Nutrition in Aboriginal and Torres Strait Islander Peoples

An Information Paper

NHMRC
National Health and Medical Research Council
Nutrition in Aboriginal and Torres Strait Islander Peoples

An Information Paper

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Food is essential for everybody. It is part of our daily lives and forms an integral part of social, cultural and religious activities of various types in different cultures around the world.

The food we eat has a significant impact on our physical well-being. Dietary energy is needed for growth and for our daily work, play and other activities. Nutrients such as proteins, fats and carbohydrates are required for the body’s normal metabolic functions and during recovery from illness and injury. Micronutrients, such as iron and zinc, have special functions and their deficiencies result in important clinical disorders which can include anaemia and growth restriction. Vitamins are also required for a normal, healthy life.

Food and nutrition must be viewed in the context of the food supply, its availability, freshness, cleanliness and year-round variety. Social, economic and cultural factors also have strong impacts on the foods that people eat. In rapidly transitional societies including Aboriginal and Torres Strait Islander communities, these factors are having very strong influences on food consumption patterns which, in turn, affect health.

Knowledge and attitudes to foods and nutrition also have a crucial impact on food choices.

The environment in which people live, their ability to safely store and refrigerate foods and protect them from contamination in their food stores and at home, also influences the quality and variety of food consumption patterns. Infections, particularly gastroenteritis, have an impact on how effectively we metabolise and utilise our foodstuffs and can therefore have a significant impact on nutrition and, for infants and children, growth.

Aboriginal and Torres Strait Islander peoples lived for many thousands of years in harmony with their environment and lived off what it provided for them in very diverse parts of the country. They lived in harmony with sometimes harsh and hostile environments and survived through times of scarcity until food was found elsewhere or became available again. They were the world’s largest and most successful group of hunter-gatherers until colonisation by Europeans changed their lifestyles and their patterns of food consumption.

This changed lifestyle has had and continues to have very powerful influences on their health and well-being. It is also largely responsible for some of the chronic diseases like cardiovascular disease, high blood pressure and Type 2 diabetes which are now so common in Aboriginal and Torres Strait Island peoples. This is complicated by socio-economic and environmental factors including underemployment, poor housing, overcrowding, inadequate infrastructure services and high rates of infectious and parasitic diseases.

These are highly important issues for Aboriginal and Torres Strait Islander peoples. They are discussed in this guide in such a way that will be useful for Indigenous and non-Indigenous people. We hope this information will be put to practical use by Aboriginal and Torres Strait Island Health Workers in their day-to-day work. We trust this document meets its main objective of helping to improve the nutrition and health of Aboriginal and Torres Strait Islander communities.
Aboriginal and Torres Strait Islander people have always relied heavily on bush tucker to survive. Not only did our great untouched land provide our ancestors with the right foods to survive, it also offered the same services which we would more recently obtain from a local shopping centre, hospital or chemist.

Australia’s vast bushland ranges from arid deserts, to the magnificent tropical rainforests. It is surrounded by a coastal barrier which also offers a wide selection of nutritious bush tucker.

A traditional diet includes many different types of fresh plant and animal foods. Our diet was especially high in protein, complex carbohydrates, vitamins and minerals. The animals we hunted were low in fat and high in ‘soft’ fats, which helped to protect our people from heart disease.

Medicines that are used around the world today, for healing and treatment, were used for centuries by traditional Aboriginals.

Our spiritual knowledge of the land is a tool that has balanced our diet for many years.

Mundullullu (Joan) Koops
SUMMARY

Aboriginal and Torres Strait Islander peoples continue to suffer a much greater burden of ill-health than other Australians. The health disadvantage begins in early life, continues through the life cycle and is exacerbated by low socio-economic status. Although birthweights in Aboriginal and Torres Strait Islander peoples are increasing, low birthweights are still more likely than in the general population. As well, infant and child malnutrition contribute to growth restriction and predispose children to infectious disease.

The causes of mortality in the Aboriginal and Torres Strait Islander population have changed from acute infections to chronic non-communicable diseases, such as coronary heart disease, diabetes, high blood pressure and renal (kidney) disease. Holistic approaches need to be further developed and implemented to address current patterns of ill-health and mortality. Appropriate health promotion and prevention programs are also necessary to ensure that present trends do not continue into the future.

This report presents background information about nutrition and nutrition-related disease in Aboriginal and Torres Strait Islander peoples. This information is specifically targeted to practitioners working to improve the nutritional health of Aboriginal and Torres Strait Islander peoples. The report may be useful as reference material in both practice and teaching.

Section I of the report gives a summary of the current situation in Aboriginal and Torres Strait Islander health and nutrition through presentation of demographic and health data. Changes to diet since European colonisation and other factors that have an impact on Aboriginal and Torres Strait Islander nutrition are also considered. Reviews of health promotion initiatives and their findings are discussed and principles of good practice outlined.

Sections II and III deal with the effects of poor nutrition on the Aboriginal and Torres Strait Islander population, describing the resultant conditions and the extent to which they affect that population. Approaches to prevention and management are suggested. Section II outlines the importance of proper nutrition in pregnancy, infancy and childhood and discusses assessment of childhood growth and iron-deficiency anaemia. Section III addresses those nutrition-related diseases of particular relevance to the Aboriginal and Torres Strait Islander population. These are obesity, Type 2 diabetes, cardiovascular disease (including high blood pressure), renal disease, and dental health problems.

Nutrition and Aboriginal and Torres Strait Islander health

The importance of good nutrition to health has long been recognised. There is increasing evidence that nutrition prenatally and in the first months/years of life may play an important role in the development of later disease as well as leading to slower mental and physical growth. In particular, under-nutrition during fetal growth and over-nutrition during childhood have been linked to obesity, diabetes and cardiovascular disease in adults.
As well, Syndrome X (or the insulin resistance syndrome) is particularly evident in Indigenous populations exposed to rapid lifestyle change in the 20th century, including Aboriginal Australians. This syndrome becomes evident after the transition from a traditional to an ‘urbanised’ or ‘westernised’ lifestyle. It includes high rates of obesity, Type 2 diabetes and cardiovascular and renal disease.

To understand the present state of Aboriginal and Torres Strait Islander nutritional health it is necessary to set it in historical perspective. The limited information about the diet and nutritional health of these people before European colonisation suggests slim, strong people living in harmony with their environment. With the transition from a traditional hunter-gather lifestyle to a settled westernised existence, Aboriginal and Torres Strait Islander diet has changed from a varied, nutrient-dense diet to an energy-dense diet, high in fat and refined sugars. This has had a serious negative impact on the health and well-being of these peoples.

An adequate supply of healthy and affordable food is important in maintaining good nutrition but this is not always available to Aboriginal and Torres Strait Islander peoples. There are socio-economic, geographical, environmental and social factors that may influence availability and cost of food.

• Socio-economic status— A link exists between low socio-economic status and higher rates of various diseases in adults. As most Aboriginals and Torres Strait Islanders fall into the lowest socio-economic group, it must be inferred that at least a proportion of their higher disease rates is attributable to their low socio-economic status.

• Geographical isolation— There is still an extremely limited range of foods stocked in remote community foodstores, relative to larger rural towns and urban centres. Costs of commodities vary across States/Territories and are higher in more remote communities.

• Environmental factors— Adequate housing, access to clean water, and the removal of human waste are important factors in achieving and maintaining good health. The absence of any one of these factors can result in a variety of infectious and parasitic diseases. Aboriginal and Torres Strait Islander housing in rural areas has fewer services than in the capital cities.

• Social factors— High rates of substance misuse have been reported in the Aboriginal and Torres Strait Islander population. In addition to the direct effect of alcohol and tobacco on nutrient intake, the use and abuse of substances can direct substantial amounts of money away from the purchase of food and other necessities.

• Government regulations— Regulations relating to quarantine, fisheries and National Parks can affect access of Aboriginal and Torres Strait Islander peoples to their traditional foods, and in some places to gardening initiatives.

As outlined above, nutritional problems in Aboriginal and Torres Strait Islander communities may be very complex and are not due solely to lack of knowledge about contemporary foods. Such problems will not be alleviated by nutrition education.
alone but require social and political action within the framework of community development programs. Previous policies aimed at improving Aboriginal and Torres Strait Islander nutrition were well intentioned but often inappropriate. Recent reviews of programs have shown that it is important that programs address community priorities. Communities need to be involved in all aspects of development, implementation and evaluation of programs. As Aboriginal and Torres Strait Islander peoples live in urban, rural and remote locations, programs need to be developed that are accessible, culturally appropriate and relevant to people in each of these areas.

Several recent reports have noted that there is still a lack of well-evaluated nutrition/health programs for Aboriginal and Torres Strait Islander peoples. Without evaluation results, limited information is available to assist health service providers to make informed decisions about effective nutrition projects for their community.

**Maternal and child health**

Proper nutrition is important at all stages of development of the fetus, infant and child. Malnutrition of the mother during pregnancy can lead to health consequences for the child in adulthood. Assessment of growth during infancy and childhood aims to determine whether nutrition is adequate.

Low birthweight may lead to death early in life or ill-health in infancy and childhood. Low birthweight may result from a short gestation period (ie preterm delivery), intrauterine growth restriction or both. Babies with intrauterine growth restriction may have under-developed organs and consequent metabolic changes that could compromise their future health, making them more prone to conditions including Type 2 diabetes and cardiovascular disease.

Nutritional determinants of intrauterine growth restriction include low maternal dietary energy intake, maternal malnutrition, inadequate weight gain during pregnancy and low pre-pregnancy weight. Other factors include maternal age, socio-economic status, cigarette smoking and alcohol consumption. Low birthweight due to preterm delivery is related primarily to reproductive history.

Although there is currently a trend towards higher birthweights in Aboriginal babies, the proportion of low birthweight among babies born to Aboriginal mothers is about twice that of babies born to non-Aboriginal mothers. There is still some discussion about the degree to which this may be due to preterm delivery or intrauterine growth restriction.

As the risk factors and health outcomes for preterm delivery and intrauterine growth restriction differ, it is important to determine the causes of low birthweight so that appropriate preventive strategies can be developed and implemented. Poor utilisation of antenatal services by Aboriginal women in urban and rural areas has been found to be a very important cause of poor pregnancy outcomes. This results from a combination of poorer health education and cultural barriers. Expanded Aboriginal medical and outreach services and more specialist obstetricians and gynaecologists working in regional and remote areas could help improve services for Aboriginal and...
Nutrition in Aboriginal and Torres Strait Islander Peoples

Existing programs have been shown to have a positive effect on birthweight.

Adequate nutrition for infants and children is essential to their normal growth. Initially an infant will be breast-fed and/or bottle-fed. The introduction of adequate quantities of solids at four to six months to complement milk feeding is important.

- Breast-feeding is associated with reduced infant and child mortality and is increasingly recognised as fundamental for long-term health.
- Bottle-feeding is appropriate if breast-feeding is not possible or feasible or may be hazardous for the infant (e.g., in cases of maternal illness, separation, or substance misuse).
- Introduction of solid foods at four to six months is particularly important in groups where the nutritional status of the mother may be marginal. Studies have shown that if children are fed solids in adequate quantities at this age they grow normally even in microbiologically contaminated circumstances.

Childhood growth results from the combined effects of genetic and environmental influences. If the environmental factors that affect growth are persistently negative (e.g., because of living in overcrowded, unhygienic conditions, with repeated infections and poor nutrition), growth performance is likely to be chronically or persistently restricted. This may lead to ‘failure-to-thrive’ or clinically evident under-nutrition or malnutrition, sometimes with important nutritional complications such as specific nutrient deficiencies and anaemia. Under-nutrition in infancy and early childhood, often preceded by low birthweight, can lead to permanent growth restriction and stunting.

Access to curative health care can clearly improve ill-health arising from poor nutrition. However, improved health care can reduce childhood mortality without improvements in nutritional status. Therefore, approaches to eradicating malnutrition and its consequences need to be multi-faceted and include improved physical infrastructure, better food supplies, health and nutrition education and access to culturally appropriate clinical care.

Iron-deficiency anaemia is the most common nutrient deficiency in the world and the available evidence suggests that it is more common in Aboriginal and Torres Strait Islander peoples than in the general Australian population. Iron-deficiency anaemia in infants and children is associated with lower scores on tests of development, learning, and school achievement. In adults, diminished levels of serum iron have been linked to significant reductions in work productivity and mental performance.

In infants and children, rapid growth imposes large iron needs and inappropriate nutrition is likely to play a major role in iron deficiency. In women of child-bearing age, iron loss resulting from pregnancy or heavy menstrual periods may, on a long-term basis, not be matched by dietary iron. In men and in post-menopausal women the major cause of iron deficiency is pathological bleeding. Most commonly, this occurs from the gastrointestinal tract (e.g., peptic ulcers, gastritis, haemorrhoids).
Nutrition-related conditions

Aboriginal and Torres Strait Islander peoples have high rates of ill-health and/or death from Type 2 diabetes and cardiovascular disease, for which obesity is a risk factor. They also have high rates of renal disease and dental health problems.

Obesity increases the risk of cardiovascular disease and diabetes. Rates of obesity are higher in the Aboriginal and Torres Strait Islander population than in the total Australian population.

Obesity can be prevented and managed through a healthy lifestyle (i.e., balanced meals, regular physical activity, and avoidance or reduction of alcohol consumption and/or cigarette smoking). Such approaches also minimise the risks of related disorders such as Type 2 diabetes and cardiovascular disease. A focus on primary prevention should target children and adolescents.

Type 2 diabetes is a major cause of ill-health and death among Aboriginal and Torres Strait Islander peoples. Onset of Type 2 diabetes occurs at a far earlier age than in the general population, and a high prevalence of Type 2 diabetes and its risk factors in Aboriginal and Torres Strait Islander children and adolescents has been documented.

The modification of diet, to reduce dietary intake of fat, refined carbohydrate and total energy, and increased physical activity can help to correct abnormal metabolism associated with diabetes. Therefore, lifestyle modifications should be the base for health promotion programs. Young people in particular need to be targeted for primary prevention of risk factors for diabetes.

Cardiovascular disease is the leading cause of death among both Aboriginal and Torres Strait Islander peoples as well as among non-Indigenous Australians. Coronary heart disease is the major cardiovascular cause of death among Indigenous people. Mortality from stroke in the Indigenous population is about double that in the non-Indigenous population. The self-reported prevalence of high blood pressure in Indigenous people aged 45 to 54 years is about twice that in the non-Indigenous population. Rates of premature death from heart disease are very high among Indigenous people, with men and women dying in their 20s, 30s and 40s. In the non-Indigenous population, most cardiovascular disease deaths are in those aged over 60 years.

The major risk factors for cardiovascular disease are high blood cholesterol, high blood pressure, and cigarette smoking. More recently, among Aboriginal and Torres Strait Islander peoples, it has become apparent that diabetes, high triglycerides, and obesity also play an important role.

Renal disease has become recognised as an important and increasingly prevalent and severe disease in Australian Aboriginals, particularly in association with diabetes, cardiovascular disease (in particular high blood pressure) and infections. Prevalence is now so high that it is difficult to meet the needs for renal dialysis (and in some instances transplantation) in Aboriginal patients with end-stage renal disease.

Lifestyle changes to prevent renal disease and improve outcomes include dietary measures to reduce high blood pressure, help prevent diabetes, and control weight.
Dental health may sometimes be overlooked among the many serious health problems experienced by Aboriginal and Torres Strait Islander peoples. The incidence of decayed, missing and filled teeth in Aboriginal and Torres Strait Islander children is almost double that of non-Indigenous children. There is limited information about the dental health of Aboriginal and Torres Strait Islander adults. The loss of all natural teeth, which tends to be a result of appropriate treatment failure, has been found to be more common among low-income earners. Many Indigenous people were included in this group.

It is clear that poor nutrition has a great impact on the health of Aboriginal and Torres Strait Islanders. Nutrition needs to be addressed across the life-span, with education and programs targeting women in pregnancy, parents, adolescents, children and people at risk of lifestyle-related ill-health. Priorities for prevention of nutrition-related disease in the Aboriginal and Torres Strait Islander population include:

- improving access to good quality affordable fresh fruit and vegetables;
- increasing levels of physical activity;
- improving maternal and child health; and
- reducing the prevalence of cigarette smoking and the hazardous consumption of alcohol.

Education programs need to be culturally relevant to the particular community and its setting and need to be modified for individual communities and different regions around Australia.
Aboriginal and Torres Strait Islander peoples continue to suffer a much greater burden of ill-health than other Australians. The health disadvantage begins in early life and continues through the life cycle (ABS & AIHW 1999). Although birthweights are increasing, many infants are still being born underweight. Infant and child malnutrition contribute to growth restriction and predispose children to infectious disease.

The main causes of death in the Aboriginal and Torres Strait Islander population have changed from acute infections to chronic non-communicable diseases, such as coronary heart disease, diabetes, high blood pressure and renal disease. Holistic approaches need to be further developed, resourced and implemented to address current patterns of ill-health and death. Appropriate health promotion and prevention programs are also necessary to ensure that present trends do not continue into the future, given the current health disadvantages experienced by mothers and children (HoR Standing Committee on Family and Community Affairs 1999).

Data availability

Data on Aboriginal and Torres Strait Islander peoples and their health is limited and varies across the country. Assessment of trends requires comparability over time and in the quality and availability of the data. With respect to data about Aboriginal and Torres Strait Islander peoples, this means a requirement for highly accurate and stable identification of Aboriginal and Torres Strait Islander people in administrative data collections; stable population estimates; and repeated surveys providing accurate data for all sections of the Indigenous population (ABS & AIHW 1999).

Current data collection includes the following.

• All States and Territories now collect information about Indigenous status in their birth and death notification forms, perinatal data collection forms, hospital separation data collections and cancer registration forms. The quality of the identification of Indigenous people in these and other collections is not always adequate to allow for reporting.

