y ↓
- Satiety ↓
- Gastric emptying ↓
- Nitric oxide ↓ leading to ↓ adaptive relaxation of stomach
- Postprandial cholecystokinin secretion ↑ in elderly with undernutrition
- Chemical senses ↓
- Taste ↓ (accuracy and threshold)
- Smell ↓↓ (accuracy and threshold)

(Marcus & Berry 1998)

**Body composition**

One of the major changes that occur with ageing is a loss of lean body mass. This is caused by a reduction in skeletal muscle activity and by a loss of cells from body tissues and organs. The relative roles played by ageing and by a sedentary lifestyle in the reduction of lean body mass remain to be established, but it is likely that reduced physical activity with increasing age is at least partly responsible.
In contrast to lean body mass, body weight tends not to decrease with age, except in advanced old age. A consequence of the maintenance of body weight is that ageing is also accompanied by an increase in the proportion of body fat and a decrease in lean mass and the percentage of body water, 60% in adults compared to 50% in the elderly (Table 10.1).

### Table 10.1 Body composition changes in Caucasian adult males

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>% fat mass</th>
<th>Lean Mass/Height² (kg/m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>20</td>
<td>23.4</td>
<td>19.0</td>
</tr>
<tr>
<td>30</td>
<td>25.7</td>
<td>19.6</td>
</tr>
<tr>
<td>40</td>
<td>27.5</td>
<td>20.0</td>
</tr>
<tr>
<td>50</td>
<td>29.0</td>
<td>20.2</td>
</tr>
<tr>
<td>60</td>
<td>30.5</td>
<td>19.9</td>
</tr>
<tr>
<td>70</td>
<td>31.4</td>
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</tr>
<tr>
<td>80</td>
<td>31.6</td>
<td>18.6</td>
</tr>
</tbody>
</table>

(Adapted from Kelly et al. 2009)

An increased proportion of body fat predisposes individuals to the development of glucose intolerance and to higher blood insulin levels and an increased risk of diabetes at older ages, while the decrease in body water increases the susceptibility of older people to dehydration.

The distribution of body fat also changes with age and more adipose tissue tends to accumulate in the abdominal region and to increase the adverse metabolic effects associated with obesity.

### Nutritional requirements and common nutritional concerns

The physiological changes that occur with age affect the requirements for several nutrients. In addition, the higher incidence of disease and the widespread use of medications by older people also have an impact on nutritional requirements. Until recently few studies have attempted to define the nutrient needs of older people and reference values for older people are usually based on values extrapolated from information on younger adults. Recommended nutrient intake values are usually expressed according to chronological age, but older people (> 65 years) represent a very heterogeneous group both in terms of physiological features and health status.

**PRINT READING**

Please now read Rivlin (2007), ‘Keeping the young-elderly healthy’.

The nutrient reference values (NRVs) and the 2011 Australian Guide to Healthy Eating now include two age categories of older people 51–70 years and > 70 years, recognising that dietary requirements are altered by ageing, with the older group requiring greater amounts of some nutrients. It is difficult to define the appropriate
criteria for the establishment and expression of nutrient requirements for elderly persons. For example, should nutritional requirements for older people be adjusted on the basis of observed age-associated changes in body composition and physiologic function or should optimal body composition and levels of function, including physical activity level, be determined for different age groups and nutrient recommendations developed to achieve them? One possible solution is to ‘adjust’ the recommendations for younger adults to take into account factors such as disease, drug use, nutritional status and physical activity as well as age and sex.

It is important to note that older people are an extremely diverse group and therefore it is not appropriate to make the same nutritional recommendations for all.

Groups of elderly
- ‘Young’ elderly i.e. 55–70 years.
- ‘Old’ elderly i.e. 70+ years.

Other classifications (functional age)
- Well fit elderly.
- Disabled elderly.
- Frail elderly.
- Elderly living at home.
- Elderly living in residential care.

NB: Most elderly suffer from some form of chronic illness.

The 2006 NRVs have increased recommendations for a number of nutrients for older adults compared with younger adults and compared with the 1991 recommended daily intakes (RDIs), e.g. vitamin D, folate, vitamin B₆ and calcium. The 2006 NRVs for Australia recommend the same RDI for folate for all adults 400 µg, which is twice that of the previous 1991 RDI, the 2006 RDI for vitamin B₆ is higher in those over the age of 50 years increasing from 1.3 mg/day in men to 1.7 mg/day, and from 1.3 to 1.5 mg/day for women after the age of 50 years. For older adults, the estimated average requirement (EAR) appears to be higher, and there is a gender difference with higher requirements in men. The increase due to age and gender appears to be in the order of 0.2 to 0.3 mg of food vitamin B₆ a day. Vitamin D increases progressively from 51–70 years and 70+ years.