• Survey collections of relevance to the health and nutrition of Aboriginal and Torres Strait Islander peoples include the five-yearly Census of Population and Housing, the 1994 National Aboriginal and Torres Strait Islander Survey (NATSI) and the 1995 National Health Survey (NHS).

High and stable accuracy of Indigenous identification in administrative collections has not yet been achieved. Many collections and/or jurisdictions do not have adequate identification to allow for national reporting. The Australian Bureau of Statistics (ABS) currently publishes detailed death statistics for Indigenous people only for Western Australia, South Australia and the Northern Territory. The stability of Indigenous population estimates is also uncertain because adequate data on births, deaths and migration are not available.
Most survey-based information about Indigenous people has only been collected on one occasion. The value of surveys depends on the representativeness of Indigenous people sampled as well as the quality of the data collected. The following concerns have been raised about the Australian surveys relevant to Indigenous health (ABS & AIHW 1999):

- the number of Indigenous people included in standard national surveys is usually insufficient to provide reliable estimates due to the relatively small size of the Indigenous population;
- self-reported data relating to health recorded from individual and household surveys vary in reliability; and
- data in the 1995 NHS may be misleading or biased because the sample is not truly representative.¹

There are a number of initiatives in progress to improve data collection, across the area of Indigenous affairs, which may have some impact on health data, including (HoR Standing Committee on Family and Community Affairs 1999):

- development and implementation of a National Aboriginal and Torres Strait Islander Health Information Plan;
- agreement by Health Ministers on performance indicators for Aboriginal and Torres Strait Islander health;
- development of a draft National Indigenous Housing Agreement; and
- collaborative work under way to improve the identification of Indigenous people in administrative datasets.

Research

There is a lack of research aimed at identifying nutrition interventions that are effective in the Aboriginal and Torres Strait Islander population. Possible explanations for this include:

- the perception that changing behaviour in regard to risk factors is difficult and rarely successful;
- the notion that few benefits can be achieved through intervention (which may lead to too little consideration of the time, resources and action required for prevention programs by fund holders); and/or
- the fact that the lengthy time period between the introduction of an intervention and the development of results may influence fund holders to assign priority to short interventions with short-term results (Butlin et al, unpublished).

¹ The ABS has excluded data for both Indigenous and non-Indigenous people from remote areas in the publication of results in the NHS. Data from the 1995 NHS included in this publication therefore refer only to people living in non-remote areas.
It should be noted that these criticisms can also be made about many areas of nutrition intervention in the non-Indigenous population.

There is concern among Aboriginal and Torres Strait Islander peoples about research in their communities. The feeling is that there is more than enough research that identifies problems when what is required is research that assists in solving problems identified by Aboriginal and Torres Strait Islander people (NHMRC 1997a). There is a need for Aboriginal and Torres Strait Islander people to undertake their own research and to develop methods to present data so that individuals and communities can use the information themselves (Weeramanthri & Plummer 1994; McMasters 1996; Houston & Legge 1992). Despite the extent of research undertaken to date, there is also a need for research into health problems in urban Indigenous people as most research has been conducted in remote communities.

The development of guidelines on ethical matters by the NHMRC (1995) and the establishment of Aboriginal Research Ethics Committees in the Northern Territory, South Australia, Victoria, New South Wales and Western Australia have been helpful. However, further effort is needed to ensure that the guidelines and processes are adhered to (NHMRC 1997a). Guidelines have also been produced by the National Australian Indigenous Health Organisation (NAIHO) (1987) and the Aboriginal Health and Medical Research Council in New South Wales (AH & M RC 1999; 1997).

Research and publications of statistical data on Aboriginal and Torres Strait Islander health should meet the following principles (AH & M RC 1999):

- research proposals must advance scientific knowledge so as to result in demonstrated additional benefit to communities;
- there should be community control over all aspects of the proposed research, including design, data, data interpretation and publication of findings;
- research should be conducted in a culturally sensitive manner;
- communities and organisations should be reimbursed for all costs arising from their participation in the research process;
- communities and organisations should be able to benefit from the transfer of skills and knowledge arising from the research project; and
- consent should be obtained from the community with consent forms approved before the commencement of any research.

Health promotion programs

Indigenous people live in urban, rural and remote locations around the country. Health promotion programs need to be accessible and relevant to people in all these areas. They should address community priorities and engage communities in all aspects of development, implementation and evaluation. A whole of government
approach is required to address the problem with greater communication within and between State/Territory and Commonwealth governments.

An appropriate model for Aboriginal and Torres Strait Island health promotion is one that (NHMRC 1997a):

- acknowledges and addresses the effects of their history on the health and well-being of each community;
- is based on strengthening Aboriginal and Torres Strait Island cultures and identity;
- acknowledges cultural influences on attitudes to health and illness;
- uses educational and environmental strategies that are suited to the setting in which the program is to take place;
- operates and is controlled from the community level, involving community members in each step of the process; and
- recognises the relationship between socio-economic disadvantage and health status.

The limited infrastructure support for Indigenous health promotion has meant that evaluation of individual interventions has been limited. There is no nationally agreed framework or mechanism to allow evaluation of the levels of input to promoting Indigenous health. Standards have not been developed to enable measurement of the quality of interventions (NHMRC 1997a).

**Purpose and structure of the report**

Within this context, this report presents background information about nutrition and nutrition-related disease in Aboriginal and Torres Strait Islander peoples. This information is specifically targeted to practitioners working directly to improve the nutritional health of Aboriginal and Torres Strait Islander people. The report may be useful as reference material in both practice and teaching.

The report aims specifically to:

- promote greater understanding of the importance of food and nutrition in achieving and maintaining good health;
- outline the relationship between nutrition and the health of the Aboriginal and Torres Strait Islander peoples; and
- provide examples of approaches that have been trialed and/or implemented to improve the nutritional health of Aboriginal and Torres Strait Islander peoples.

Although some programs have been formally evaluated, most programs and approaches are not evaluated. Therefore programs cited in this report should be considered only as an indication of the types of initiatives undertaken in Australia.
Section I of the report gives a summary of the current situation through:

- outlining the role of nutrition in health (Chapter 1);
- providing an overview of the demography and health status of the Australian Indigenous population (Chapter 2);
- outlining the main features of traditional, transitional and contemporary diets of Aboriginal and Torres Strait Islander peoples and discussing their relative impact on health (Chapter 3);
- discussing factors affecting nutrition and the impact these may have on the Indigenous population (Chapter 4);
- discussing the interrelationship between hygiene and infection (Chapter 5); and
- detailing approaches to the promotion of nutritional health in the Indigenous population and summarising reviews of these approaches (Chapter 6).

Section II outlines the importance of proper nutrition in pregnancy (Chapter 7) and in infancy and childhood (Chapter 8). The assessment of childhood growth (Chapter 9) and iron-deficiency anaemia (Chapter 10) are also discussed.

Section III discusses those nutrition-related diseases of particular relevance to the Indigenous population. These are obesity (Chapter 11); Type 2 diabetes (Chapter 12); cardiovascular disease (Chapter 13); renal disease (Chapter 14); and dental health problems (Chapter 15).

A guide for Aboriginal and Torres Strait Islander health workers has also been developed to assist in improving nutritional health in their communities. The guide includes information on diet and how it can affect the health of a community. It discusses health problems of particular relevance to Aboriginal and Torres Strait Islander communities and gives examples of health promotion programs that have been used in Australia to try to improve these problems in some communities. The development of local resources in communities is also encouraged.
SECTION I

NUTRITION AND ABORIGINAL AND TORRES STRAIT ISLANDER HEALTH
FOOD, NUTRITION AND HEALTH

The major causes of death, illness and disability in Australia that have diet as a risk factor include:

- cardiovascular disease (coronary heart disease, stroke, high blood pressure, atherosclerosis);
- Type 2 diabetes;
- some forms of cancer (stomach, colon, rectal, breast and endometrial);
- obesity;
- gall bladder disease;
- gastrointestinal diverticular disease and constipation;
- dental caries; and
- iron-deficiency anaemia.

A problem that is clearly increasing is renal disease, which has some dietary links, such as obesity in adulthood. Type 2 diabetes and high blood pressure are also risk factors for renal disease.

The causes of nutrition-related morbidity and mortality in Aboriginal and Torres Strait Islander peoples differ from those of the non-Indigenous population. Major causes of death, illness and disability in the Indigenous population that have diet as a risk factor include (ABS & AIHW 1999):

- cardiovascular disease;
- Type 2 diabetes; and
- renal disease.

Clinical observations and research have demonstrated that dietary deficiencies of single, identifiable nutrients can cause certain diseases. However, research into the relationships between dietary excesses and imbalances and the development of chronic disease can rarely prove a direct causal link. Instead, investigators must piece together information from different sources. Nevertheless, there is a substantial quantity of current animal, laboratory, clinical, and epidemiological evidence that associates dietary excesses and imbalances with chronic disease (USD Department of Health and Human Services 1988).

Diet-related diseases are caused by combinations and interactions of environmental, behavioural, biological, social and hereditary factors. Specific lifestyle factors
associated with the development of many diet-related diseases include cigarette smoking, physical inactivity and stress. Some important conditions linked to dietary causes are shown in Figure 1.1.

**Figure 1.1 Conditions linked to diet**

![Diagram showing conditions linked to diet]({})

Source: Adapted from James (1985).

Several reports have comprehensively reviewed the most important scientific evidence in support of current nutrition policy to reduce the chances of developing chronic degenerative diseases (English 1987; US Department of Health and Human Services 1988; NHMRC 1992a).

The Dietary Guidelines for Australians (NHMRC 1992a) present the best consensus of scientific evidence as key messages to the general Australian population about healthy food choices. The Guidelines are based on current scientific knowledge about the relationships between diet and disease, nutrients available in the Australian food supply, and the profile of morbidity and mortality in this country. Guidelines specific to children and adolescents (NHMRC 1995) and to older Australians (NHMRC 1999) have also been developed. Recommendations from the Guidelines are given in Appendix 3.
1.1 Quantifying the relationship between diet and disease

In 1989–90, coronary heart disease was the leading diet-related disease in Australia in terms of potential years of life lost. Dental caries cost the Australian community the most, in terms of direct and indirect health expenses. Other major diet-related diseases in Australia are shown in Table 1.1.

Table 1.1 Top six diet-related diseases in Australia 1989–1990

<table>
<thead>
<tr>
<th>Ranked by:</th>
<th>Potential years of life lost to age 65</th>
<th>Total costs</th>
<th>Health-care costs</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary heart disease</td>
<td>Dental caries</td>
<td>Dental caries</td>
<td></td>
</tr>
<tr>
<td>Certain neoplasms</td>
<td>Coronary heart disease</td>
<td>High blood pressure</td>
<td></td>
</tr>
<tr>
<td>Stroke</td>
<td>High blood pressure</td>
<td>Coronary heart disease</td>
<td></td>
</tr>
<tr>
<td>Type 2 diabetes</td>
<td>Stroke</td>
<td>Stroke</td>
<td></td>
</tr>
<tr>
<td>Gall bladder disease</td>
<td>Type 2 diabetes</td>
<td>Type 2 diabetes</td>
<td></td>
</tr>
<tr>
<td>High blood pressure</td>
<td>Certain neoplasms</td>
<td>Certain neoplasms</td>
<td></td>
</tr>
</tbody>
</table>


A statistic commonly cited is that 56.6 per cent of all deaths in Australia in 1983 were diet related (English 1987). This should not be interpreted as meaning that 56 per cent of all deaths are directly attributable to diet but simply that diet is an important risk factor for this proportion of all deaths (Crowley et al 1992). The conditions generating the most deaths are not necessarily those that generate the greatest costs. Given the lack of hard quantifiable evidence, a range of estimates has been given (Crowley et al 1992).

The direct health-care costs attributable to diet in Australia in 1989–90 were estimated at between $1,241 and $2,851 million (Crowley et al 1992). Total health-care costs attributable to diet in the same period were estimated to be between $2,479 and $5,190 million, which was slightly lower than the estimate of $6,000 million in 1988, which was extrapolated from US data (Health Targets and Implementation Committee 1988).

Describing the burden of disease by calculating the years of potential life lost does not allow for the fact that some conditions reduce the quality of life, but do not necessarily lead to death. The reduction in quality of life has been combined with mortality information to derive an index called the ‘disability-adjusted life year’ (DALY) (Mathers et al 1999). Evaluating the burden of disease in Australia using DALYs leads to a different ranking of important conditions than that which results when only mortality is considered (Table 1.2).
### Table 1.2 The 15 leading causes of burden of disease and injury in Australia, 1996

<table>
<thead>
<tr>
<th>Per cent of total burden</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1</strong> Coronary heart disease</td>
</tr>
<tr>
<td><strong>2</strong> Stroke</td>
</tr>
<tr>
<td><strong>3</strong> Chronic obstructive pulmonary disease</td>
</tr>
<tr>
<td><strong>4</strong> Depression</td>
</tr>
<tr>
<td><strong>5</strong> Lung cancer</td>
</tr>
<tr>
<td><strong>6</strong> Dementia</td>
</tr>
<tr>
<td><strong>7</strong> Diabetes</td>
</tr>
<tr>
<td><strong>8</strong> Colorectal cancer</td>
</tr>
<tr>
<td><strong>9</strong> Asthma</td>
</tr>
<tr>
<td><strong>10</strong> Osteoarthritis</td>
</tr>
<tr>
<td><strong>11</strong> Suicide and self-inflicted injuries</td>
</tr>
<tr>
<td><strong>12</strong> Road traffic accidents</td>
</tr>
<tr>
<td><strong>13</strong> Breast cancer</td>
</tr>
<tr>
<td><strong>14</strong> Hearing loss</td>
</tr>
<tr>
<td><strong>15</strong> Alcohol dependence and harmful use</td>
</tr>
</tbody>
</table>


The proportion of the burden of disease that can be attributed to various risk factors has been calculated (Table 1.3). With all calculations like this, the interactions between the risk factors mean that the totals are more than 100 per cent. To date, these calculations have not been done separately for Aboriginal and Torres Strait Islander peoples.

### Table 1.3 Burden of disease attributable to 10 major risk factors, Australia, 1996

<table>
<thead>
<tr>
<th>Per cent of DALYs</th>
<th>Males</th>
<th>Females</th>
<th>Persons</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tobacco</td>
<td>12.1</td>
<td>6.8</td>
<td>9.7</td>
</tr>
<tr>
<td>Physical inactivity</td>
<td>6.0</td>
<td>7.5</td>
<td>6.7</td>
</tr>
<tr>
<td>High blood pressure</td>
<td>5.1</td>
<td>5.8</td>
<td>5.4</td>
</tr>
<tr>
<td>Alcohol harm</td>
<td>6.6</td>
<td>3.1</td>
<td>4.9</td>
</tr>
<tr>
<td>Alcohol benefit</td>
<td>-2.4</td>
<td>-3.2</td>
<td>-2.8</td>
</tr>
<tr>
<td>Obesity</td>
<td>4.3</td>
<td>4.3</td>
<td>4.3</td>
</tr>
<tr>
<td>Lack of fruit and vegetables</td>
<td>3.0</td>
<td>2.4</td>
<td>2.7</td>
</tr>
<tr>
<td>High blood cholesterol</td>
<td>3.2</td>
<td>1.9</td>
<td>2.6</td>
</tr>
<tr>
<td>Illicit drugs</td>
<td>2.2</td>
<td>1.3</td>
<td>1.8</td>
</tr>
<tr>
<td>Occupation</td>
<td>2.4</td>
<td>1.0</td>
<td>1.7</td>
</tr>
<tr>
<td>Unsafe sex</td>
<td>1.1</td>
<td>0.7</td>
<td>0.9</td>
</tr>
</tbody>
</table>

1.2 Non-traditional risk factors for chronic diseases in adults

As well as diet, other factors play a role in the development of chronic diseases in adulthood. Socio-economic status and people's control over the circumstances of their lives, which are of particular relevance to the Australian Indigenous population, may have an impact on health in adulthood. As well, Syndrome X, which becomes evident after transition from a traditional to a westernised lifestyle and includes high rates of obesity, Type 2 diabetes and cardiovascular and renal disease, has been postulated as a cause of the high levels of morbidity and mortality among Indigenous Australians from these conditions.

It has also been hypothesised that nutrition prenatally and in the first months/years of life is associated with obesity, diabetes and cardiovascular disease in adults (‘Barker hypothesis’). Links between low birthweight and coronary heart disease, high blood pressure and Type 2 diabetes have been confirmed (Leon et al 1996; Curhan et al 1996a; 1996b; Rich-Edwards et al 1997; 1999) but the attribution of adult disease to prenatal or early postnatal nutritional deprivation is less secure. The hypothesis will require further investigation and refinement in the future.

Socio-economic differentials and the control factor

The 1992 National Health Strategy found that the lowest socio-economic group of the population experienced the highest death rates for a variety of conditions, including diabetes, coronary heart disease and cerebrovascular disease. The association between low socio-economic status and higher rates of various diseases in adults has also been noted in a number of other studies (summarised in National Health Strategy 1992). The prevalence of risk factors such as cigarette smoking and obesity is usually higher in the lower socio-economic groups. However, analyses of similar data in the United Kingdom have shown that the higher rate of mortality remains unexplained after differences in risk factor profiles are adjusted for (Marmot et al 1987; 1997). More recent analyses in Australia have confirmed this (Mathers et al 1999). In other words, programs to improve risk factor profiles are not enough. The effect of socio-economic status on Indigenous health is discussed in Subsection 4.1.

In addition to the variation in the accepted risk factors, the socio-economic differentials are also related to differentials in finances, education and housing. Health differentials are apparent even within the British civil service (Marmot et al 1997), which indicates that relative social standing, rather than absolute poverty, is an important factor.

The concept that the capacity of people to control the circumstances of their lives has an effect on their health is now called ‘the control factor’. This concept is supported by some evidence that psychosocial stress influences nervous, immune and endocrine function. This factor may be particularly important for groups such as Indigenous Australians that have experienced social and cultural changes at a pace that exceeds their ability to accommodate (Evans et al 1994). Syme calls this notion ‘mastery’ and proposes that the challenge is ‘to give people secrets as to how they can negotiate their lives’ (Mastering the Control Factor, Radio National, November
This may explain why health transition research consistently shows that improving maternal education has a positive influence on child health (Devitt et al, in press). However, more than education is involved (Marmot et al 1997). The long-standing focus on providing health services and health promotion and the relative neglect of addressing empowerment (Devitt et al, in press) may help to explain why the health of Indigenous Australians is relatively poorer than the health of some other indigenous groups such as the Maori and Native Americans.

**Syndrome X and the thrifty gene**

Syndrome X (or the insulin resistance syndrome) (Reaven 1988) occurs in all populations exposed to western lifestyle, but is particularly evident in indigenous populations exposed to rapid lifestyle change in the 20th century. This syndrome becomes evident after the transition from a traditional to an ‘urbanised’ or ‘westernised’ lifestyle and includes high rates of obesity, Type 2 diabetes and cardiovascular and renal disease (Rowley et al 1997). It has recently been proposed that albuminuria should be included as a component of Syndrome X and that reduction in levels of albuminuria could prove useful as a marker of effectiveness in treatment programs targeting cardiovascular disease as well as renal disease (Hoy et al 1996a).

At least two hypotheses have been put forward to explain the occurrence of Syndrome X: firstly that it may result from fetal under-nutrition as postulated by Barker, and secondly, that it may develop or be exacerbated by consequences of westernisation (energy-dense diet, physical inactivity, obesity). There is a growing consensus that these explanations may not be mutually exclusive, and that development of many chronic diseases may be precipitated by stresses occurring across the life span. Key questions now under intensive investigation include the influence of environmental factors on gene expression, and the concept of ‘critical periods’ in development. For example, if undernourishment occurs at a critical period in embryonic life the consequences may be irreversible (eg lower number of nephrons predisposing to renal disease in later life if an additional stress such as high blood pressure or diabetes is superimposed).

Australian Aboriginals appear to be susceptible to insulin resistance, which is a precursor of Type 2 diabetes. It has been suggested that a degree of selective insulin resistance previously conferred a survival advantage under the ‘feast or famine’ conditions that existed as part of the hunter-gatherer lifestyle in traditional populations (Neel 1963; O’Dea 1991a). The dietary energy intake of Aboriginals varied daily and seasonally, depending on food availability (‘feast-and-famine’ food pattern). The conversion of excess dietary energy into body fat during ‘feast’ periods as an energy store for the ‘famine’ periods was probably important for survival. While there is an assumption that genetic factors are an important contributor to insulin resistance, the evidence for the role of lifestyle factors is much stronger.

A ‘thrifty genotype’ (or genes) has been proposed as an explanation for the disposition of certain populations to Type 2 diabetes following westernisation and the consequent consistent availability of a range of foods (Neel 1963). The occurrence of diabetes has recently been reinterpreted in terms of a ‘thrifty
phenotype'. The concept underlying the hypothesis is that poor fetal and early postnatal nutrition may predispose the individual to the development of Type 2 diabetes in later life as postulated by Barker (Hales & Barker 1992). This appears consistent with the Aboriginal experience in which there is consumption of high-energy, high-fat diets by adults who were undernourished as children and subject to poor living conditions (Barker 1989; Guest 1995). In this context, it is interesting to note that Neel himself in a recent review (Neel 1999) has highlighted lifestyle interventions as the most promising approach to minimising the risk of diabetes.

**Fetal origins of adult disease**

Many years ago, it was postulated that chronic diseases had their origins in early life. Over the last decade a British research group, Barker and co-workers, has pursued this idea in a series of studies using a variety of outcomes, and a range of measures of malnutrition (Barker 1994). The studies comprise ecological, individual follow-up and case-control studies, as well as studies of trends over time. They show associations between different measures of birthweight and growth during the first year of life, and the prevalence of coronary heart disease and associated conditions (stroke, high blood pressure, Type 2 diabetes) among adults.

The hypothesis, which has become known as the ‘Barker hypothesis', proposes that these diseases originate through adaptations which the fetus makes when it is undernourished. These adaptations may be structural, metabolic or endocrine and may permanently change the structure and function of the body (introducing the concept of fetal ‘programming' of susceptibility to chronic disease in adulthood). Prevention of the diseases may depend on prevention of imbalances in fetal growth or between prenatal and postnatal growth, or imbalances in nutrient supply to the fetus (Barker 1999a). However, this hypothesis does not mean that preventive actions after birth are ineffective.

Animal studies provide many examples of adaptation that occurs because the systems and organs of the body mature during periods of rapid growth in fetal life and infancy. It is proposed that there are critical windows of time during which maturation must be achieved and that failure of maturation is largely irrecoverable (Barker & Clark 1997).

The Barker hypothesis does not necessarily conflict with other theories. For example, if birthweights are lower but obesity is more frequent in the lower socio-economic groups (Bennett 1995; Mathers 1994), this might be one mechanism by which low socio-economic status exerts its influence on chronic disease. The Barker group interprets their findings by suggesting that good nutrition in fetal and early life is crucial. However, alternative interpretations of their findings have been suggested. The most important objection is perhaps that socio-economic conditions at birth tend to persist throughout life and that these conditions are more decisive for adult health than the conditions associated with fetal life and birth. Longitudinal, prospective studies that could provide conclusive evidence are lacking.

A list of studies investigating the Barker hypothesis is given in the review by Joseph and Kramer (1996). Not all the results have agreed with the hypothesis and many of
the studies suffer from extensive loss to follow-up and failure to adjust for other important factors. However, the criticism that the Barker hypothesis is refuted by the observation that rises in heart disease rates in less developed countries were not preceded by a decline in birthweight fails to consider two factors (Hoy et al 1998a):

- the role of increasing levels of adult obesity as a part of the hypothesis; and
- improvements in medical care in recent decades leading to a reduction in infant mortality, which mean that low birthweight infants who would have died in earlier times, now survive to adulthood.

The critical factor here is that low birthweight alone is not sufficient to precipitate disease — it has to be followed by additional stresses, such as high blood pressure, obesity, or psychosocial stress, later in life.

**Cardiovascular disease**

Results of a study in the United Kingdom which investigated the health of babies born from the year 1911 onwards into later life showed that men who had lowest birthweights (below 2,500 g) had the highest death rates from coronary heart disease. Men who had a greater birthweight (above 4,500 g) had a lower death rate from this disease (Barker et al 1989). A study in South India also showed that coronary heart disease is associated with small size at birth (Stein et al 1996). A review by Barker et al (1993) concluded that babies who are small at birth or during infancy, due to under-nutrition, have increased risks of cardiovascular disease as adults. The underlying mechanisms are not understood, but may relate to early influences on the development of the vascular system itself.

**Type 2 diabetes**

Studies linking low birthweight and poor growth to the age of 12 months with disease later in life suggest an increased lifetime risk for Type 2 diabetes (Hales & Barker 1992; Barker 1993; Lithell et al 1996; Barker 1999b; Rich-Edwards et al 1999). The association is independent of gestational age, gender, adult body mass index, waist-to-hip ratio, and social class at birth and in adulthood (Rewers & Hamman 1995). The risk also extends to impaired glucose tolerance and insulin resistance (Phillips et al 1994).

In a study in India, low birthweight babies tended to be insulin resistant as adults. However, short, fat babies of heavier mothers tended to develop Type 2 diabetes and were both insulin resistant and insulin deficient, with a low 30-minute insulin response to glucose loading (Fall et al 1998). This may help to explain the current epidemic of Type 2 diabetes in urban and migrant Indian populations (Barker 1999b). Widespread fetal malnutrition in the Indian population whose average birthweight is only 2,900 g predisposes them to insulin resistance.

It is important to differentiate between under-nutrition *in utero*, which may lead to reduced beta cell function, and over-nutrition (via hyperglycaemia of the diabetic pregnancy), which could lead to increased beta cell development. Diabetes risk in later life may be increased by both, but by different pathways.
Studies in the Pima Indians in the United States indicate a strong effect of diabetes in pregnancy on the subsequent risk of premature obesity and diabetes in the offspring (Pettitt et al. 1987). Infants exposed to hyperglycaemia \textit{in utero} develop a much greater insulin secretory capacity in an attempt to reduce the hyperglycaemia. Once they are born, the hyperinsulinaemia leads to hypoglycaemia (they only have their own circulation to influence) and hunger. It is therefore possible that an increased appetite is secondary to the hyperinsulinaemia acquired \textit{in utero}. The results of a recent study in the Pima seem to bear this out. Pettitt and co-workers demonstrated that the risk of diabetes was halved in offspring who had been breast-fed for at least two months (Pettitt et al. 1997). The explanation may be related to the greater satiating effect of breast milk (the composition of which changes through the feed) compared to infant formula.

Other studies agree that gestational hyperglycaemia, under-nutrition in prenatal life, followed by over-nutrition in postnatal life may also predispose to obesity, which may influence the onset of Type 2 diabetes in the later years (Ravelli et al. 1998; Dorner & Plagemann 1994).

Renal disease
The Barker group did not include renal disease as one of the outcomes. However, recent work done in the Tiwi Islands (off the coast of the Northern Territory) has shown that there is a relationship between low birthweight and/or poor growth in the first year of life and overt albuminuria in people aged 18–38 years (Hoy et al. 1998a). It is clear that birthweight is only one of a number of factors that are related to the condition. Other important predictive factors of the renal outcomes were obesity in adulthood, a history of post-streptococcal glomerulonephritis, presence of scabies, raised glucose, insulin, cholesterol and triglyceride levels and high alcohol consumption.

Obesity
The hypothesis that prenatal and early postnatal nutrition determines subsequent obesity was tested using a cohort study of young men exposed to the Dutch famine of 1944–1945. The children of women who were exposed to famine in the first half of pregnancy had significantly higher rates of obesity when they grew up than those who were not exposed to famine at all \textit{in utero}. By contrast, if exposure to famine occurred in the last trimester or the first few months of (post-natal) life, the children had significantly lower rates of obesity when they grew up (Ravelli et al. 1998).
Blood pressure in Aboriginals — genes or environment?

In the 1960s a researcher from Adelaide, Professor Vic MacFarlane, conducted a series of studies on water balance and blood pressure in several groups of central Australian Aboriginals with different exposures to western lifestyle. The first group he studied were still living a relatively traditional hunter-gatherer lifestyle. He and his team observed the drinking patterns, collected urine samples and sweat from under the armpits, and measured blood pressure. The results were very different from those obtained for Europeans:

- the Aboriginals drank about 2 litres of water very rapidly first thing in the morning;
- their urine remained dilute throughout the day, whereas that of the Europeans became very concentrated as the day progressed;
- the potassium/sodium ratio in the sweat and urine of the Aboriginals was high, whereas it was low in the Europeans; and
- blood pressure was low in the Aboriginals and did not rise substantially with age in Aboriginal adults, in contrast to the well-established pattern among Europeans.

These initial results led MacFarlane to conclude that Aboriginals were probably genetically different from Europeans in the way that they regulated their salt and water balance and their blood pressure.

In subsequent studies he carried out similar measurements in more westernised Aboriginals and the findings were very different:

- the potassium/sodium ratio in sweat and urine was much lower than in the Aboriginals living traditionally, and particularly in the most westernised, where it was similar to that in Europeans;
- blood pressures were highest in the most westernised; and
- blood pressure increased with age in the most westernised.

These subsequent studies illustrated very clearly that his initial observations were not due to genetic differences, but rather to marked differences in diet and lifestyle of the people living a traditional lifestyle. The explanation for the earlier results was:

- the traditional diet had a much higher content of potassium than sodium (unprocessed foods with no added salt and no depletion of potassium);
- this type of dietary mineral balance could explain the low blood pressures; and
- the lack of an increase in blood pressure with age could be explained by the leanness of the adults throughout their life (no weight gain with age), and by their high level of physical activity.

The two important messages from this study are:

- the very powerful impact of diet and lifestyle on risk factors for cardiovascular disease, Type 2 diabetes and renal disease; and
- the pitfalls in too readily attributing differences between populations to genetics.

2.1 Demography

About one in fifty Australians (2.1 per cent) is an Aboriginal or Torres Strait Islander. At 30 June 1996, approximately 386,000 Aboriginal and Torres Strait Islander people lived in Australia. Approximately 8 per cent identified themselves as Torres Strait Islanders and 3 per cent as both Torres Strait Islander and Aboriginal persons with the remainder identifying themselves as Aboriginal (ABS 1997a).

Geographical distribution

Approximately half of the Indigenous population lives in New South Wales (28 per cent) and Queensland (27 per cent); 13 per cent live in the Northern Territory and 15 per cent in Western Australia (ABS 1998a) (Figure 2.1). Most Torres Strait Islanders (57 per cent) live in Queensland (ABS 1997a).

Figure 2.1 Proportions of the Indigenous population by domicile

Source: ABS (1998a)

Aboriginal and Torres Strait Islander peoples are more likely to live outside of urban areas than are non-Indigenous people. Approximately one in three Indigenous people live in non-urban areas compared to about one in seven non-Indigenous people. While Indigenous people comprise a large proportion of the population in northern and central Australia, most live in south-eastern Australia (ABS & AIHW 1997).

The Northern Territory has a much higher proportion of Indigenous people in its total population (28.5 per cent) compared with other States and Territories. The proportion for other States/Territories ranges from 0.5 per cent in Victoria to 3.2 per cent in Western Australia and Tasmania (Figure 2.2).
Age

The Aboriginal and Torres Strait Islander population is relatively young compared to the total Australian population. At 30 June 1996, nearly 40 per cent of the Indigenous population was less than 15 years of age compared with approximately 21 per cent of the total Australian population. Less than 3 per cent of the Indigenous population comprised persons aged 65 years and over compared with 12 per cent of the total Australian population (ABS 1998a).

At 30 June 1996, the Aboriginal and Torres Strait Islander population had a median age of 20.1 years, 14 years younger than that of the total Australian population (34.0 years). The median age of Indigenous males was 19.3 years, which was between one and two years younger than the median age of Indigenous females (20.9 years) (ABS 1998a).


26 Nutrition in Aboriginal and Torres Strait Islander Peoples
Urbanisation and ‘westernisation’

- Although many Indigenous people live in rural or remote areas, their lifestyles are now heavily influenced by mainstream patterns of living and eating. This process is called ‘urbanisation’ but it is occurring increasingly in non-urban areas.\(^3\)
- Recently introduced ‘western’ diets are associated with high rates of ‘lifestyle’ diseases such as overweight, high blood pressure, coronary heart disease, Type 2 diabetes and renal disease.
- Loss of traditional food gathering and consumption patterns and less active daily lives are thought to contribute to the very high rates of these diseases in Aboriginal and Torres Strait Islander peoples.
- Similar changes are occurring in other traditional populations, such as Native Americans and Pacific Islanders, where lifestyles and diets have changed dramatically in recent years (Zimmet et al 1991; Gracey 1995).

2.2 Health status

The poor health of Aboriginal and Torres Strait Islanders is well known. While the health status of Australians as a whole continues to improve, the death rates for Aboriginals in Western Australia, South Australia and the Northern Territory remain largely unchanged (ABS & AIHW 1997). This means that Indigenous people have a shorter life expectancy than other Australians. Life expectancy continues to remain lower because of high death rates in young and middle-aged adults, largely due to chronic diseases such as Type 2 diabetes, cardiovascular disease and renal disease, all which have nutritional determinants.

Morbidity\(^4\)

In the 1995 NHS, the most commonly reported types of recent and/or long-term condition among Indigenous people were diseases of the respiratory system (37 per cent) and diseases of the nervous system (34 per cent) (ABS & AIHW 1999). Reporting of diabetes was seven to eight times higher among Indigenous people than non-Indigenous people among those aged 25–44 and 45–55 years and more than twice as high among those aged 55 years or more (ABS 1999a).

Hospitalisation

A hospital separation occurs when an individual is discharged, is transferred to another facility or dies. Although hospitalisation statistics may provide an indicator of health, they are not a measure of the prevalence of disease as individuals may be admitted to hospital on more than one occasion. As well, hospital admission policies vary and tend to reflect need, access and demand (ABS & AIHW 1997).

\(^3\) The term ‘westernisation’ has been used in this report.

\(^4\) As noted previously, data originating from the 1995 NHS refer only to people living in non-remote areas.
In 1995–1997, Indigenous separations for many diseases or conditions were between one and a half and three times the number of hospital separations expected based on all-Australian rates. For all causes combined, there were almost twice as many Indigenous separations as expected for both males and females. Despite data quality concerns (and the resultant underestimation of Indigenous separations), it is clear that Aboriginal and Torres Strait Islander peoples suffer more illnesses and conditions that result in hospitalisation than non-Indigenous Australians (ABS & AIHW 1999). Table 2.1 shows Indigenous hospital separations for 1996–97 by cause.