Energy requirements, overweight/obesity and underweight

Energy expenditure and physical activity
Basal metabolic rate (BMR), the major component of total energy expenditure, is around 10% lower in 70-year-old adults than in young adults aged 18–30 years because it depends largely on the amount of lean body mass (Figure 10.6).
Physical activity is the second largest component of energy expenditure in adults. A reduction in physical activity contributes directly to a reduction in total energy expenditure through a decrease in the amount of energy expended and indirectly through loss of lean body mass and the resulting reduction in BMR. In addition to its negative influence on lean body mass, a decrease in total energy expenditure in older people may also be accompanied by a decrease in food intake and a concomitant decrease in nutrient intake.

Energy requirements for Australian adults are calculated from BMR estimated on the basis of age, sex and weight. The estimate of BMR allows for a reduction in lean body mass with age. For adult males aged 19–30 years, weighing 79 kg with a BMI of 22 kg/m² and with a physical activity level of 1.6 × BMR, the average estimated energy requirement is 12.6 MJ per day, whereas for adult male of the same height, weight and level of physical activity aged > 70 years the energy requirement is reduced to 10.2 MJ/day (NHMRC 2006; see Appendix 2.)

**Energy intake**

Mean energy intakes reported in the 1995 National Nutrition Survey (ABS 1998) approximated these recommendations for energy intake in younger males (1.5 × BMR), but were lower in older males (1.4 × BMR) and in both younger (1.3 × BMR) and older females (1.2 × BMR). Since the average body weight of the male and female National Nutrition Survey subjects was close to 80 and 65 kg, respectively, the findings for energy intake suggest that current energy expenditure levels in older males and females are lower than desirable or that energy intake was underreported.

**Body weight**

According to data collected in the 1995 National Nutrition Survey (ABS 1998), the proportion of adults who are overweight or obese starts to decline in males at approximately 50–54 years of age and at 60–64 years of age in women. Weight loss is an emerging problem in the elderly and being underweight confers a greater mortality risk. Weight loss is generally associated with a reduction in skeletal muscle mass which can lead to an increased susceptibility to injury and bone fracture.
Fat intake and cardiovascular disease

Coronary heart disease is a significant cause of mortality and morbidity in the elderly. High plasma LDL cholesterol is one of the risk factors for the development of coronary heart disease. Plasma LDL cholesterol levels can be reduced by decreasing saturated fat intakes. It has been recommended that saturated fat intake should not exceed 10% of total energy intake. Current saturated fat intake, expressed as the mean contribution to energy intake, was 12% in males aged 65 and over and 12.4% in females aged 65 and over (see Appendix 4 on DSO).

Calcium and osteoporosis

Bone mass in old age is determined both by the peak bone mass achieved in young adult life and by the rate of loss associated with ageing. The reduction in bone mass resulting from osteoporosis is associated with increased rates of bone fracture, particularly of the vertebrae, wrist and femur. Osteoporosis occurs predominantly in old age, and affects more women than men. In the 1995 National Health Survey, 11% of women aged 65–74 years reported osteoporosis as a long-term condition. Osteoporosis is a multi-factorial condition, which is influenced by hormonal, genetic and environmental factors. The latter include physical activity, smoking, alcohol consumption and diet.

Calcium and vitamin D are the main nutritional factors associated with osteoporosis. Recommendations to reduce the prevalence of osteoporosis are aimed primarily at maximising peak bone mass during the second and third decade of life. In later life, recommendations for reducing the prevalence of osteoporosis are aimed at maintaining bone mass and usually include a combination of hormone replacement therapy, weight-bearing exercise and supplements of both calcium and vitamin D. The RDI for post menopausal women increases to 1300 mg/day to reflect the loss of calcium from skeleton, which mainly results from an increase in urinary losses rather than changes in absorption. In older individuals, the focus of preventive measures is on ways to reduce the risk of falls, increasing exposure to sunlight when possible, supplementation with vitamin D and on provision of an adequate calcium intake from the diet. In frank vitamin D deficiency, the condition which develops is osteomalacia not osteoporosis. Osteomalacia results from the accumulation of unmineralised bone matrix and is not due to a loss of mineralised bone. The mean intake of calcium for women aged 65 and older was 685 mg/day, well below the RDI for calcium (see Appendix 4 on DSO).

Fibre, fluid and constipation

Fibre

Cereals breads and pastas are an excellent source of dietary fibre and its intake has been linked to a reduced prevalence of coronary heart disease, obesity, diabetes, cancer, hypertension, constipation and diverticular disease. The presence of chewing and swallowing difficulties can deter the elderly from consuming sufficient quantities of fibre.

Fluid

Older people are at greater risk of water imbalance due to a decrease in fluid consumption and an increase in fluid loss. Dehydration should be avoided as it is associated with increased risk of developing hypotension, elevated body
temperature and constipation. It has been recommended the elderly should consume 2.1–2.6L/day (fluid only component). The mean daily intake of fluid was 1644 g/day and 1714 g/day for both males and females, respectively (see Appendix 4 on DSO). These intakes appear to be below the recommended range. Anecdotally it is known that the elderly consume insufficient amounts of fluid.