Table 2.1 Indigenous hospital separations, by cause, 1996–97

<table>
<thead>
<tr>
<th>Cause</th>
<th>Age-standardised hospital separation ratio</th>
<th>Proportion of total separations (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Males</td>
<td>Females</td>
</tr>
<tr>
<td>Infectious/parasitic diseases</td>
<td>2.0</td>
<td>2.2</td>
</tr>
<tr>
<td>Neoplasms</td>
<td>0.7</td>
<td>0.7</td>
</tr>
<tr>
<td>Endocrine/metabolic</td>
<td>2.8</td>
<td>2.8</td>
</tr>
<tr>
<td>Blood and blood-forming organs</td>
<td>0.6</td>
<td>1.1</td>
</tr>
<tr>
<td>Mental disorders</td>
<td>2.5</td>
<td>1.6</td>
</tr>
<tr>
<td>Nervous system</td>
<td>1.4</td>
<td>1.3</td>
</tr>
<tr>
<td>Cardiovascular disease</td>
<td>1.7</td>
<td>2.0</td>
</tr>
<tr>
<td>Respiratory system</td>
<td>2.0</td>
<td>2.3</td>
</tr>
<tr>
<td>Digestive system</td>
<td>1.1</td>
<td>1.0</td>
</tr>
<tr>
<td>Genitourinary system</td>
<td>1.1</td>
<td>1.2</td>
</tr>
<tr>
<td>Complications of pregnancy and childbirth</td>
<td>-</td>
<td>1.4</td>
</tr>
<tr>
<td>Diseases of the skin and subcutaneous tissue</td>
<td>2.8</td>
<td>3.2</td>
</tr>
<tr>
<td>Musculoskeletal/connective tissue</td>
<td>0.8</td>
<td>0.9</td>
</tr>
<tr>
<td>Congenital anomalies</td>
<td>0.5</td>
<td>0.6</td>
</tr>
<tr>
<td>Perinatal</td>
<td>0.8</td>
<td>0.9</td>
</tr>
<tr>
<td>Ill-defined</td>
<td>1.5</td>
<td>1.5</td>
</tr>
<tr>
<td>Injury and poisoning</td>
<td>1.7</td>
<td>2.2</td>
</tr>
<tr>
<td>Other reasons</td>
<td>2.9</td>
<td>3.7</td>
</tr>
<tr>
<td>All causes</td>
<td>1.7</td>
<td>1.7</td>
</tr>
</tbody>
</table>

Note: Includes data from public and private hospitals except in the Northern Territory (public hospitals only). Age-standardised hospital separation ratio is equal to hospital separations identified as Indigenous divided by expected separations, based on all-Australian rates.


Age-specific hospital separation rates for Indigenous males and females exceeded those of non-Indigenous Australians at all ages. For both males and females, the differences were smallest among children aged 1 to 14 years and largest among infants and older people (ABS & AIHW 1999).
Analysis of the hospitalisation statistics in Aboriginal and Torres Strait Islander peoples for 1997–98 shows the increasing impact of renal failure on the health of this group (Cunningham & Beneforti 2000).

**Figure 2.4 Age-specific hospital separations for all causes excluding dialysis, 1997–98**

Source: Cunningham & Beneforti (2000).

**Figure 2.5 Age-specific hospital separations for all causes including dialysis, 1997–98**

Source: Cunningham & Beneforti (2000).

**Life expectancy at birth**

In the period 1991–1996, life expectancy at birth for all Australians was 75.2 years for males and 81.1 years for females. By contrast, life expectancy at birth was estimated to be 56.9 years for Indigenous males and 61.7 years for Indigenous females (ABS & AIHW 1999). Therefore, the life expectancy at birth for Indigenous males and females is estimated to be about 18–19 years less than their all-Australian counterparts (ABS & AIHW 1999).
Table 2.2  Estimated Indigenous life expectancy at birth, 1991–1996

<table>
<thead>
<tr>
<th></th>
<th>Western (years)</th>
<th>Eastern (years)</th>
<th>Australia (years)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td>53.7</td>
<td>59.2</td>
<td>56.9</td>
</tr>
<tr>
<td>Females</td>
<td>58.9</td>
<td>63.6</td>
<td>61.7</td>
</tr>
</tbody>
</table>

Notes: ‘Western’ comprises Western Australia, South Australia and Northern Territory combined; and ‘Eastern’ comprises New South Wales, Victoria, Queensland, Tasmania and Australian Capital Territory combined.

Source: Cunningham & Paradies (2000).

Perinatal mortality

In 1994–1996, the perinatal mortality rate for babies of Aboriginal and Torres Strait Islander mothers was more than twice that for babies born to non-Indigenous women (Figure 2.6) (ABS & AIHW 1999).

There have been reductions in perinatal mortality in at least some jurisdictions over the last decade or so. In Western Australia, the perinatal mortality rate for babies of Indigenous mothers fell from 23.3 per 1,000 births in 1986 to 17.2 in 1995, although the rates remained more than double the non-Indigenous figure of 6.8 per 1,000 in 1995. In the Northern Territory, the perinatal mortality rate per 1,000 births for babies born to Indigenous mothers fell from 48.9 in 1986 to 26.4 in 1995 (Markey et al 1998). The fall in perinatal mortality in the Northern Territory may be related to an 85 g increase in the mean birthweight and the associated decrease in low birthweight babies (Markey et al 1998).

Figure 2.6 Perinatal mortality by mother's Indigenous status, 1994–1996

Note: Perinatal deaths are stillbirths and neonatal (<28 days of age) deaths. Data for the Australian Capital Territory and Tasmania are not presented separately due to small numbers but are included in the rate for Australia.

Infant mortality

The infant mortality rate is much higher in Aboriginal and Torres Strait Islander peoples than in the general population. Infant mortality rates for Indigenous Australians have been available for several decades in the Northern Territory but were first reported for Western Australia in 1977, for South Australia in 1986 (ABS & AIHW 1997) and for Queensland in 1998 (ABS 1999a). Information is not yet available for the other States/Territories owing to incomplete data relating to race on the death certificates. In 1997, the infant mortality rate for South Australia, Western Australia and the Northern Territory was 18.1 per 1,000 live births (ABS 1998b; 1998c). In 1998 it was 15.2 per 1,000 live births in those States/Territories plus Queensland (ABS 1999a). The rates per 1,000 live births in the total Australian population were 5.3 in 1997 (ABS 1999a) and 5.0 in 1998 (ABS 1999b).

The Northern Territory is the only area that has data extending back to the early 1970s. In 1972–1974, the infant mortality rate in Northern Territory Aboriginals was 83.4 per 1,000 live births compared to 16.6 per 1,000 live births for the total Australian population (ABS & AIHW 1997). Even though the data are reported annually by the ABS, it is unclear what the exact trends are. In recent years Indigenous people have shown an increasing preparedness to identify themselves as Indigenous. This may cause differences between race breakdown statistics of the total population and records of deaths as these are collected in different systems. In particular, recent trends have a discontinuity following the 1996 Census when 51,800 more people identified themselves as Indigenous than had been predicted from the 1991 Census. In addition, trends are difficult to assess over a short time when the number of births and deaths is small. For example, there are less than 700 Indigenous live births in South Australia per year (ABS 1998c), so a few more deaths registered in a particular year will make quite a large difference in the infant mortality rate for the year and this may not reflect the long-term trend.

Mortality after infancy

In 1995–1997 in Western Australia, South Australia and the Northern Territory combined, there were three times as many deaths among males and females identified as Indigenous as would have been expected if Indigenous people had experienced the same death rates as all Australians (Cunningham & Paradies 2000).

No information about the mortality of Torres Strait Islanders living outside the Torres Strait has been available to date due to the lack of separate identification of Torres Strait Islanders and Aboriginal people in death registrations. Reliable information about deaths of Torres Strait Islanders in the Torres Strait has not been available for the last several years due to under-registration of deaths in the Torres Strait region (ABS & AIHW 1999). Over the period 1976–1994, the estimated mortality rates for Torres Strait Islanders living in the Torres Strait area were about two and a half to three times higher than those for all Queenslanders after adjusting for age (ABS & AIHW 1999).
Age-specific death rates

In 1995–1997, the age-specific death rates for Indigenous males and females exceeded those of their non-Indigenous counterparts in every age group. Deaths identified as Indigenous occurred at younger ages than other deaths. The differences were greatest in relative terms among those aged 35–54 years. The death rates in this age group were six to seven times higher for Indigenous people than they were for their non-Indigenous counterparts (Cunningham & Paradies 2000).
Cardiovascular disease accounted for the largest number of deaths identified as Indigenous among males and females in Western Australia, South Australia and the Northern Territory combined in 1995-1997. Cardiovascular disease, injury, neoplasms, respiratory diseases and endocrine diseases (i.e. diabetes) together accounted for about three out of every four deaths identified as Indigenous. Although these causes were responsible for the majority of deaths in Australia as a whole, they occurred at greater rates among the Indigenous population (Cunningham & Paradies 2000).

Note: Data are from Western Australia, South Australia and the Northern Territory, based on year of registration.

Figure 2.9 Deaths in Indigenous males and females from selected causes, 1995-1997

Note: Data from Western Australia, South Australia and the Northern Territory combined, based on year of registration.


The main causes of excess deaths in the Torres Strait area for 1989–1994 were diabetes and coronary heart disease (ABS & AIHW 1997).

Rural and remote areas

While mortality patterns have been described in different States and Territories, little data is available comparing mortality patterns between Aboriginals in metropolitan, rural and remote areas of Australia (Peach et al 1998). Peach et al compared the ranges and variances of disease-specific standardised proportional mortality ratios (SPMR) between geographical areas in 1990 to 1993 — within Western Australia, South Australia, Northern Territory, New South Wales and Victoria — to those of corresponding standardised mortality ratios (SMR) (expected number of deaths obtained by age-specific mortality rates among Australian adults in 1990–1993 to the 1991 population census data).

The SMR for endocrine, cardiovascular, respiratory and digestive diseases were significantly higher among Aboriginals than non-Aboriginal peoples in all areas. There was an increasing mortality gradient from metropolitan to rural areas for neoplasms and endocrine, cardiovascular and genitourinary diseases among males and females; and for respiratory diseases among males only. The authors suggested that differences in access to health care, goods, services and facilities combined with reduced education and employment opportunities are implicated in the higher death rates among Aboriginals in rural and remote communities.

The study by Peach et al (1998) did not address the possibility that differences in observed rates by area can be explained at least in part by differences in the quality of identification of Indigenous deaths. That is, deaths of Indigenous people in rural areas may be more likely to be categorised as such than deaths of Indigenous people in metropolitan areas. It is not known how great a difference this would make.
CHAPTER 3

ABORIGINAL AND TORRES STRAIT ISLANDER DIET

To understand the present state of Aboriginal and Torres Strait Islander nutritional health it is necessary to set it in historical perspective. The limited information about the diet and nutritional health of Aboriginal and Torres Strait Islander peoples before European colonisation suggests slim, strong people living in harmony with their environment (Coyne & Darnton-Hill 1979). There is no evidence that Aboriginal people living traditionally as hunter-gatherers had diabetes or diseases of the cardiovascular system (Naughton et al 1986). Therefore, an understanding of the nutrient composition and the physiological effects of traditional foods should help provide background information for the development of appropriate dietary guidelines (Brand Miller & Holt 1998).

3.1 Traditional lifestyle and diet

The ancestors of Australian Aboriginal and Torres Strait Islanders first migrated to Australia from south-east Asia at least 40,000 to 50,000 years ago and moved slowly south across the continent (Blainey 1982). The migration would have necessitated dietary adaptations to unfamiliar species, through the process of ‘eat, die, and learn’ (Webb 1973).

Aboriginal diet

Aboriginals successfully adopted a hunter-gatherer lifestyle and continued to live as hunter-gatherers under widely different geographical and climatic conditions until European colonisation (O’Dea 1991b). Survival depended on intimate knowledge of the land, sources of water, and the detailed effects of the seasonal cycles on plant foods and game.

Aboriginals were omnivorous, deriving their diet from a wide range of uncultivated plant foods and wild animals. The composition and diversity of the food supply, and the relative proportions of plant and animal foods, were greatly influenced by both the season and the geographical location (O’Dea 1991b). Most observers describe a varied and ample range of both plant and animal foods, even in arid zones (Cleland & Johnston 1933; Campbell 1939; Marbutt 1971; Gould 1973). However, the quantity and quality of food intake also varied greatly on a day-to-day basis. The usual pattern was of subsistence intake supplemented by ‘feasts’ when large game were successfully hunted.

The most highly prized components of the Aboriginal hunter-gatherer diet were the relatively few energy-dense foods — depot fat, organ meats, fatty insects and honey (O’Dea 1991b). Generally, high fat content was considered a principal indicator of

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5 Material on Aboriginal diet in this chapter is largely derived from Lee (1996).
meat quality. Other favoured foods such as witchetty grubs (*Cossidae* sp) and marine mammals, also have a high fat content (Basedow 1925; Blainey 1982; Cherikoff et al 1985; Rose 1987).

Traditional diets were generally low in sugars (Rose 1985) although sweetness was provided by honey ants (*Melophorus inflatus*), the honey of the native bee, blossoms (eg *Grevillea* sp), lerp (secretion from the insect *Psylla* living on the leaves of *Eucalyptus* sp) and gums.

**Torres Strait Island diet**

Torres Strait Islanders were traditionally a marine-hunting, horticultural and trading society. They possessed an intimate knowledge of the tides, feeding patterns and movements of a variety of marine life (Broomehead et al 1995). The traditional food supply in the Torres Strait varied from island to island (Beckett 1987) and the Islanders were more reliant on subsistence agriculture than the Aboriginal population, supplementing this with fishing, hunting and foraging, in varying proportions on different islands throughout the year. Garden foods were stored and preserved, and trade of produce was particularly important (Leonard et al 1995).

Turtle and dugong occupied a particular place in the cultural life of the Torres Strait. No feast was staged without either turtle or dugong meat being on the table (Fuary 1991). Like the Aboriginals, Torres Strait Islanders prized fat, distinguishing good meat from bad on the basis of its fat content.

**Food collection, preparation and storage**

Foraging parties generally gathered sufficient food for immediate requirements and rarely stored food for later use (Thompson 1949; Latz & Griffin 1978). Women provided the subsistence diet of highly reliable foods including small marsupials, shellfish, reptiles and insects, honey, eggs and plant foods (Berndt 1978; Hamilton 1980; Bell 1983). Men primarily hunted for large game (mammals, marsupials, birds, reptiles and fish), procuring fruits and other plant foods for themselves during the hunt. When hunting was successful, the catch was shared by the whole group.

Many foods such as fruits, bulbs, nectar, gums and flowers were eaten fresh and raw; frequently as they were collected. If a food was not eaten raw, it was usually roasted on coals or baked in an earthen oven. Most food processing was done to remove harmful substances or make the food more digestible or palatable (O’Dea 1991b).

**Food distribution**

Foods were prepared, proportioned and distributed according to traditional law, with strict cultural practices being determined by kin obligations (Gould 1967; Berndt & Berndt 1970; Elphinstone 1971; Penny & Moriarity 1978). Sharing of food had a social, as well as a physiological function, enabling the affirmation of relationships (Stacy 1975) and confirming the significance of ceremonies and rights of passage (Meehan 1982).
Children were traditionally breast-fed until approximately three years of age, the age of weaning depending on the arrival of another sibling. Solids were not introduced until eruption of teeth (McArthur 1960; Hamilton 1981). Responsibility for feeding tended to rest with the child, who was expected to indicate desire for food, and was fed on demand, deciding ‘what and when to eat’ (Hamilton 1982). Feeding of older children had priority over the feeding of small infants (Hamilton 1981; 1982).

In the Torres Strait, butchering of a turtle or dugong was (and remains) a complex process, occurring on the beach in view of the community. Meat from the catch was distributed with great care according to prescribed protocols. It was possible for the successful hunter to take home less meat than other people with whom the catch was shared (Nietschmann & Fitzpatrick 1981; Fuary 1991).

Health aspects of traditional diets and lifestyles

On the basis of available information, the traditional diet was generally low in energy density but high in nutrient density, being high in protein, low in sugars, high in complex carbohydrate of low glycaemic index and high in micronutrients. Even though the traditional Aboriginal diet contained a high proportion of animal foods, it would have been low in total fat, extremely low in saturated fat and relatively high in polyunsaturated fatty acids including the long-chain highly polyunsaturated fatty acids of both the omega-3 and omega-6 families, and hence protective against cardiovascular disease and related conditions.

The composition of most traditional vegetable foods is typical of uncultivated plants worldwide, being high in fibre and relatively high in protein with a generally low energy density (Brand Miller et al 1993). The carbohydrate in most traditional plant foods is of low glycaemic index, producing lower glucose and insulin levels than similar Western foods. Their consumption may be protective against diabetes (Thorburn et al 1987).

Although some animal foods, such as witchetty grubs (Cossidae sp.) and green ants (Oecophylla smaragdina), have a relatively high fat content (Cherikoff et al 1985), most native land animals are very lean. Traditional meat foods have a much lower carcass fat content and intramuscular lipid content than meat from domesticated animals, such as cattle and sheep (Lee 1996; Naughton et al 1986). Most carcass fat is stored in discrete depots within the abdomen. These fat depots tend to be small and were traditionally shared by many people.

Several accounts highlight the labour-intensive aspects of collecting and preparing traditional foods (Spencer & Gillen 1899; Tindale 1974; Devitt 1988). Food procurement and preparation by Aboriginal hunter-gatherers were energy-intensive processes that could involve sustained physical activity for many hours daily. Activities included walking long distances, digging for tubers, digging for reptiles, eggs, honey ants, and witchetty grubs, chopping with a stone axe, winnowing and grinding of seeds, digging pits for cooking large animals, and gathering wood for fires (O’D ea 1991b).
3.2 Transitional diet

With decreased availability of, and access to, traditional foods following European settlement, Aboriginal people were increasingly forced to depend on European foods (Gould 1972). These were available from railway sidings and telegraph stations, from pastoralists, miners and from religious missions. Early rations included flour, sugar, tea and to a lesser extent meat (fresh, tinned or salted) (Walker & Roberts 1988). These foods were chosen mainly because of their relative durability, low bulk, transportability, inexpensiveness and the simple cooking and storage facilities required for their preparation.

Aboriginal groups were centralised at cattle stations, government settlements or missions (Long 1970). Flour, sugar and rice were the staple ration foods at settlements and missions, with supplementary and irregular quantities of fruit and vegetables from the local gardens (McArthur 1960). Beef and lamb were available in pastoral areas and fish and marine products were harvested at the coastal settlements. Sugar was eagerly sought and was consumed in large quantities from early contact (MacFarlane 1978).