**Constipation**

The clinical definition of constipation includes:

- straining when passing a stool on 25% or more occasions
- < 3 stools/week
- feeling of incomplete evacuation more than 25% of the time
- hard stools more than 25% of the time.

Constipation can be also defined by an individual’s self perception of the problem.

There are limited data on the prevalence of constipation in Australia. It has been estimated approximately 30% of the elderly population are constipated. These are predominantly based on self-reported data and are likely to be overestimated. Other factors that contribute to the development of constipation include the use of medications and inadequate physical activity.

**Salt intake and hypertension**

The elderly, with established hypertension, are usually more responsive to a lower salt intake. Sodium intake was not measured in the 1995 National Nutrition Survey; however, studies that have looked at mean urinary sodium excretion, which reflect sodium intake, indicates that intakes generally exceeds the RDI.

**Alcohol**

Alcohol contributes approximately 5% of energy intake and consumption declines with age. The 1995 National Nutrition Survey observed that the average intake of alcohol and the proportion of people who had a drink on the day of the survey were lower for people aged over 65. Individuals who consume an average of 1–2 drinks per day have a better life expectancy than those who drink to excess (‘J-curve’). With ageing there is a diminished ability to metabolise alcohol leaving the older adult more likely to become intoxicated after drinking alcoholic beverages. Excessive alcohol intake will displace the appetite for more nutritious foods, contributing to an insufficient intake of macro- and micronutrients, increase the likelihood of a fall and can interfere with metabolism of commonly prescribed medications.

**Iron and iron deficiency anaemia**

The 2006 RDI of iron for males and females over the age of 50 years is 8 mg/day. The RDI for women is now similar to that of males due to the cessation of menstruation. Iron intakes are generally adequate and exceed the RDI (see Appendix 4 on DSO). Diet appears to play only a minor role in the development of iron deficiency anaemia, rather it is primarily associated with chronic disease. Pernicious anaemia, which is associated with atrophic gastritis, is predominantly a disease of advancing age and may affect 2–3% of older people.
Macronutrients

Protein

The NRVs for protein distinguish between males and females: adult females < 70 years 0.75 g/kg (body weight)/day versus males 0.84 g/kg/day. This increases over the age of 70 to 0.93 g/kg/day for females and 1.10 g/kg/day for males. Older adults, over 53 yrs, appeared to have 25% higher requirements for maintenance than younger adults. For this reason, the EAR for adults 70 years and over was increased by 25% over that of younger adults.

The average reported level of protein intake in the 1995 National Nutrition Survey was in excess of these amounts in both younger and older males and females (see Appendix 4 on DSO). It should be noted, however, that recent data on nitrogen balance studies in healthy elderly subjects indicate that protein requirements may increase with ageing. The British Nutrition Foundation suggests an increase ≥1.0 g/kg/day and may be necessary to cover the needs of nearly all elderly subjects (BNF 1996). The average intake of older Australian men and women appears to be adequate to meet this higher recommended level of intake for community dwelling elderly, although many in residential care establishments may be consuming inadequate amounts of protein (Nowson et al. 2003).

Micronutrients

Gastrointestinal function is well preserved with ageing with regards to the digestion and absorption of macronutrients, but the ageing gastrointestinal tract becomes less efficient in absorbing vitamin B<sub>12</sub>, vitamin D, and calcium. In addition lack of sunlight exposure, impaired skin synthesis and decreased responsiveness of the kidneys to parathyroid hormone impacts vitamin D intake requirements. The NRVs for Australia have taken into account studies in older adults and have increased the NRVs for vitamin D and calcium, but not for vitamin B<sub>12</sub>. The RDI for calcium increases progressively from 1000 mg/day for women 18–50 years to 1300 mg/day above 50 years, whereas for men the increase to 1300mg/day occurs after 70 years. The rationale for this is that for the elderly there is little known about calcium metabolism, but it is known that absorption decreases with age in both sexes. Although the data for increased need at menopause is strong, evidence for older men is not, but as a precaution an additional average requirement of 250 mg/day is recommended translating to an additional 300 mg of calcium for the RDI. The NRVs are also increased for protein, riboflavin, B<sub>6</sub>, with age. This is a rapidly advancing field, so it will be important to closely follow new research on nutrient requirements and ageing over the next several years.

Energy requirements, overweight/obesity and underweight

Table 10.2 presents the recommended dietary patterns that would meet the nutritional requirements for men and women aged 51-70 years and 70+ years who are of average height and have sedentary to moderate activity levels.
## Table 10.2
Recommended dietary patterns for men and women aged 51–70 and 70+ years of
average height and with sedentary to moderate activity levels

<table>
<thead>
<tr>
<th>Food group</th>
<th>Serve definition</th>
<th>Recommended number of serves per day</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Men aged</td>
</tr>
<tr>
<td></td>
<td></td>
<td>51–70 years</td>
</tr>
<tr>
<td>Vegetables and legumes / beans</td>
<td>1/2 cup cooked vegetables (75 g) 1 cup salad vegetables 1 small potato ½ cup cooked dried or canned beans or lentils, no added salt</td>
<td>5½</td>
</tr>
<tr>
<td>Fruits</td>
<td>1 medium piece of fresh fruit (150 g) 30 g dried fruit (e.g. 4 dried apricot halves) 1 cup canned fruit (150 g)</td>
<td>2</td>
</tr>
<tr>
<td>Grain (cereal) foods</td>
<td>1 slice of bread or ½ a medium roll or flat bread (about 40 g) ½ cup cooked rice, pasta, noodles ½ cup cooked porridge or polenta, 2/3 cup breakfast cereal flakes (30 g) or ½ cup muesli</td>
<td>6</td>
</tr>
<tr>
<td>Lean meat and poultry, fish, eggs, nuts and seeds, and legumes / beans</td>
<td>65 g cooked lean red meats or ½ cup of lean mince, 2 small chops 80 g cooked poultry 100 g cooked fish fillet or 1 small can of fish 2 large eggs 170 g tofu</td>
<td>2½</td>
</tr>
<tr>
<td>Milk, yoghurt, cheese and/or alternatives</td>
<td>250 mL cup of milk 200 g tub of yoghurt 40 g or 2 slices of cheese 120 g ricotta cheese</td>
<td>2½</td>
</tr>
<tr>
<td>Additional serves from the five food groups or discretionary choices for those who are taller or more active</td>
<td>0 – 2½</td>
<td>0 – 2½</td>
</tr>
</tbody>
</table>
Table 10.3 Micronutrient issues with ageing

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Nutritional issues</th>
<th>Activity:</th>
</tr>
</thead>
<tbody>
<tr>
<td>Protein</td>
<td>Loss of muscle mass, evidence that higher intakes required to maintain nitrogen balance</td>
<td>Review the NRVs and note any changes in the RDI for the following nutrients with age.</td>
</tr>
<tr>
<td>Riboflavin</td>
<td>High incidence low intakes in elderly (US 20–27%), 17–76% deficiency. No decrease in requirement with increasing age and reduced energy intake</td>
<td></td>
</tr>
</tbody>
</table>
| Vitamin B₆       | 10% elderly (US)  
↓ immune function                                                                                                                                                                                                                       |                             |
| Vitamin B₁₂      | High incidence of atrophic gastritis 20–40% in elderly which is associated with low B12 status.                                                                                                                                                           |                             |
| Folate/ vitamin B₁₂ | High intakes reduce homocystenuria (CVD risk)  
High intake reduces megaloblastic anemia                                                                                                           |                             |
| Zinc             | High incidence of low intakes  
Important in wound healing, immune function, taste sensation                                                                                                                                                                                  |                             |
| Vitamin A        | Older people—liver content of vitamin A does not decrease  
? ↑ absorption, ↓ clearance                                                                                                                                                                                                                 |                             |
| Vitamin D        | ↓ vitamin D receptors in intestinal mucosa  
↓ dietary intake  
↓ exposure to sunlight  
↓ ability to synthesise vitamin D in skin  
↓ kidney function—to convert to 1,25 (OHD) –active metabolite                                                                                                                                 |                             |
| Calcium          | Ca supplements + vitamin D  
↑ bone density and ↓ fracture risk                                                                                                                                                                                                                           |                             |
Factors affecting nutritional status in older people

Food patterns
A food intake pattern associated with a higher intake of breakfast cereals, fruits and milk is consistently associated with not only with higher intakes of nutrients, but also with higher blood nutrient levels. Because older individuals generally consume less energy, food patterns associated with nutrient-dense diets are more critical for the maintenance of an adequate nutrient intake in this age group than they are at younger ages. Older adults are recommended to consume at least three meals per day. The reasons for recommending at least three meals per day are:

• loss of appetite is common in the elderly
• living alone can lead to poor intake
• weight loss can lead to loss of muscle mass and poor recovery from illness
• limited ability to chew can lead to poor food intake
• those who ate 2–4 meals per day were more likely to meet the RDIs for macro- and micronutrients than those who ate fewer than two meals per day.

Ill health
Illness is probably the major factor influencing nutritional status in older people.

Most people suffer from an episode of acute illness as they get older, but may also have repeated episodes of ill health or a chronic disease that may contribute over time to an unobtrusive decline in their nutritional status. The adverse effect that ill health has on nutritional status is especially evident in older people. Ageing-related impairment of immune function increases the risk of infections. In turn, illness is likely to affect nutrient intake, and nutrient absorption and increase nutrient requirements. Some conditions may also incur a significant loss of nutrients and in this way increase requirements.

Illness can reduce food intake not only by reducing appetite, but also by interfering with mobility and the ability to buy, prepare and cook foods. In addition, drug treatments are often accompanied by side effects which include reduced appetite, nausea and sometimes vomiting. Illnesses and infections are often accompanied by a raised body temperature (fever) which increases the basal metabolic rate and by other metabolic changes which increase the requirement for energy, protein and specific nutrients.