Rations were not necessarily adequate either in quality or quantity (Lee 1996). Specific nutritional problems included inadequate supplies of energy, protein, iron, calcium, vitamin B\(_2\), vitamin A and vitamin C (Billington 1960; Corden 1962). Recourse to bushfoods occurred at all communities whenever rationing was inadequate.

From the 1930s, trading posts were established in remote areas to encourage people to remain on their own lands (Kyle-Little 1957). Some failed due to a shortage of staff and funds, but others developed into service centres and eventually became settlements. Communal dining rooms were established at many settlements and missions. Hence, Aboriginal people were deprived of all responsibility for the acquisition and preparation of their own food, as for most other aspects of their lives (Berndt & Berndt 1942; McArthur 1960; Long 1970; Hamilton 1972).

Communal feeding at settlements and missions during the middle of this century meant that responsibility to share food resources declined and a breakdown in the well-defined traditional food distribution pattern was observed (Sinclair 1977). This tended to affect the more vulnerable groups such as toddlers, pregnant and breast-feeding mothers and the elderly (Stacy 1975; Sinclair 1977; Cutter 1978). The food given to the young children was inadequate in energy and was low in fat. The Aboriginal women were prevented from giving their children any additional food. Pregnant women were given extra rations, but rations for breast-feeding women were often inadequate.

Early settlements and missions lacked the equipment, staff and regular transport required to provide a comprehensive, nutritious and hygienic communal feeding service (Wilson 1953; Sinclair 1977). Compared with other Australian diets of the time, the diet provided included more flour, bread and meat and less fruit, vegetables and dairy products (Wilson 1953). A study from south-western Australia showed that the dietary consumption levels of calcium, vitamin B\(_2\) and vitamin C were low.
in people who lived on reserves compared to those who lived in transitional housing and Government Housing Commission dwellings (Hitchcock & Gracey 1975).

In the 1960s, Aboriginals were given the right to be counted in census, the right to vote, the right to drink alcohol and the right to equal pay. Soon after, communal feeding was discontinued on most settlements. Instead, Aboriginals were given their full wages in cash so that they could purchase their own provisions. No spectacular improvement in diet resulted because, with little money and little choice of foodstuffs, they had no option but to make do with a poor diet consisting mainly of white flour, sugar, tea and meat (Reid & Trompf 1991).

By the mid-1970s, Aboriginal settlements had access to well-stocked foodstores. However, observations at the time revealed an average purchase for a family of five would include 11.5 kg of white flour, 2.75 kg white sugar, 7 oranges (or apples if available) and 12 eggs. Most weeks no bush fruits or vegetables would be collected (White 1977). People living some distance from the community stores, particularly in outstations, tended to purchase easily stored foods, such as flour and sugar. Much of this was stored in flour drums on the roofs of humpies, or in the forks of trees to keep it from dogs and children (Cutter 1978).

In 1972, land rights legislation and a policy of self-determination was introduced by the Commonwealth Government (Deane 1997), facilitating the gradual return of local political power to some Aboriginal people. Social security entitlements were extended and increased and award wages were maintained, although few Aboriginals were employed (Peterson 1979). By 1980, most Aboriginals received social security entitlements, including unemployment benefits, and were economically dependent on government. Aboriginal councils were established to administer settlements, while Aboriginal representative bodies, such as Land Councils and Progress Associations, were formed. Opportunity was provided for community ownership and/or management of settlement retail stores (Young 1984). These political changes coincided with the outstation or homelands movement, whereby small family groups were supported to re-inhabit traditional lands in remote areas (Coombs 1974; 1978).

At the time, it was considered that nutrition could improve in the setting of self-reliance and self-help arising from the outstation movement. Varying combinations of feral animals, natural plants, fruit and vegetables grown in gardens, supplemented from the store, provided the basis for an adequate diet and better nutrition than in the past (Hetzel & Frith 1978).

It had been suggested that, in the short-term, after establishment of ‘new’ settlements or outstations, there may be an increase in yields of traditional foods due to high initial availability and the use of Western technology (Sinclair 1977; Cutter 1978; Altman 1987). In some areas, introduced feral animals such as rabbits and buffalo were also popular (Calaby 1971; Altman 1987). However, the longer-term effect appears to be a reduction in the availability of traditional foods due to several factors. These include (Peterson 1978; Cane & Stanley 1985):

- the effect of environmental degradation caused by stock and feral animals;
- the introduction of exotic plant species;
• the increasing incidence of hot, destructive bush fires due to poor land management practices;

• the restricted access to some areas of land;

• depletion of resources and population pressure around permanent settlements;

• high costs associated with the acquisition and maintenance of equipment, firearms, vehicles, and fuel;

• changing demographic patterns; and

• cultural loss from generation to generation.

In summary, the fundamental changes that European settlement imposed on Aboriginal life led to a loss of control over their own destinies. The lack of access to adequate housing, hygiene, education, and employment opportunities available to Indigenous Australians has had a serious negative impact on their health and well-being. Changes in their diet and lifestyle have been integral parts of the process (Reid & Trompf 1991).

3.3 Contemporary diet

With the transition from a traditional hunter-gather lifestyle to a settled westernised existence, Aboriginal and Torres Strait Islander diet has changed from a varied, nutrient-dense diet to an energy-dense diet, high in fat and refined sugars. As discussed, preferred foods such as meat, fat and sweet foods were not readily available traditionally and were shared among a large number of people (Naughton et al 1986). Foods now eaten to substitute for traditional foods include fresh, frozen and tinned meat, fatty cuts of beef and lamb, honey, treacle, golden syrup, jam, confectionery and refined sugar. These are now readily available in comparatively large quantities (Devitt 1991; Lee et al 1994a).

Contemporary dietary practice reflects the continuing influence of the high value associated with fat. A major consequence of its persistence is the incorporation of new and different ‘fats’ (including oils) in the diet. It underlies the rapid adoption of a new cooking technique (frying) which enables the addition of fat to meat.

In the same way, sugar consumption among the Aboriginal and Torres Strait Islander population is high. In the NATSIS, older adults reported consuming less sugar than younger adults, but there does not seem to be a clear difference in consumption by men and women or for those living in capital cities versus other areas (ABS 1996a).

Contemporary foraging patterns may be explained in terms of necessity, dietary preferences and cultural satisfaction (Mcchan 1982; Devitt 1988). Now that staples such as flour, other carbohydrates, fats, oils, meats, tinned and packaged products are readily available, the uncertainty of survival has been removed. Unlike some traditional food which may be taboo at certain times, European food is usually considered acceptable (Taylor 1979). The social function of traditional food has been seen to be more important than the fact that it tastes good and stops hunger; European food is now seen to fulfil the latter role (Stacy 1975). Table 3.1 compares
characteristics of the traditional hunter-gather lifestyle with contemporary westernised lifestyle.

**Table 3.1  Characteristics of hunter-gatherer and western lifestyles**

<table>
<thead>
<tr>
<th></th>
<th>Hunter-gatherer lifestyle</th>
<th>Western lifestyle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physical activity level</td>
<td>high</td>
<td>low</td>
</tr>
<tr>
<td>Principle characteristics of diet</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Energy density</td>
<td>low</td>
<td>high</td>
</tr>
<tr>
<td>Energy intake</td>
<td>usually adequate</td>
<td>excessive</td>
</tr>
<tr>
<td>Nutrient density</td>
<td>high</td>
<td>low</td>
</tr>
<tr>
<td>Nutrient composition of diet</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Protein</td>
<td>high</td>
<td>low-moderate</td>
</tr>
<tr>
<td>Animal</td>
<td>high</td>
<td>moderate</td>
</tr>
<tr>
<td>Vegetable</td>
<td>low-moderate</td>
<td>low</td>
</tr>
<tr>
<td>Carbohydrate</td>
<td>moderate (slowly digested)</td>
<td>high (rapidly digested)</td>
</tr>
<tr>
<td>Complex carbohydrate</td>
<td>moderate</td>
<td>moderate</td>
</tr>
<tr>
<td>Simple carbohydrate</td>
<td>usually low (honey)</td>
<td>high (sucrose)</td>
</tr>
<tr>
<td>Dietary fibre</td>
<td>high</td>
<td>low</td>
</tr>
<tr>
<td>Fat</td>
<td>low</td>
<td>high</td>
</tr>
<tr>
<td>Vegetable</td>
<td>low</td>
<td>low</td>
</tr>
<tr>
<td>Animal</td>
<td>low (polyunsaturated)</td>
<td>high (saturated)</td>
</tr>
<tr>
<td>Sodium:potassium ratio</td>
<td>low</td>
<td>high</td>
</tr>
</tbody>
</table>


**Urban Aboriginal communities**

Contemporary food choices among urban Aboriginals have not been widely studied (Guest & O’D ea 1993). The results of a small study using 24-hour recall in the coastal town of Kempsey in New South Wales suggested that the overall dietary patterns and nutrient intakes of Aboriginals living in cities and towns is more similar to that of the general population than it is to Aboriginals living in remote parts of Australia (Sibthorpe, unpublished).

A comparison of the food habits of Aboriginals and non-Indigenous Australians in a city and a country town showed that in both localities Aboriginals consumed take-away meals more frequently than their non-Indigenous counterparts, with urban Aboriginals being the most frequent consumers. Similar proportions of Aboriginals in a city and in a country town used salt at the table ‘most of the time’. More Aboriginals added salt at the table ‘most of the time’ than non-Indigenous people from the country town. Comparisons of Aboriginals with other Australian population groups and of city with country groups may have been confounded by socio-economic differences (Guest & O’D ea 1993).
Remote Aboriginal communities

The diet in remote Aboriginal communities tends to be high in energy and sugars, moderately high in fat and relatively low in complex carbohydrate, fibre and nutrient density (Hitchcock & Gracey 1975; Cutter 1978; Coles-Rutishauser 1979). Results of dietary studies suggest a style of diet in remote communities that consists of a monotonous and limited variety of foods with a very low intake of fresh food, particularly fruit and vegetables (Lee et al 1995a). In contrast to the high intakes of energy, sugars and fats, the intake of dietary fibre, some minerals (calcium and zinc) and some vitamins (vitamin B₂, vitamin E, betacarotene and folic acid) appear to be low (Lee et al 1994b). Compared with national Australian apparent consumption data, intakes of sugar, white flour and sweetened carbonated beverages are much higher in Aboriginal communities in the Northern Territory and intakes of wholemeal bread, fruit and vegetables are much lower (Lee et al 1994b).

A study into diet in six remote Aboriginal communities in the Northern Territory using the ‘store-turnover’ method (see Appendix 1) showed that sugar, flour, bread and meat provided more than half the apparent total energy intake. Fatty meats contributed nearly 40 per cent of the total fat intake in northern coastal communities and over 60 per cent in central desert communities. In both regions, white sugar per se contributed approximately 60 per cent of all sugars consumed (Lee et al 1994b).

Table 3.2 Apparent mean consumption of selected foods in Aboriginal communities compared with national data

<table>
<thead>
<tr>
<th>Food</th>
<th>Aboriginal Communities</th>
<th>Australian data</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Central Desert (n=3)</td>
<td>Northern Coastal (n=3)</td>
</tr>
<tr>
<td>Flour (white)</td>
<td>37.6</td>
<td>44.4</td>
</tr>
<tr>
<td>Bread (all)</td>
<td>34.1</td>
<td>30.5</td>
</tr>
<tr>
<td>Beef and veal</td>
<td>51.6</td>
<td>25.8</td>
</tr>
<tr>
<td>Poultry</td>
<td>22.3</td>
<td>19.7</td>
</tr>
<tr>
<td>Lamb</td>
<td>22.8</td>
<td>3.3</td>
</tr>
<tr>
<td>Fish</td>
<td>0</td>
<td>4.8</td>
</tr>
<tr>
<td>Fruits</td>
<td>33.2</td>
<td>17.6</td>
</tr>
<tr>
<td>Vegetables</td>
<td>24.3</td>
<td>19.6</td>
</tr>
<tr>
<td>Sugar</td>
<td>54.1</td>
<td>50.3</td>
</tr>
<tr>
<td>Carbonated beverages</td>
<td>67.9</td>
<td>224.6</td>
</tr>
<tr>
<td>Fruit juice</td>
<td>48.3</td>
<td>12.8</td>
</tr>
<tr>
<td>Tinned meat</td>
<td>9.4</td>
<td>10.1</td>
</tr>
<tr>
<td>Pie/Pastie</td>
<td>9.6</td>
<td>15.1</td>
</tr>
<tr>
<td>Snack Foods (potato crisps, extruded snacks)</td>
<td>1.8</td>
<td>2.7</td>
</tr>
</tbody>
</table>

Note: Australian data are from ABS (1987).
Bread includes flour used in bread making.
NA = not available


A dietary assessment study from remote Aboriginal communities in north-east Arnhem Land compared the diet of Aboriginal people foraging for bushfoods and
diets in town where store foods were available (Maggiore 1990). The energy intake of all subjects was approximately 50 per cent of the Australian recommended dietary intake, and the vitamin C, retinol activity, magnesium and calcium intakes were low in all diets, bush and town. However zinc and iron intakes were higher and iron equal to or above the recommended dietary intake in the diets that included bushfoods. The study showed that the Aboriginals had low levels of fruit and vegetables in diets both while foraging and when eating store food (Maggiore 1990).

A study in a central Australian settlement showed a close relationship between dietary quality and pay day (Cutter 1978). Meat, fruit and vegetables were included in the diet after pay day but were usually absent for at least several days before the next pay day. The staple diet was bread (or damper) and sweet tea which accounted for 80 per cent of energy intake. The inclusion of bushfoods (grubs, lizards, rabbits, kangaroos, wallabies, emu, bush tomatoes, wild oranges and potatoes) in the diet depended on the availability of transport to go and hunt or gather these items.

A study using 24-hour recall (see Appendix 1) to investigate dietary patterns of Aboriginal children in the Kimberley showed that bread and damper (usually with butter and often also with jam or honey) together with tea (usually with large quantities of powdered milk and sugar) were the most frequently eaten foods (King et al 1985). Fried or stewed beef, sometimes with vegetables (mainly potato, onions and carrots) were the main sources of meat and vegetables. Limited quantities of bushfoods were eaten. Dietary analysis showed evidence of low energy intake and marginal to low intake of folic acid and vitamins A, B₁ and B₆.

Even in remote traditionally orientated outstations, foods purchased at the store accounted for most of the energy intake while bushfoods provided the greatest proportion of protein intake (Meehan 1982; Altman 1987; Devitt 1988). All studies showed that flour, sugar, sweets and fats provided much of the energy intake from store-purchased foods. Animal foods, particularly those high in fat such as lizards, provided most of the energy from bushfoods. In general, dietary patterns described were meat-orientated.

**Torres Strait Island communities**

The diet of the Torres Strait Islanders in the early 1970s has been described as consisting mainly of white flour, white rice, corned beef, syrup and dripping (Duncan 1974). Demand for fresh fruit, both locally produced and imported from mainland Australia, far exceeded supply. A recent study conducted in a store of a small Torres Strait community showed little change in dietary patterns over the ensuing 20 years, with the exception that vegetable oils replace dripping. The demand for fruit and vegetables still exceeds supply (Leonard et al 1994).

Production of traditional staple crops both for household consumption and for ceremonial purposes continues in the Torres Strait (Bird et al 1995). However, the custom of contributing plant foods for ceremonies such as funeral feasts is now impeded by quarantine regulations restricting the transport of plants between the islands (Leonard et al 1995).
Marine foods continue to make substantial contributions to the diet. Men, women and children are involved in different aspects of gleaning, fishing and hunting. Torres Strait Islanders living on three outer islands were estimated to consume 191 g to 450 g per person per day of seafood (including turtle and dugong), considerably more than Japanese seafood intake (102 g per day) (Johannes & Macfarlane 1991).

A study using the ‘store-turnover’ method (see Appendix 1) was undertaken in a small island community in the Torres Strait to describe the food available through the island store for the months of February, March and April 1993. More than half the energy in the diet came from white flour, white rice, tinned meat and vegetable oil. The amount of fruit and vegetables available through the store was low. Fruit available per person was about one-sixth of the amount recommended and vegetables about one-third of the amount recommended. People who depended on store foods would not meet their needs for vitamins A, C, and E and folic acid (Leonard et al 1994).

**Bushfoods**

Over 100 years ago an English botanist’s writing of Australian edible plants suggested that many of them were ‘eatable but not worth eating’ (Cribb & Cribb 1987). More recently there has been a growing commercial interest in traditional foods (Dingle 1997; Sinclair et al 1997). While this commercialisation reflects a recently acquired preference among the non-Aboriginal community, there is also a strongly stated continued preference for traditional foods by Aboriginal people living in remote (Devitt 1988; Lee et al 1994b; Leonard et al 1995) and urban areas (DHSH 1994a).

Until the publication of the *Tables of Composition of Australian Aboriginal Foods* (Brand Miller et al 1993), which contains the nutrient contents of about 500 different traditional Aboriginal foods, many researchers (eg Meehan 1982; O’Dea 1984) estimated nutrient intake in different groups of Aboriginal people by using published and unpublished sources of nutrient composition data. In many instances assumptions were made based on extrapolations from Western foods or Aboriginal foods of similar nature (Brand Miller et al 1993).

It is important to appreciate the incompleteness of the nutrient content of bushfoods data (Maggiore 1990). Most samples analysed have come from one location collected during a specific time of the year. Details of location, season, preparation for consumption and state of maturity are frequently unavailable. For example many foods are cooked in ashes, and some of this ash will be consumed with the food. It was not known whether the analysis allowed for this.