Reduced appetite
Older adults frequently report a reduced appetite even when they are not ill; changes in sensory perception may be partly responsible. Taste acuity is diminished and the thresholds for detection of odours may be ten times higher than in younger adults.

Changes in hormonal responses can also reduce food intake. For example, satiety may be induced earlier, as the production of cholecystokinin, a gut hormone released in response to eating, is raised with age. Changes in the muscular structure of the intestinal wall which reduce the motility of the gut may also influence feelings of satiety.
Psychological factors such as bereavement, confusion and depression, and physical and social factors such as immobility and social isolation can all reduce appetite in the elderly.

**Poor dentition**
Older people have fewer teeth and many rely completely on dentures. Because of the physical changes associated with ageing, dentures may not fit very well and make chewing difficult. Inadequate dentition need not, but often does, influence nutrient intake because food choice is more limited and may not include foods which require chewing such as meats, fruits and vegetables.

**Drug-nutrient interactions**
Older people often take several drugs, some for long periods of time, and so may be at risk of drug-induced nutritional deficiencies. In addition, physiological and pathological changes associated with ageing may reduce the effectiveness of drugs and lead to higher doses being prescribed. At the same time, the rate of elimination of drugs tends to decline with age, probably because both liver size and blood flow decline with age.

Some drugs induce an increased requirement for a particular nutrient, while others affect taste and smell or may simply decrease appetite. Other drugs alter the absorption, metabolism and excretion of drugs.

In the elderly, drug-nutrient interactions can be classified as physicochemical, physiological and pathophysiological. Physicochemical interactions are characterised by binding with metal ions (chelation) to form chelation complexes and by changes in the stability of nutrients. Physiological interactions include drug-induced changes in appetite, digestion, gastric emptying, biotransformation and renal clearance. Pathophysiological interactions occur when a drug impairs nutrient absorption or inhibits nutrient-related metabolic processes.

Nutrition problems in the elderly may be related not only to multiple drug use, but also to the long-term consumption of special diets for one or more chronic illnesses. Drug-induced adverse outcomes can also compromise drug therapy by affecting the nutrition status of the patient. Hypertension, cardiac failure and renal insufficiency are of particular concern when using certain drugs. Digoxin, for example, has anorectic properties and some diuretics facilitate not only loss of sodium but also of potassium, magnesium, calcium and thiamin.

**Mobility and physical activity**
Regular exercise is an important component of successful ageing since it can significantly improve physical fitness, in older as well as younger adults. There is good evidence that regular physical moderate activity such as walking provides protection from cardiovascular disease (Bauman & Smith 2000).

Weight training has also been shown to lead to gains in muscle strength, muscle size and mobility even in the frail elderly. Prompt resumption of appropriate physical activity after bouts of illness also helps to maintain mobility and independence in the longer term.
From a nutritional viewpoint an important advantage of physical activity, and the associated increase in metabolic rate, is that a higher energy intake also makes it easier for older people to obtain an adequate intake of essential nutrients.

**Nutrition and health promotion**

Please read DiMaria-Ghalili & Amella (2005), ‘Nutrition in older adults’.

During recent decades, the concept of health promotion has become a legitimate part of health care because of the ageing of the post-war baby boom generation. As this population ages, the potential strain on health care systems will increase because the greatest use of health care services occurs during the last years of life. In older adults, there are many correctable health factors that can be assessed through screening protocols. Hypertension, cholesterol, hearing, vision, diabetes, and cancer screening are well integrated into health promotion programs; nutrition promotion programs are not as well integrated. Reluctance to develop health promotion programs for older adults exists because of a perception that they would not follow such plans or change their lifestyles. However, longitudinal studies have shown that health promotion activities extend the number of years of health in older people, although the relationship weakens in older age. Changes in diet and exercise patterns are most effective in the prevention of nutrition-related conditions when they are instituted early in life, but positive effects can occur at any age. If nutritional interventions are instituted early, a substantial reduction in health care expenditures may result from a decrease in the incidence or the delayed onset of these conditions. Changes in behaviours (reducing salt and fat intake) were positively associated with a belief that consuming a healthful diet would contribute to better health.

**TEXT QUESTIONS**

1 Are recent gains in life expectancy likely to be associated with increased years of disability?
2 What are the nutritional implications of the major age-related changes in the cardiovascular, renal and pulmonary systems?
3 What are the changes associated with a reduction in total energy expenditure due to a reduction in physical activity?
4 What changes occur in body composition during ageing and what implications do they have for increased risk of ill health?
5 What aspects of gastrointestinal function are most likely to reduce nutrient intake and/or availability in the elderly?
6 Why is fluid intake important in the elderly?
7 What are some reasons why elderly people tend to have low intakes of micronutrients?
8 What strategies are most appropriate for minimising the effects of osteoporosis in old age and how do they differ from those used at younger ages?
In what ways can ill-health compromise the nutritional status of an older person?

What are some of the mechanisms by which drugs influence nutritional status?

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Introduction

It is important to keep in mind some of the basic nutritional concepts that you have learnt in the previous unit (HSN201 Principles of Nutrition). It is recommended that you revise and make sure that you understand the following topics in this revision chapter.

Nutrient needs: summary

Nutrients are substances that are essential for growth and good health. Nutrients have three general functions in the body:

• they provide energy
• they form structural components of the body
• they regulate body functions.

Some nutrients such as proteins fulfil all three functions; whereas others such as vitamins have only one function. Macronutrients provide a source of energy to perform body functions. Dietary carbohydrates, proteins and lipids are all macronutrients and each provides the following amount of energy:

Carbohydrate = 16kJ/g
Protein = 17kJ/g
Lipid = 37kJ/g

Alcohol is also a source of energy in the diet, providing 29 kJ/g; however, it is not considered a nutrient as it has no required function. The macronutrients provide energy for building body compounds, performing muscular movements, promoting nerve transmission, and maintaining iron balance. Any portion of these macronutrients not used for energy is restructured for body tissue storage, either in the form of glycogen or fat. These storage forms can be used for fuel to use between meals or for use in synthesising other metabolic compounds needed in the body. If energy intake exceeds energy use, excess energy will be stored in the body leading to weight gain. If energy intake is insufficient to meet the energy needs of the body, this will result in weight loss.

To maintain body weight, the food energy consumed needs to approximate energy expenditure over time.

The three components of energy expenditure are:

• Basal metabolism: Basal Metabolic Requirement (BMR)*
• thermic effect of food
• physical activity.

* Basal Metabolic Rate (BMR) is the amount of energy expended by an individual at complete rest in a comfortable environment after fasting for 12 hours.
The largest component of energy expenditure is BMR. For adults, BMR is estimated to be 5.1–8.4 MJ/day. BMR accounts for 50–75% of total energy expenditure. A small portion of energy (6–10%) is expended by the thermic effect of foods or the process of converting food to energy. The remainder, physical activity, is highly variable. The level of energy expended depends on the individual’s body weight and the intensity and duration of physical activity. In addition, the various stages of growth increase children’s energy requirements.

**Macronutrients**

The primary function of dietary carbohydrates and lipids is to provide energy. Dietary protein also provides energy, but its primary role is to provide amino acids for synthesis of body protein and other essential compounds.

**Protein**

Protein is the fundamental structural material of every living cell in the body. In fact, the largest portion of the body, excluding water is made up of protein. Dietary protein provides amino acids that are used to synthesise body proteins, which build and maintain structural body tissues such as muscle, bone matrix, and connective tissue. Other non structural body proteins include serum proteins such as albumin, blood cell membranes, immune factors, enzymes and some hormones. All these tissues must be constantly repaired and replaced. The primary functions of protein are to repair worn-out, wasted, or damaged tissue and to build up new tissue. Thus during early life protein is needed for growth and development, whereas during the adult years protein maintains tissue health.

Proteins are found combined with other compounds e.g. nucleic acids (DNA and RNA), carbohydrates (glycoproteins or mucoproteins), lipid (lipoproteins) and metals (haemoglobin). The amount of dietary protein in the Australian diet provides 12–18% of energy. In diets that provide sufficient energy to maintain body weight, this level exceeds recommended levels of intake (15-25%) (NHMRC 2004).

**Amino acids**

Amino acids are the basic building materials for cells. All protein, whether in our bodies or in the food we eat is made up of amino acids. These amino acids are joined by peptide linkages to form specific proteins. When we eat protein foods, the protein e.g. casein in milk and cheese, albumin in egg white and gluten in wheat products, is broken down into its constituent amino acids during the digestive process. According to need, depending on the body’s overall metabolic ‘pool’ of amino acids, amino acids are then reassembled in the body in the specific order to form specific tissue proteins that are required by the body e.g. collagen in connective tissue, myosin in muscle tissue, haemoglobin in red blood cells, cell enzymes and insulin.

**Protein balance**

The term balance refers to the relative intake and output of substances in the body to maintain normal levels needed for health in various circumstances during the life cycle. We can apply this concept of balance to life-sustaining protein and the
nitrogen it supplies. The body’s tissue proteins are constantly being broken down, a process called catabolism, and then re-synthesised into tissue protein as needed, a process called anabolism. To maintain nitrogen balance, the nitrogen-containing amino may be removed by deamination, the amino acid converted to ammonia (NH$_3$) and the nitrogen excreted in the urine as urea. The remaining non-nitrogen residue of the amino acid can be used to make carbohydrates or fats or make new amino acids by attaching an NH$_2$ according to need. The rate of this protein and nitrogen turnover varies in different tissues, according to their degree of metabolic activity. This process involves a continuous reshaping and rebuilding, adjusting as needed to maintain overall protein balance within the body. Also, the body maintains an internal balance between tissue protein and plasma protein. In turn, these two body stores of protein are further balanced with dietary protein intake. With this finely balanced system, a metabolic ‘pool’ of amino acids from tissue protein and dietary protein is always available to meet construction needs. Therefore, as there is continual breakdown of amino acids, the body is continually excreting nitrogen in the urine.