Due to the difficulties of quantifying nutrient results, individual dietary studies have tended to focus on qualitative and semi-quantitative assessment of the diet which tend to reflect dietary patterns and preferences, rather than actual, habitual intake (Guest & O’Dea 1993).

Bushfoods contribute only a small proportion of nutrients in many areas (Lee et al 1994b). In a study of a northern coastal Aboriginal community an average of less than 15 per cent of the population sought traditional foods at least three days per
fortnight throughout the year. It was estimated that the proportion of total energy intake derived from bushfoods averaged over the population would be less than 8 per cent during the dry season and less than half this proportion during the peak of the wet season (Lee 1992).

In the NATSIS, 10 per cent of people aged over 14 years reported spending more than one hour per week acquiring traditional foods, and of these, more than half reported spending more than five hours per week doing this (ABS 1996a). Even though the actual intake of traditional foods is low, traditional foods are still popular and culturally important (Rae et al 1982).

3.4 Micronutrients

Nutrients are classified chemically as macronutrients (protein, carbohydrate, fat, ethanol and water) and micronutrients (vitamins, minerals and trace elements).

The assessment of micronutrient deficiencies has evolved from the presence of clinical abnormalities or growth failure to subclinical depleted body stores to assessing functional deficits. It has been suggested that it is useful to differentiate between type I micronutrient deficiencies in which there is a reduction in tissue concentration (e.g. iron, selenium, iodine, vitamins) and type II deficiencies (e.g. nitrogen, magnesium, chloride, zinc) in which cessation of growth occurs with normal tissue concentrations because there is no body store of the nutrient (Golden 1991). Since growth is the limiting factor for the second group, catch-up growth should be the response to treatment for deficiency, unless there are other limiting nutrients. Conversely, catch-up growth may not occur if energy alone were increased, if a deficiency of one or more of these nutrients also exists. In this situation, energy supplementation may lead to adiposity rather than growth (Golden & Golden 1981a; 1981b).

In a study of 12 Queensland settlements, the levels of folate, iron, carotene and Vitamin C were all found to be lower in children with poor growth versus children with normal growth (Jose & Welch 1970). Physical signs of rickets were also described. However, the mean values in the ‘normally’ grown children were all at the lowest end of the normal laboratory range. More recently, disturbances in the metabolism of zinc, iron and copper in children from one South Australian location have been reported (Cheek et al 1989).

Vitamins

Vitamin status has been measured infrequently in Aboriginal communities but some studies have been conducted in a variety of groups and environments. Samples have generally been small and have often been selected from ‘stress’ groups of the community (infants and pregnant and breast-feeding women). Quantitative comparison of prevalence of vitamin deficiencies may be misleading as a varying methods and ‘normal’ ranges have been used in these studies. Multiple vitamin deficiencies have frequently been described in the same subject and suggest the generally poor nutritional status of such individuals, rather than a specific micronutrient problem.
Vitamin C
Low plasma levels of vitamin C have been reported in high-risk Aboriginal groups (Lee 1996) and very low levels have been described in a remote community (Lee et al 1994a). Levels of vitamin C and folate are statistically related, consistent with the very low contemporary dietary intakes of fruit and vegetables (Lee et al 1994a).

Vitamin A
Meta-analyses of the trials of vitamin A in less developed countries agree that childhood mortality is reduced when vitamin A is given to groups with mild deficiency (Glasziou & Mackerras 1993). As morbidity from diarrhoeal disease also seemed to be affected, there has been some speculation that vitamin A may be useful in Aboriginal populations where this condition is common. There is little information concerning vitamin A status in Aboriginal children. Various studies in Aboriginal communities have found normal levels of vitamin A (O’Dea et al 1988; Cheek et al 1989; Lee et al 1994b), which may be due to the widespread fortification of margarine with vitamin A in Australia. However, better assessment of vitamin A status may be required among Indigenous children and in some groups of Indigenous adults.

B vitamins
Low vitamin B$_1$ levels have been described for most age and sex groups, but were particularly marked in women of child-bearing age (Lee 1996; Kamien et al 1974; Nobile 1974). Wide ranges of vitamin B$_1$ levels have been described in remote Aboriginal communities (Lee 1996; Lee et al 1994a; Gault 1990). These may be related to seasonally high intake of traditional foods rich in this vitamin (Lee 1996; Lee 1992).

High levels of vitamin B$_12$ have been described for both traditional and ‘westernised’ Aboriginal groups (Lee 1996). These have been attributed to increased levels of vitamin B$_12$ binding protein (secondary to infection) and to liver damage associated with alcoholism. However, it has also been suggested that there is a dietary component and that the high levels may reflect very high meat intakes (Lee et al 1994a).

Levels of vitamin B$_6$ that are low in relation to the ‘normal’ reference range for non-Indigenous Australians have been described in high-risk groups in some Aboriginal communities (Lee 1996; Lee et al 1994a; Davis et al 1975; Kamien et al 1974). However, acceptable levels have been described more frequently (Lee et al 1994a).

Relatively low vitamin B$_2$ status has been noted in one Aboriginal study (Lee 1996; Kamien et al 1974).

Vitamin E
Relative to normal ranges relevant to the non-Indigenous population, low levels of plasma vitamin E have been described in some Aboriginal groups (Lee et al 1994a; Kamien et al 1974; Cheek et al 1989). However, these results may not have been important as the Aboriginal diets tended to be low in polyunsaturated fatty acids and...
an acceptable ratio of serum vitamin E to total lipid ratio has been consistently described in Aboriginal subjects (Lee 1996; Kamien et al 1975).

Folate
Low circulating folate levels (red cells, plasma) are associated with increased levels of homocysteine, which are in turn linked to increased risk of cardiovascular disease (Panchuranti et al 1994). Homocysteine increases the risk of cardiovascular disease through oxidative damage to the vascular endothelium (Jacobsen 1998). Low folate intake is also associated with increased risk of neural tube defects.

Increasing intakes of folate in the early weeks of pregnancy is associated with a reduction in the rate of neural tube defects. There is no information about the folate status of the general Australian population, and the Australian food composition tables do not have data on folate. One study found that serum folate levels in remote Aboriginal communities were low and that this was corrected when the store began to supply more fruit and vegetables (Lee et al 1995b).

Investigation of the folate status of the Aurukun community on the western side of the Cape York Peninsula found low levels compared to residents on the eastern side of the Peninsula, especially during the wet season (Wong & Behm 1991). Estimated dietary intake of folate in the community was low. Staple foods such as damper, bread and rice were the main sources of folate. Green leafy vegetables, a rich source, were expensive and not often available as they are not produced locally and only limited quantities were flown in weekly. Bushfoods, which would augment intake, were also less available during the wet season. The presence of intestinal parasites and excessive alcohol consumption compromised the folate status.

Low folate status is believed to be implicated in the high prevalence of neural tube defects described in Aboriginal infants (Lee 1996; Bower et al 1989).

Phytochemicals
Phytochemicals are bioactive components of plant foods, many of which are also described as micronutrients: carotenoids (antioxidants derived from green, yellow and red vegetables and fruit), phyto-estrogens (estrogen-like activity), and numerous other classes of substances with antioxidant activity.

Carotenoids
Plasma carotenoid concentrations reflect current dietary intake (Olson 1984; Gibson 1990). Compared to the ‘usual’ levels in Caucasians, low carotenoid levels have been reported in Aboriginals (O'Dea et al 1988; Cheek et al 1989), and this probably reflects the low intake of carotenoid-containing fruit and vegetables (Lee 1996). Lee et al (1994a) found betacarotene concentrations in adults improved when there was an increase in the ‘turnover’ of fruit and vegetables in the store.

The circulating levels of vitamin E, vitamin A and the major dietary carotenoids were measured in Aboriginals in a remote community in the Kimberley region (Rowley et al 1997). Preliminary data indicate that the average plasma levels of the carotenoids were well below those seen in healthy Australian subjects and those
reported in other studies. High rates of smoking, particularly among men, probably further compromise carotenoid status. Thus sub-optimal intake of carotenoids may be a marker for the excessive cardiovascular risk seen among Australian Aboriginal people.

**Minerals**

**Iron**

Iron deficiency remains the most common nutrient deficiency in the world (NHMRC 1992a). In infants and children, because rapid growth imposes large iron needs and the bioavailability of iron in the infant’s diet is low (Dallman et al 1980; MacPhail & Bothwell 1989), inappropriate nutrition is likely to play a major role (Lovric et al 1972). In women of child-bearing age, iron loss resulting from pregnancy or heavy menstrual periods may, on a long-term basis, not be matched by dietary iron. In men and in postmenopausal women the major cause of iron deficiency is pathological bleeding. Most commonly, this occurs from the gastrointestinal tract (e.g. peptic ulcers, gastritis, haemorrhoids, tumours). Iron-deficiency anaemia is discussed in Chapter 10.

**Zinc**

Zinc is important for growth and found in many of the same foods as iron. Zinc deficiency is thought to cause a block in protein and nucleic acid synthesis, affecting skin, gastrointestinal and immune cells and causing anorexia (loss of appetite) and a high energy cost of growth. A more recognisable but less specific feature is loss of appetite, which may be a toxic effect of a relative excess of the other nutrients, such as amino acids, needing to be metabolised and excreted. This implies that the anorexia of zinc deficiency depends on a high protein intake (Golden & Golden 1981a; 1981b). Unlike iron, there is no single tissue that gives a good overall summary of zinc status (Gibson 1990).

The high prevalence of anaemia and poor growth in Indigenous children suggests that low intake of zinc may be an important, overlooked factor. Hypozinaemia has been documented in communities in northern Western Australia (Holt et al 1980) and also in South Australia (Cheek et al 1989); this might have been partly due to chronic diarrhoea and malabsorption. However, a later zinc supplementation program was unsuccessful (Smith et al 1985). This throws some doubt over the importance of zinc in overall nutritional status and growth of Aboriginal children.

Recent dietary studies have suggested that zinc intake is high in remote areas where meat forms a substantial proportion of the diet (Lee 1996).
Healthy diet

The Australian Guide to Healthy Eating provides information about the amounts and kinds of food that provide the nutrients essential for good health and well-being. The main food groups are:

- bread, cereals, rice, pasta, noodles;
- vegetables, legumes;
- fruit;
- milk, yogurt, cheese; and
- meat, fish, poultry, eggs, nuts, legumes.

A healthy diet includes enough food from each of these groups every day. Different varieties of foods from within each of the five groups should be chosen from day to day, week to week and at different times of the year. Eating a wide variety of foods has a very positive effect on health. A healthy diet includes:

- plenty of water;
- plenty of plant foods (bread, cereal, rice, pasta, noodles, vegetables and fruit);
- moderate amounts of animal foods (milk, yogurt, cheese, meat, fish, poultry, eggs); and
- small amounts of oils and fats (including margarine) and extra foods (ie snacks, soft drinks, lollies).

Nutrients are classified chemically as macronutrients (protein, carbohydrate, fat, ethanol and water) and micronutrients (vitamins, minerals and trace elements). Deficiencies of micronutrients can also influence health and well-being. Dietary sources of micronutrients that have been found to be deficient in some Aboriginal and Torres Strait Islander populations are given below.

**Vitamin C**
citrus fruits, orange juice, guava, pawpaw, tomatoes, kiwi fruit, broccoli

**Vitamin B<sub>1</sub>**
whole grains, offal (such as liver, kidney), meat, fish, poultry, egg yolk

**Folate**
green leafy vegetables, brussels sprouts, dried beans (ie baked beans), offal (such as liver), root vegetables, whole grains, orange juice, avocado, milk, yeast spreads (ie Marmite<sup>TM</sup>, Promite<sup>TM</sup>, Vegemite<sup>TM</sup>)

**Carotenoids**
dark-green leafy vegetables, carrots, mango, pawpaw

**Iron**
offal (such as liver, kidney), meat, eggs, fish, poultry, leafy green vegetables, dried fruits

**Zinc**
seafood, offal, mushrooms, eggs, meat, whole grains, seeds

**Bushfoods** are good sources of both macronutrients and micronutrients.

FACTORS INFLUENCING NUTRITION

Knowledge is just one factor that may influence dietary change, and it may not be the most significant one. An awareness of the factors influencing food habits is essential for the development of food and nutrition interventions. A framework to consider factors that influence food choices builds on the concepts of availability and acceptability of foods (Fieldhouse 1986) (Table 4.1). These influences, listed below, operate at different stages of the food choice process and their importance to groups will vary.

Table 4.1  Elements of food selection

<table>
<thead>
<tr>
<th>Availability:</th>
<th>Political</th>
<th>Economic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physical</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Land availability</td>
<td>Agricultural policies</td>
<td>Price</td>
</tr>
<tr>
<td>Water availability</td>
<td>Subsidies</td>
<td>Farm costs</td>
</tr>
<tr>
<td>Climate</td>
<td>Business controls</td>
<td>Marketing costs</td>
</tr>
<tr>
<td>Type of soil</td>
<td>Legislation</td>
<td>Packaging</td>
</tr>
<tr>
<td>Pest and plant control</td>
<td>Distribution</td>
<td>Processing</td>
</tr>
<tr>
<td>Transportation</td>
<td>Welfare programs</td>
<td>Transport</td>
</tr>
<tr>
<td>Storage facilities</td>
<td>Rationing</td>
<td>Storage</td>
</tr>
<tr>
<td>Nutrition policies and guidelines</td>
<td>Nutrition policies and guidelines</td>
<td>Consumer demand</td>
</tr>
<tr>
<td>Government-sponsored research</td>
<td>Government-sponsored research</td>
<td>Income</td>
</tr>
<tr>
<td>Trade and aid policies</td>
<td>Trade and aid policies</td>
<td>Patterns of expenditure</td>
</tr>
<tr>
<td>Tariffs and quotas</td>
<td>Tariffs and quotas</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Acceptability:</th>
<th>Individual choice</th>
<th>Socio-psychological</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cultural and religious</td>
<td>Preference</td>
<td>Prestige</td>
</tr>
<tr>
<td>Cuisine</td>
<td>Taste</td>
<td>Status</td>
</tr>
<tr>
<td>Myths</td>
<td>Therapeutic needs</td>
<td>Friendship</td>
</tr>
<tr>
<td>Superstitions</td>
<td>Personality</td>
<td>Communication</td>
</tr>
<tr>
<td>Taboos</td>
<td>Beliefs</td>
<td>Reward and punishment</td>
</tr>
<tr>
<td>Ritual</td>
<td>Personal values</td>
<td>Emotions</td>
</tr>
<tr>
<td>Morals</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Doctrines</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prohibitions</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Source: Fieldhouse (1986).

These factors operate within the food and nutrition system. This links the agricultural system to the health system (Heywood & Lund-Adams 1991) (see Figure 4.1).
Figure 4.1  Relationship of the food and nutrition system to the agricultural system and the health system

![Diagram showing relationships between agricultural system, food and nutrition system, and health system]


Within the food and nutrition system there are four sub-systems, ranging from production of foods and their processing and distribution through to consumption and health consequences (Heywood & Lund-Adams 1991) (see Figure 4.2).

Figure 4.2  The four subsystems of the food and nutrition system

![Diagram showing the four subsystems of the food and nutrition system]


Another way of considering the factors influencing food choices and, in turn, the consequences on nutritional status is by considering the causes of nutrition-related problems (Figure 4.3).
There are socio-economic, geographical, environmental and social factors that may influence food choices. While in remote communities the available information relates directly to Aboriginals, in urban communities the information is not specific to Aboriginal people and relates to factors of relevance to low-income and otherwise disadvantaged groups. Aboriginal and Torres Strait Islander peoples are disproportionately represented in this group.

### 4.1 Socio-economic factors

A recent study examined the relationships between deaths from various diseases and socio-economic status, defined as the index of relative socio-economic disadvantage of the postcode listed on the death certificate. Death data for the entire Australian population aged 25 to 64 years for the period 1985-1987 were divided into quintiles of status (National Health Strategy 1992). The bottom 20 per cent of the population experience higher rates of deaths from a variety of causes, including diabetes, coronary heart disease and cerebrovascular disease. Higher rates of morbidity for similar conditions for groups with low income, education or socio-economic status were noted when the data from the 1989–1990 NHS were analysed (National Health Strategy 1992).

As most Aboriginals and Torres Strait Islanders fall into the lowest group according to these definitions, it must be inferred that their excess disease rates are at least
partly due to their lower status within society. The association between low socio-economic status and higher rates of various diseases in adults has been noted in a number of other studies of non-Indigenous Australians and other populations (National Health Strategy 1992). The prevalence of risk factors such as smoking and obesity is usually higher in the lower socio-economic groups but analyses of similar data in the United Kingdom have shown that most of the excess mortality remains unexplained after differences in these are adjusted for (National Health Strategy 1992). In other words, programs to improve risk factor profiles are not enough.

**Income**

Most Aboriginal people receive their main source of income from government benefits in the form of unemployment benefits, the Community Development Employment Program (CDEP) or government pensions. Government payments are the main source of income for 54.9 per cent of the Aboriginal population over 15 years of age; CDEP income for 8.5 per cent and 10.7 per cent reported no income (ABS 1995).

In 1996, the median weekly individual income for Indigenous males aged 15 years and over was $189, compared with $415 for non-Indigenous males. The disparity among females was not as pronounced, with median weekly incomes of $190 for Indigenous females and $224 for non-Indigenous females (ABS & AIHW 1999).