**Nitrogen balance**

The body’s nitrogen balance indicates how well tissues are being maintained. The intake and use of dietary protein are measured by the amount of nitrogen intake in the food protein and the amount of nitrogen excreted in the urine. Total 24-hour urinary urea nitrogen excretion is used to measure nitrogen lost from the body. Nitrogen intake can be measured directly in the food eaten (by chemical measurement of duplicate portions of food eaten) or calculated from dietary records, using tables or a computerised dietary analysis program.

**Positive nitrogen balance**

Positive nitrogen balance exists when the body takes in more nitrogen than it excretes. This means that the body is storing nitrogen by building more tissue than it is breaking down. This situation occurs normally during periods of rapid growth, such as infancy, childhood and adolescence, and during pregnancy and lactation. It also occurs in persons who have been ill or malnourished and are being ‘built back up’ with increased nourishment. In such cases, protein is being stored to meet increased needs for tissue building and associated metabolic activity.

**Negative nitrogen balance**

A negative nitrogen balance exists when the body takes in less nitrogen than it excretes. This means that the body has an inadequate protein intake and is losing nitrogen by breaking down more tissue than it is building up. This situation occurs in states of malnutrition and illness. For example, negative nitrogen balance is seen not only in underdeveloped countries, but also where specific protein deficiency occurs, even though there is adequate energy from carbohydrates and fats. Failure to maintain nitrogen balance may not be apparent for some time, but will eventually cause loss of muscle tissue, impairment of body organs and functions and increased susceptibility to infections. In children, negative nitrogen balance will cause growth retardation.
Carbohydrate

Carbohydrate is available in a wide range of foods and is the least expensive and quickest source of energy. Except for lactose or milk sugar, starches and sugars are obtained primarily from plant foods.

The Dietary Guidelines recommend moderating sugar intake due to its association with dental caries in children and the association with high sugar soft drinks and obesity. From a practical point of view, a high intake of sugary foods is incompatible with the goal of eating a nutritious diet that maintains a body weight within the healthy weight range.

Dietary fibre

Dietary fibre consists of nondigestible plant food and lignin in which the plant matrix is largely intact. Epidemiologic research on dietary fibre has suggested that dietary fibre may be important in the prevention of constipation, diverticulosis and other gastrointestinal disturbances.

High intakes of dietary fibre from fruit, vegetables and certain cereal grains (low glycaemic index foods) lower blood glucose, insulin, and lipids in patients with non-insulin diabetes.

Lipids and fatty acids

Lipids are an important source of energy and provide fatty acids for the synthesis of many body compounds. Most dietary lipids are in the form of triglycerides. The characteristics of individual dietary fats are determined by which fatty acids are present. Fatty acids have been classified, based on chain length and structure, as saturated (no double bonds between carbon chains), monounsaturated (one double bond) and polyunsaturated (two or more double bonds). Polyunsaturated fatty acids (PUFAs) are further classified by the location of the double bonds in the carbon chain. The type of dietary fat in the diet has been implicated in the prevention and/or the development of cardiovascular disease. It has been demonstrated that saturated fat raises total cholesterol and LDL cholesterol (low density lipoprotein), both of which are established risk factors for cardiovascular disease. Whereas monounsaturated fats (predominately from olive) have the potential to lower cardiovascular disease, as do polyunsaturated fats. It is not clear if mono and/or polyunsaturated fats have a cholesterol lowering effect that is independent of the saturated fat content of the diet.

Fatty acids that are considered essential for humans are derived from linoleic acid (18 carbons, two double bonds) and alpha-linolenic acid (18 carbons, three double bonds). Linoleic acid is referred to as an omega-6 fatty acid and alpha-linolenic acid as an omega-3 fatty acid, indicating that the double bond occurs at the sixth and third carbons from (-CH₃) end of the fatty acid, respectively. Epidemiologic and clinical studies have documented an association between omega-3 fatty acids and the prevention and amelioration of many diseases, particularly heart disease. These protective effects are attributed to their omega-3 PUFA content, particularly eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). These PUFAs are incorporated into the cell membrane, where they influence membrane fluidity, receptor function, enzyme activity, cytokines, and eicosanoid production.
Cholesterol

Cholesterol is a fat-like substance that is found in foods of animal origin. It is synthesised in the human body and is found in many tissues, such as brain and nervous tissues. It is an important component of cell membranes. Cholesterol and its derivatives are also precursors of vitamin D, and various steroid hormones and bile acids. Elevated serum cholesterol is associated positively with the risk of coronary heart disease.

Lipoproteins

Lipids and cholesterol are not soluble in water and they can only move around in the blood stream as a component of lipoprotein molecules. These lipoprotein molecules are categorised as HDL (high density lipoprotein), LDL (low density lipoprotein) and VLDL (very low density lipoprotein). Cholesterol found in each of these lipoproteins is referred to as HDL-cholesterol (HDL-C), LDL-C and VLDL-C respectively. The sum of all these cholesterols in the lipoproteins is the total cholesterol. LDL-C is positively associated with cardiovascular disease, whereas HDL-C is inversely associated with cardiovascular disease i.e. the higher the HDL-C the lower the risk of cardiovascular disease.

Water

Approximately 60% of the adult body is water and water is essential for almost all body functions. It serves as a solvent, lubricant, and a medium for transporting nutrients to body cells and waste products for excretion. Water is essential in the regulation of body temperature and facilitation of chemical processes. For most individuals thirst mechanisms automatically ensure adequate fluid intake. Under circumstances of rapid water loss from the body or impairment of the thirst sensation, fluid levels become depleted and this leads to dehydration, which if continued for a few days could result in death.

Micronutrients

The body uses macronutrients carbohydrates, fats and protein as an energy source, to rebuild tissue and to perform a variety of physiological and metabolic processes which maintain life. To maintain life and health these multiple physiological tasks must proceed in a highly organised and orderly fashion, without which metabolic chaos, illness and death would occur. Such order requires specific control agents. The remaining micronutrients, which include vitamins and minerals, allow this. Vitamins and minerals work in harmony with hormones and have specific partnerships with key enzymes to enable the initiation and control of metabolic processes.

Minerals

Minerals are inorganic substances that are essential for the diverse functions of the human body. Minerals are critical to hundreds of body reactions in cellular processes, water balance, and regulation of the nervous system. They are constituents of body compounds such as bone, haemoglobin, and enzymes and regulators of most body functions.
Vitamins
Vitamins are essential organic substances that the body is unable to manufacture and therefore must consume in the diet. These essential regulators of body metabolism occur in food in very small quantities. Vitamins are either water soluble (all B vitamins, C, folate) or fat soluble vitamin (A, D, E, K). Vitamins have diverse chemical structures and may participate in one, several or even hundreds of chemical reactions within the body. The absence of any vitamin can lead to lowered tissue levels and eventually vitamin specific deficiency signs and symptoms can develop, and in extreme circumstances can lead to death.

Vitamins and minerals in modern diets
It is now apparent that nutrients seldom act alone in the body. They are integrally related to one another. Macronutrients cannot be used without the assistance of micronutrients, and both categories interact to make body tissues, promote use of energy and regulate body functions. Some dietary components such as phytochemicals, carotenoids, carnitine, and glutamine have relevance to human health in their potential to decrease risk of or prevent chronic diseases or developmental abnormalities.

RECOMMENDED REVISION SUMMARY ACTIVITIES

1. Draw up a table listing all the macronutrients and indicate: the components, the function and the dietary source.

2. Draw up a table listing all the minerals and indicate: the major function and the significant food sources in the Australian diet.

3. Draw up a table listing all the vitamins and indicate the major function and the significant food sources in the Australian diet.

   NB. Put in additional column ‘groups most at risk’ and fill this column in as you progress through the unit.

REVISION QUESTIONS

1. What is the major component of energy expenditure in an adult?

2. List the three components for energy expenditure for adults? How does the situation differ in childhood?

3. List the macronutrients in order of energy density (the most energy produced per gram of macronutrient).

4. What is the estimated per cent energy from fat in the Australian diet (refer to Appendix 4 on DSO).

5. Which macronutrient provides most of the energy in the Australian diet (refer to Appendix 4 on DSO).

6. Where is cholesterol found in the body and what function does it have?

7. Where is cholesterol found in the Australian diet?

8. Name three substances that have anti oxidant properties and explain the role of antioxidants in health.
9 Explain calcium balance and indicate under which circumstances a person would be in positive calcium balance, negative calcium balance or in calcium balance. (Hint: refer to the explanation of nitrogen balance and revise calcium metabolism.)

References


List of appendices on DSO

PREPARED BY THE UNIT TEAM

The following appendixes are available on DSO

Appendix 1.1

*Birth to 36 months*
*Boys, length-for-age and weight-for-age percentiles*

Appendix 1.2

*Birth to 36 months*
*Girls, length-for-age and weight-for-age percentiles*

Appendix 1.3

*2 to 20 years: Boys*
*Stature-for-age and weight-for-age percentiles*

Appendix 1.4

*2 to 20 years: Girls*
*Stature-for-age and weight-for-age percentiles*

Appendix 1.5

*Body mass index-for-age percentiles:*
*Boys, 2 to 20 years*

Appendix 1.6

*Body mass index-for-age percentiles:*
*Girls, 2 to 20 years*

The above six documents are from:

Appendix 2

*Nutrient Reference Values for Australia and New Zealand—executive summary*


Appendix 3

*National Nutrition Survey 1995—summary of findings*


Appendix 4

*National Nutrition Survey 1995—tables*


Appendix 5

2007 Australian National Children’s Nutrition and Physical Activity Survey: Main findings