**Table 4.2 Main source of income for Aboriginal people aged 15 years and over, 1994**

<table>
<thead>
<tr>
<th>Earned income</th>
<th>All income</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-CDEP</td>
<td>CDEP</td>
</tr>
<tr>
<td>Males</td>
<td>30.1</td>
</tr>
<tr>
<td>Females</td>
<td>18.5</td>
</tr>
<tr>
<td>Persons</td>
<td>24.1</td>
</tr>
</tbody>
</table>

**Part of State**

<table>
<thead>
<tr>
<th></th>
<th>Capital city</th>
<th>Other urban</th>
<th>Rural</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td>35.0</td>
<td>1.3</td>
<td>50.8</td>
</tr>
<tr>
<td>Females</td>
<td>23.1</td>
<td>5.8</td>
<td>59.7</td>
</tr>
<tr>
<td>Persons</td>
<td>16.0</td>
<td>18.4</td>
<td>52.1</td>
</tr>
</tbody>
</table>

**Mean Annual Income**

<p>| | | | | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Males</td>
<td>26,689</td>
<td>12,165</td>
<td>8,888</td>
<td>15,448</td>
</tr>
<tr>
<td>Females</td>
<td>21,873</td>
<td>13,002</td>
<td>10,038</td>
<td>12,702</td>
</tr>
<tr>
<td>Persons</td>
<td>24,802</td>
<td>12,403</td>
<td>9,576</td>
<td>14,046</td>
</tr>
</tbody>
</table>

Source: ABS (1996a).

Thirty per cent of Aboriginal adults worry at least occasionally about going without food (ABS 1995). More people who were aged 25–44 years old, lived in rural areas, lived in a household where no-one was working and who were living in households with one or more dependent children expressed concern about food security (ABS 1996a).
In addition to the total amount of available income in the community, there is
evidence that distribution of that income within the community is important.
Women tend to spend available income on market foods. They usually buy for the
household, while men tend to purchase other consumer goods, such as soft drinks or
non-food items (Rowse et al 1994).

Low income combined with high food costs results in Aboriginal and Torres Strait
Islander peoples spending a large percentage of their incomes on food. Increases in
the Remote Area Allowance have not kept up with inflation. The allowance in the
Kimberley region has been found to fall well short of the differential in food costs
between that region and metropolitan areas (Sullivan et al 1987). Recipients of
CDEP, a common means of distributing unemployment benefits in remote
communities, are not entitled to receive the Remote Area Allowance.

Access to banking facilities for money control may also be a very important factor in
remote Aboriginal communities that have virtually no access to banks. Wider access
to EFTPOS facilities may improve this situation. Available income from different
sources (eg CDEP) may be managed through the community's administrative offices.
This often involves an arrangement which makes credit available to the purchaser at
the foodstore checkout. This can be very difficult to administer, for the purchaser and
the store, unless it is very well controlled.

Food access of low-income groups in urban communities

Researchers in the United Kingdom and the United States have suggested that
persons of low socio-economic status experience disproportionate difficulties in their
attempts to procure healthy food (Turrell 1996). These difficulties relate to the
following:

• low socio-economic groups often live in areas where there are few large
supermarkets — as a consequence, these groups are reliant on smaller shops,
which typically stock a limited range of foods, have higher prices and (often)
provide food of a lesser quality; and

• difficulties in obtaining access to large (and often distant) shopping facilities
because they lack private transport or live in areas where public transport is
inadequate or non-existent.

Reduced spending power often forces low income groups to purchase food less
frequently and in smaller quantities. The range and diversity of foods that can be
purchased is limited and the foods bought are very often of a poorer quality (Turrell
1996). The 'Food in Redfern' found that access to fresh food outlets was limited with
food at the most accessible supermarket in the area being more expensive and of
poorer quality (Hodge 1991).

A study of the food and nutrient intake, food purchasing patterns and budgeting
strategies of 29 sole-parent low-income families with dependent children showed that
low-income families, in nutritional terms, can manage well under difficult
circumstances (Crotty et al 1992). Despite the large differences in amounts of money
spent in the first and second weeks of a social security payment period, the nutrient
density of the parents' diets was maintained at a similar level in both weeks. Planning was a major strategy to manage expenditure. Low-income groups had to allocate a much greater proportion of their household income for food, yet they spend significantly less on food per household member (Crotty et al 1992).

4.2 Geographical factors

Food prices

Costs of commodities vary across States/Territories (Sullivan et al 1987; Leonard et al 1994) and are higher in communities that are more remote. Costs for handling and freight of goods to remote communities are high, and few suitable local products are available for purchase (Young 1984; McMillan 1991). Retail prices must allow for high accountancy costs, the purchase and maintenance of capital equipment, wages and salaries and ‘shrinkage’ (Lee 1996; Young 1984).

Factors contributing to the higher costs of food include (McMillan 1991):

- transport — the cost of food in rural and remote communities includes the cost of transporting the product from its point of production to the point of consumption. The mode of transport allows for some containment of cost, with rail and sea transport being cheaper than road or air (although this may be offset by the cost of refrigeration or higher wastage during a longer transit period). Transport of items that require chilling or freezing demands appropriate equipment and part of the cost can be attributed to the cost of buying and maintaining such equipment. Transport costs also include handling costs, with efficient and appropriate handling being accompanied by lower levels of wastage.

- storage — many rural and remote communities receive fresh or frozen produce every one or two weeks and sometimes more infrequently in the wet season. It is therefore necessary to be able to store produce for reasonable periods of time. Many communities now have large cool rooms and freezers to keep produce at optimum temperature and humidity. The capital cost of establishing and maintaining such equipment is substantial and can contribute to some of the cost of the sale price of produce. Where the power supply is unreliable, the further investment in independent power sources also adds to costs.

- wastage — the cost of every kilogram of food that has been ordered by the store but is not sold due to wastage (poor handling, long transit times, poor storage) is added to the price paid by the consumer for other goods that they purchase.

The cost of freight from Darwin to communities with the Arnhem Land Progress Association (ALPA) stores represents approximately 15 per cent of unrefrigerated grocery prices and around 26 per cent of chiller/freezer prices. This 26 per cent is approximately $1 per kilogram. The cost of air transport is much higher, with a minimum $15 charge plus an average $2.50 per kilogram (McMillan 1991).
Transport of perishable foods to remote communities can be long, complicated and expensive. A typical sequence is as follows: supplier’s cool room; unrefrigerated truck; air freight cool room; unrefrigerated van; aeroplane; unrefrigerated utility; retailer’s cool room; and sometimes to unrefrigerated shelves (McMillan 1991). Clearly this is not conducive to high product quality at the point of sale to the consumer. The quality of the food in remote communities is greatly affected by the conditions of transport, in particular, the frequency, duration and temperature of transport.

Market basket surveys
Studies are being conducted to compare food prices in some areas of Queensland, Western Australia and the Northern Territory with those in the capital city of that State/Territory. These surveys vary in the groceries included in the ‘basket’ and while patterns can be compared, prices cannot.

Queensland
The Queensland market basket survey is based on a basket of food needed to feed a family of six for two weeks. This basket provides 95 per cent of energy needs and 70 per cent of recommended dietary intakes (Dympna Leonard, Tropical Public Health Unit, personal communication) (see Figure 4.4).

Figure 4.4 Market basket cost comparisons, Queensland, 1999

Source: Tropical Public Health Unit, Queensland Health.

Another study has shown that fruit and vegetables in the Torres Strait cost about 40 per cent more than those in Cairns (Leonard et al 1994).
Western Australia

The Western Australian Market Basket survey (Sullivan et al 1987) determines the cost of a ‘basket’ of the most popular food purchases needed for a family of five for two weeks — groceries include meat, bread, flour, tea, sugar, fruit, vegetables, eggs, breakfast cereals, cool drink and some toiletries. Cigarettes, alcohol and takeaway foods are not included. The average price of the basket is compared with the ‘basket’ available in Perth (Robyn Bowcock, personal communication Kimberley Public Health Unit) (see Figure 4.5). In the Kimberley community stores, the price of the ‘basket’ ranged from $378 to $580. The average price of the ‘basket’ in Pilbara and Kimberley community stores is 56 per cent more than in Perth (Zakrevsky et al 1996).

**Figure 4.5 Market basket cost comparisons, Western Australia, 1997 and 1998**

* East Kimberley
* Ngaanyatjarra lands
* Pilbara
Hall’s Creek
Broome
Fitzroy Crossing
* Goldfields
Wyndham
* West Kimberley
Kununurra
Derby
Goldfields
Pilbara
Perth

* Community stores.
Source: Robyn Bowcock, Kimberley Public Health unit.

Northern Territory

The Northern Territory Nutritionists’ Market Basket Survey aims to determine the cost of a basket of foods that provides 100 per cent of the nutrients and 95 per cent of the energy required by a family of six for two weeks (Territory Health Services 1998) (see Figure 4.6).

The 1998 Northern Territory Market Basket Survey demonstrated that the cost of the basket of foods was, on average, 62 per cent more in Aboriginal community stores in the Northern Territory than the average cost of $301 in Australian capital
city supermarkets. In particular, the fruit portion of the basket was 70 per cent more expensive and the vegetable portion was 82 per cent more expensive than in capital city supermarkets.

**Figure 4.6 Market basket cost comparisons, Northern Territory, 1998**

<table>
<thead>
<tr>
<th>Location</th>
<th>Cost ($0-$600)</th>
</tr>
</thead>
<tbody>
<tr>
<td>All NT</td>
<td></td>
</tr>
<tr>
<td>Katherine District</td>
<td></td>
</tr>
<tr>
<td>Nhulunbuy District</td>
<td></td>
</tr>
<tr>
<td>Darwin District</td>
<td></td>
</tr>
<tr>
<td>Barkly District</td>
<td></td>
</tr>
<tr>
<td>Alice Springs</td>
<td></td>
</tr>
<tr>
<td>Darwin</td>
<td></td>
</tr>
</tbody>
</table>

Source: Select Committee on Territory Food Prices (1999).

**Remote community foodstores**

There is still an extremely limited range of foods stocked in remote community foodstores, relative to larger rural towns and urban centres. In particular, perishable items such as dairy foods, fruit and vegetables are frequently in short supply (Young 1984; Leonard et al 1994; Lee et al 1994b; Lee 1996).

An important influence on the food supply of remote Aboriginal communities is the store manager (Lee et al 1996a). This has been shown through comparison of the nutrient density of diets in two remote Aboriginal communities using the ‘store-turnover’ method (see Appendix 1) during the periods that three different store managers were responsible for the stores (Lee et al 1996a).

The ALPA is a retail cooperative owned by the Aboriginal residents of five communities in Arnhem Land in the Northern Territory. The ALPA developed and implemented a corporate nutrition policy that has led to dietary improvements in those communities where stores mostly complied with the policy (Lee et al 1996b).

The ALPA offers commercial activities aimed at improving health and nutrition while maintaining commercial viability. Within this context the ALPA has considered the issues that affect the demand and supply of food in remote communities (McMillan 1991). Factors that influence the demand for food include: household income; the price of substitutes (though there is usually a limited choice); strong brand loyalty due perhaps to low literacy levels; products promoted through media campaigns such as popular brand names; and seasonality. Price was not a major factor in determining purchase decision. Factors influencing supply of food to remote communities include: consumer demand; stock management; carrying capacity for chiller/freezer lines; frequency and method of deliveries. Ways to increase the sales of nutritious foods, include in-store promotions, such as shelf ‘talkers’, posters and prominent displays and cross-subsiding the price of fresh fruit and vegetables and cigarettes (McMillan 1991).
A pilot intervention aimed at helping communities to develop a nutrition policy for their community foodstore is currently in progress. *The Store Book: Food and Nutrition Guidelines for Aboriginal Community Stores* (Stronach et al 1996) is designed to be used by a wide range of community development officers working in Aboriginal communities. It gives ideas, information and activities to assist people to improve the variety, quantity, quality and affordability of foods sold in the store. The *Store Book* is an outcome of the Northern Territory Nutrition Policy.

**Agriculture**

Vegetable gardens were cultivated at many Aboriginal settlements in previous years but were often abandoned when the religious missions were disbanded. There is a tradition of community gardens in the Torres Strait Islands. Agriculture as an economic or subsistence basis in Aboriginal communities and outstations is a possibility (Hetzel & Frith 1978). However, the practicalities of doing this in tropical and arid regions need to be carefully considered (Lee 1996; McArthur 1960; MacKenzie 1980).

Some groups in rural areas have decided to cultivate gardens on a subsistence or semi-commercial basis (Latz & Griffin 1978; Uwankara Palyanyku Kanyintjaku 1987). Among many examples of such projects that have been undertaken across the country are the following.

- The Garden Kai Kai project in the outer islands of the Torres Strait seeks to improve food supplies through the establishment of community gardens, combined with workshops by nutritionists and diabetes educators (NHMRC 1997b).

- In Narromine in central western New South Wales, a market garden program has been run and worked by the CDEP participants for about five years. A variety of vegetables are grown on an 8-acre lot, which is available to participants and the community at a significantly reduced rate. The produce is delivered to local community members and also to local hospitals and nursing homes. Participants include older people as well as many young people. There has been a change in the attitudes of both participants and the town's people (personal communication, Coordinator, Narromine CDEP Program).

The worth of such programs in Aboriginal communities cannot be overstated. The benefits extend beyond improving nutritional intake. Growing some food locally reduces dependence on outsiders, helps keep money within the community and also fits within relationship and obligation systems. The programs also address issues of self worth and pride, which are pivotal to improvements in the health of Aboriginal and Torres Strait Islander peoples.

**Torres Strait Islands**

A study was undertaken to investigate factors that could contribute to an improvement in the quality, quantity, availability and affordability of nutritious foods (principally fresh fruit and vegetables) to the people of the Torres Strait Islands. A
substantial proportion of the fruit or vegetables were of poor quality due to the time taken to transport produce, inappropriate transit temperatures, infrequent delivery, lack of chiller display facilities and cost, with fresh fruit and vegetables costing around 40 per cent more than in Cairns.

Issues that need to be addressed to improve local production of fruit and vegetables include:

• raising awareness of the role of fruit and vegetables, in particular the role of traditional staple foods, in protecting health;
• resources and training in appropriate horticultural expertise;
• improved land care and water resource management systems; and
• review of quarantine regulations that restrict movement of planting materials and produce as this stifles household and commercial production.

4.3 Environmental factors
Adequate housing, access to clean water, and the removal of human waste are important factors in achieving and maintaining good health. The absence of any one of these factors can result in a variety of infectious and parasitic diseases (see Chapter 5).

Housing
Although census collectors undoubtedly missed some people living in improvised dwellings due to the transient nature of such accommodation, on census night in 1996 there were over 5,800 households counted as improvised dwellings (ABS 1998d). Of these, some 31 per cent were Indigenous households. Improvised dwellings were more commonly found in rural and remote areas than in urban areas. More than half of all Indigenous households living in improvised dwellings were counted in the Northern Territory, with most of the rest in Queensland and Western Australia (ABS 1998d).

Almost 7 per cent of Indigenous people lived in a dwelling with 10 or more people in 1996, which was almost 50 times greater than the proportion of non-Indigenous people living in such conditions (ABS 1998d).

Water and waste disposal
Access to clean water for washing and drinking and safe sewage and garbage disposal are fundamental to the health of a population. They affect nutritional status by limiting the transfer of diarrhoeal disease and hence controlling the cycle between poor nutritional status and reduced resistance to infection.
The UNICEF indicators for international reporting on the survival, promotion and protection of children are outlined in Table 4.3.

**Table 4.3 UNICEF indicators for survival, protection and development of children**

<table>
<thead>
<tr>
<th>Health goal</th>
<th>Indicator</th>
<th>Comments and actions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Universal access to safe drinking</td>
<td>Proportion of the population with access to an adequate amount of safe</td>
<td>The terms access, adequate, safe and convenient distance should be defined at a</td>
</tr>
<tr>
<td>water</td>
<td>drinking water within or a convenient distance from user’s dwelling.</td>
<td>national level</td>
</tr>
<tr>
<td>Universal access to sanitary means</td>
<td>Proportion of the population with access to a sanitary facility for human excreta disposal in a dwelling or within a convenient distance from the user’s dwelling.</td>
<td>The terms access, sanitary facility, and convenient distance should be defined at a national level.</td>
</tr>
<tr>
<td>of excreta disposal</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>


It should be noted that Table 4.3 only refers to drinking water, however in all parts of Australia an adequate level of washing water is essential. Many infectious diarrhoeal diseases are water washed, not water-borne, hence the availability of water for frequent hand washing is essential to control the spread of disease (Han & Hliang 1989). Overcrowded housing with inadequate levels of hygiene also facilitates transmission of diarrhoeal disease by the faecal-oral route (Gracey 1992; Meloni et al 1993).

Although the parameters described by UNICEF have not been formally defined, the NATIS collected information about housing characteristics. Housing in rural areas had fewer services than in the capital cities with 7.4 per cent of houses without running water connected and 6.3 per cent without access to either a private or communal bath or shower. ‘Access’ seems to be far from universal in the Aboriginal and Torres Strait Islander population, 2 per cent of whom reported that households with facilities had experienced a serious breakdown in the previous month (ABS 1996a). More recently, a survey has shown that in substantial numbers of locations, there was a water supply that failed quality tests, interruptions to electricity supply or leaky sewerage systems in the 12 months before the survey (ABS 2000).

The UNICEF requirement for drinking water does not acknowledge the need for water for gardening. Local horticulture initiatives, the Northern Territory Food and Nutrition Policy and the Queensland Aboriginal and Torres Strait Islander Food and Nutrition Strategy (see Appendix 2) clearly identify local food production as beneficial not only for nutritional purposes but for the opportunity for useful occupation and economic independence. However, community water supplies which have been planned for drinking and washing access cannot provide for either home and community gardens or for semi-commercial ventures.
Impact of health hardware on food and nutrition

Inadequate, and often dangerous, cooking arrangements and lack of cool storage and vermin-safe food storage for perishables can create serious nutritional problems for Aboriginal and Torres Strait Islander people in remote areas (Torzillo et al 1993).

• Appliances designed for the suburban household of four to six people are often used in an Indigenous household of at least 10 people. For example, a refrigerator which is used by the suburban family will be opened mainly in the morning and evening. In Indigenous households the appliance may be opened frequently throughout the day (often in very hot climates) and have the compressor running for most of the 24 hours of each day. Community power supplies are often subject to major surges, which adversely affect electric motors and compressors.

• The large stock pots used for stews for large family groups do not fit standard heating elements on stoves. This results in longer cooking times or inadequately heated food. Sometimes two elements are used for cooking with the pots covering neither. In addition to being inconvenient, spillage and boiling over can be dangerous. As the bottom of pots become uneven with wear, hot spots develop on the elements causing them to burn out.

• Difficulties in paying power bills is an ongoing problem for low-income families. If the electricity supply is turned off, refrigerators and stoves are unavailable, increasing the risk of food spoilage and limiting dietary choices. In many instances this may mean purchasing foods prepared away from the home. These frequently comprise high-fat, low-nutrient density take-away foods, often at greater cost than home-prepared meals. Alternative energy sources for cooking, such as firewood, are costly in urban areas and decreasingly available in some rural areas. The capital cost of generators to run refrigeration is usually beyond the budgets of most rural households.

• Many houses are constructed with only a few open shelves under the sink (if there is a sink) or benches. This can expose foods to physical damage, contamination, infestation by vermin and spoilage by dogs and children. The lack of adequate storage space means that families cannot take advantage of bulk buys to save money as they cannot safely keep food for the duration of its shelf life. The lack of safe storage also means that drugs and supplements cannot be given as a bottleful for people to take home as they cannot be kept out of reach of children. This can lead to poor compliance in taking medication as people may have to go to the clinic several times a week or even daily to receive their medication.

• Community food services often operate from very inadequate premises with limited equipment. Because of concerns about the risk of food-borne disease, providers are often encouraged to limit the type of foods offered for public safety reasons unless food handling and storage is entirely safe. The foods available under these circumstances are often those that are less nutritious. Negotiation to upgrade premises is often a slow process, occurring as community organisational funding permits.
4.4 Social factors

Social factors, such as consumption of alcohol and tobacco, play an important role in the nutritional status of populations. In addition to the direct effect of alcohol and tobacco on nutrient intake, the use and abuse of substances can direct substantial amounts of money away from the purchase of food and other necessities (Lee 1996).

High rates of alcoholism (Brady 1991), petrol sniffing (Brady & Morice 1982), cigarette smoking (Cunningham 1994) and kava drinking (Mathews et al 1988) have been reported in several areas (Lee 1996). Aboriginals suffer disproportionately to other Australians from the physical and social consequences of excess alcohol consumption, tobacco usage, petrol and other solvent sniffing, usage of marijuana, amphetamines, cocaine and heroin, as well as other drugs (Gracey 1998).

One study found that more than 50 per cent of resources spent in one remote community were on tobacco and beer (Hoy et al 1997). This is in contrast to less than 20 per cent being spent on these commodities in the general community (Lester 1994). The proportion of cigarettes and tobacco sales to total sales through ALPA group stores is 15 per cent (McMillan 1991); this does not include expenditure in clubs and other locations. However, many remote communities completely prohibit the sale of alcohol.

Alcohol consumption

Alcohol consumption and misuse contribute to a wide range of health problems in Aboriginal and Torres Strait Islander peoples. These include fetal-alcohol syndrome, poor outcomes of Aboriginal pregnancies, high rates of low birthweight and inadequate parenting and child care. In older children, adolescents and young adults, alcohol consumption and recently altered dietary changes are probably major contributors to risk factors for the ‘lifestyle diseases’ (Gracey 1998).

Alcohol intake of urban dwelling Aboriginal and Torres Strait Islanders was assessed in 1994 as part of the National Drug and Alcohol Strategy (DHSH 1994b). The definition of ‘urban’ was a cluster containing 1,000 people or more and so some of the larger Aboriginal communities in the north were theoretically part of the sampling frame. A smaller proportion of urban Aboriginal and Torres Strait Islander people drink alcohol (62 per cent) compared to the general population living in urban areas (72 per cent from the 1993 survey) (DHSH 1994b).

The NATSIS reported alcohol consumption among 62 per cent of all Aboriginal and Torres Strait Islanders (including those living in non-urban areas) (ABS 1996a). Aboriginal and Torres Strait Islanders who do drink alcohol, consume much higher quantities of alcohol per drinking occasion than the general population, with 68 per cent of current drinkers usually consuming harmful quantities of alcohol (ie more than four standard drinks for females, more than six for males) on occasions when they drink, compared with 11 per cent of the general population (ABS 1996a).

Estimated rates of hospitalisation for alcohol-related conditions are much higher in Aboriginals than in the non-Aboriginal population. Excess deaths attributed to alcohol-related causes include motor vehicle accidents, pedestrian deaths, other
accidents, suicide (particularly in males) and assault (particularly in females). A substantial proportion of alcohol-related deaths is also due to alcohol dependence, alcoholic liver cirrhosis, other gastrointestinal diseases, alcoholic cardiomyopathy, alcoholic psychoses and other physical consequences of acute or chronic alcohol excess (Gracey 1998). It has been suggested that the number of deaths attributed to alcohol is greatly under-reported on death certificates (Weeramanthri & Plummer 1994).

**Cigarette smoking**

Both the 1995 NHS and the NATSIS revealed a high prevalence of smoking among Indigenous adults. Among adults in the NHS aged 18 years and over and living in non-remote areas, 56 per cent of Indigenous males and 46 per cent of Indigenous females said that they currently smoked, compared with 27 per cent of non-Indigenous males and 20 per cent of non-Indigenous females (ABS & AIHW 1999). These results are largely consistent with the NATSIS.

Smokers tend to have poorer quality diets than non-smokers, possibly due to the influence of smoking on taste (Fulton et al 1988; Guest et al 1992a). Examination of the nutrient intakes of smokers and non-smokers using the 1983 National Dietary Survey of Adults and the 1983 Risk Factor Prevalence Survey showed that, for males and females, non-smokers had significantly higher intakes of starch, dietary fibre, vitamin B₁₂, vitamin C, calcium and magnesium than smokers. Smokers had significantly higher alcohol intakes. There was a statistically significant association between smoking status and hazardous intake of alcohol. Smokers have been found to have significantly lower body mass index than non-smokers or ex-smokers (English et al 1997). The NATSIS also found that Aboriginal smokers were less likely to be obese than non-smokers — this does not necessarily indicate better health (Cunningham & Mackerras 1998).

**Artificial sweeteners**

A recent investigation of intense sweetener consumption found that Indigenous people consuming large amounts of artificially sweetened cordial could be exceeding the acceptable daily intake for cyclamates. This finding was based on a survey of only 76 people from six locations and assessed intake on the day before the interview. Like the recommended dietary intake, the acceptable daily intake is intended to be compared to an intake assessed over a longer period of time than a single day. Thus, it is possible that the prevalence of high intakes is, in fact, lower than was found by this survey (National Food Authority 1995). Also, the consequences of excessive intake of artificial sweeteners are arguably of less concern than those of excessive energy intake in Aboriginal and Torres Strait Islander communities.

Many artificially sweetened drinks now use aspartame rather than saccharin or cyclamate. Aspartame is not suitable for use in cooking because heating causes aspartame to be broken down to amino acids which have a very bitter taste. This reaction may also occur in drinks being transported at high temperatures (eg in the tropics and in summer), making artificially sweetened drinks undrinkable in some parts of the country.
4.5 Government regulations

Regulations relating to quarantine, fisheries and National Parks can affect access of Aboriginal people to their traditional foods, and in some places to gardening initiatives. For example, quarantine restrictions in the Torres Strait affect the transport of fruit and vegetables in the Strait (Leonard et al 1995). In the Northern Territory, regulations concerning bag limits for magpie geese do not recognise that a small number of Aboriginal people may be hunting for a large family.
As discussed in Chapter 4, standards of personal, domestic and community hygiene have important influences on Aboriginal health (Torzillo & Kerr 1991), particularly for infants and young children. Poor living conditions may lead to diarrhoeal disease, intestinal parasites, respiratory tract infections, urinary tract infections and infections of the skin, the eyes and the ears, nose and throat (Gracey 1991; Gracey & Anderson 1989; Gracey & Gee 1994). These are often accompanied by growth faltering and sometimes by clinical malnutrition and stunting which may be permanent. Such infections and infestations may also be complicated by nutritional deficiencies as well as impaired intakes of dietary energy. Retarded psychomotor development may also occur as a result of these combined problems in infancy and early childhood.

Diarrhoeal disease (sometimes termed gastroenteritis) is a good example of the way in which unsatisfactory hygiene, exposure to high levels of microbiological contamination of the environment and under-nutrition can combine into a ‘vicious cycle’ in which repeated and sometimes chronic episodes of diarrhoea co-exist and act synergistically to harm the human host (Scrimshaw et al 1968; Mata 1992; Baqui et al 1993). These undermine the nutritional status of the young child (Gracey 1996), with the negative effects of under-nutrition on immune protection making children more susceptible to infections (Kagnoff 1993; Futura & Walker 1997). Episodes of infectious diarrhoea have negative impacts on nutritional status because of stool losses, vomiting, poor appetite, the common practice of withholding of food, and the catabolic effects of infection on the body. Reduced absorption of energy and nutrients by the gastrointestinal tract as well as excessive losses of important dietary components such as carbohydrates, fat and vitamins from the gut contribute to these negative effects.

Studies carried out overseas indicate that the following are major contributors to childhood diarrhoea in areas where environmental contamination may be a significant problem:

- non-exclusive breast-feeding in the first six months of life (Feacham & Koblinsky 1984);
- presence of rubbish in yards, poor housing conditions; and
- non-purification of water (Huttly et al 1987).

Substantial improvements in diarrhoea morbidity can be achieved by exclusive breast-feeding of all young infants and partial breast-feeding of older infants (Feacham 1988), control of fly populations (Cohen et al 1991), hand washing (Han & Hlaing 1989), and by a package of water supply, sanitation, and health and hygiene education (Huttly et al 1990).
5.1 Gastrointestinal infection and infestation

Important causes of diarrhoea in Aboriginal and Torres Strait Islander children include bacterial infections, such as Salmonella, Shigella, Escherichia coli (including strains that cause the ‘haemolytic-uraemic syndrome’), Campylobacter, Aeromonas, enteric viruses including rotavirus, and parasitic agents, such as Giardia, Strongyloides, and Cryptosporidium (Gracey 1992).

A variety of parasites can be involved (Welch & Stuart 1975; Prociv & Luke 1993; Reynoldson et al 1997), some of which might not cause acute diarrhoea but may still be important in terms of their generally negative impacts on appetite and well-being. These include protozoa (such as Giardia intestinalis [G. lamblia] and Entamoeba coli) and helminths (including Hymenolepis nana, Enterobius vermicularis, Strongyloides stercoralis, Trichuris trichiura and hookworms). Giardiasis is a very prevalent intestinal parasite infection in Aboriginal communities, particularly in children between two and five years of age (Boreham 1986; Meloni et al 1993).

Infestations by intestinal parasites can have important nutritional consequences. The popular concept of intestinal parasitic worms ‘stealing food’ from affected children and thus making them thin is an intuitively reasonable explanation of their effect on growth (Hall 1993). However, this does not take into account other important effects, such as loss of appetite, reduced intestinal digestion and absorption, and gut leakage of fluids, proteins and other nutrients.

Hookworm infection is of special importance from the nutritional viewpoint because it can cause gastrointestinal blood loss which leads to iron deficiency and anaemia (Prociv & Luke 1995; Hopkins et al 1997) (see Chapter 10). Hookworm infestations in remote Aboriginal communities in tropical north-west Australia have also been associated with low plasma levels of zinc and elevated levels of plasma copper (Holt et al 1980) where intestinal infections and parasitic infestations were common.

Sources of infection and infestation

Means of spread of these bacterial, viral and parasitic agents include:

- the faecal-oral route (by person-to-person contact);
- unsafe personal hygiene practices (ie not washing hands after going to the toilet);
- contaminated foods — foods can be contaminated with pathogenic microorganisms or their toxins during production, manufacture, cooking, holding (either heated or refrigerated), re-heating, or by the use of utensils, or by food handlers;
- contaminated water supplies — particularly in remote areas because of difficulties with regular water treatment and testing for microbiological cleanliness;
- contaminated utensils, contaminated bedding or clothes; and
diseased animals such as dogs, cats and cattle and, with some organisms, possibly even by airborne droplets or swapping of secretions. Smaller disease-carrying vectors, like flies, are very important, particularly in hot climates and where housing and food preparation areas are unhygienic and exposed to vermin (Gracey 1993).

Maternal hygiene practices are extremely important, particularly for infants and for children up to around five years of age. Mothers have a very important part to play in the hygiene of their families as their understanding of hygiene principles and their hygiene-related behaviours are essential to minimise these problems.

Treatment

Treatment of gastroenteritis depends on clinical judgement, severity of illness, remoteness of the patient from adequate supervision, and the skills of clinical staff and standards of supporting facilities available. Mild episodes usually need referral and transfer to a regional hospital or clinic or, sometimes, to a tertiary hospital (Gracey & Anderson 1993).

5.2 Lactose intolerance

Secondary lactose intolerance

Gastrointestinal infections in infants and young children, particularly those caused by viruses, often cause extensive damage to the delicate mucosal lining of the upper small intestine. This mucosal damage is important, functionally, because the small intestinal mucosa is rich in the enzyme lactase which is needed for the digestion of lactose, the main sugar in human milk and in cow's milk. Lactose is a disaccharide which is composed of two monosaccharides, glucose and galactose. Disaccharides cannot be transferred intact across the gut wall into the body's circulation but must first be digested into monosaccharides which are transported actively into the circulation. This process of intestinal digestion occurs within the small intestinal mucosa through the activity of the enzyme-rich components of the mucosal 'brush border' (Burke 1993).

Extensive damage to the small intestinal mucosa can, therefore, interfere with the normally quick absorption of lactose in the upper small intestine. When this process of lactose digestion and absorption fails, the undigested sugar in the intestinal lumen (tube) provides an osmotic load which draws fluid into the gut and distends the gut wall. This stimulates intestinal peristalsis (motor activity) which results in the passage of frequent loose or watery stools (Gracey & Anderson 1993). This is known, clinically, as lactose intolerance. If this is severe it can cause dehydration which can be serious, even potentially fatal if it is unrecognised and untreated.

If lactose intolerance is prolonged and severe, it can cause significant wastage of nutrients with faecal losses. Combined with the common symptom of anorexia and the widespread practice of withholding feeding from infants and young children with diarrhoeal illnesses, lactose intolerance can lead to malnutrition which can be very difficult to manage. This is particularly so if the lactose intolerant child is in an
environment where recurrent gastrointestinal infections or parasitic infestations are likely.

Lactose intolerance is more likely to occur after episodes of infectious diarrhoea in infants and children who are undernourished. This form of lactose intolerance is known as ‘secondary’ because it is due to an underlying disorder, in this case, gastrointestinal infections which damage the small intestinal mucosa (Gracey & Anderson 1993). This is an important cause of diarrhoea or prolonged diarrhoea in Aboriginal children (Elliott et al 1967; Walker & Harry 1972; Gracey 1973; Gracey 1992), particularly when lactose-containing milks are given to infants and children after an episode of infectious diarrhoea.

Lactose intolerance in ethnic groups

Lactose intolerance is a characteristic which Aboriginals share with many other races or ethnic groups such as Chinese, many other Asian populations, most Africans, Papua New Guineans, Indonesians and African Americans (Scrimshaw & Murray 1988). This contrasts with populations of predominantly Western or northern European origins, including non-Indigenous Australians.

It has not been determined whether lactose intolerance is genetic or environmental in origin. The main arguments are whether being a lactose ‘digestor’ or ‘non-digestor’ is a genetic characteristic, environmentally caused, or due to prolonged milk drinking after weaning. Whichever mechanism applies, the preponderance of the lactose ‘non-digestor’ trait in Aboriginals after three to five years of age (Brand et al 1985; Buttenshaw et al 1990) has important practical implications. It means that Aboriginal people (children and older) past this age are generally intolerant of moderate amounts of milk such as 500 mL of milk, a milk-shake or a carton of flavoured milk of the same volume. Drinking milk in these volumes tends to cause lactose intolerant subjects to have symptoms such as bloating, flatulence, abdominal discomfort (or pain) or diarrhoea. There may also be some subjects, particularly younger children, who could be ‘non-digestors’ of lactose as well as having secondary lactose intolerance because of an episode of gastroenteritis.

Treating lactose intolerance

A study into feeding lactose-hydrolysed milk to slightly undernourished hospitalised Australian Aboriginal infants and children showed better weight gains than if full-cream powdered milk was given (Brand et al 1977; Mitchell et al 1977). The authors of the study suggested that lactose-hydrolysed milk replace normal milk in the supplementary feeding of Aboriginal infants and children (Brand et al 1977). However, the rates and severity of gastroenteritis and malnutrition in Aboriginal infants and young children have decreased very substantially over the past couple of decades so that these suggestions are no longer as relevant as they were in the 1970s.

Furthermore, other approaches to dietary management have become more widely used in recent years. These include the use of locally appropriate solids or weaning foods in a form that is ‘chewable’ and can be easily swallowed by young children, and the use of traditional, local foods in the period after an episode of gastroenteritis.
These foods tend to be well-prepared starchy foods like yams, taro and rice (Bhatta & Hendricks 1996; Gracey 1996). This is particularly important in community-based nutrition programs for Aboriginal infants and children. Starch-containing foods, such as potatoes, sweet potatoes, rice, peas, bananas and mangoes would be appropriate for Aboriginal and Torres Strait Islander children in various parts of Australia. Pasta, including spaghetti, noodles and macaroni, is another rich source of complex carbohydrates and is usually relatively inexpensive.

5.3 Malnutrition and infection

The potential impact of diarrhoeal episodes on nutritional status (through the negative impacts of stool losses, vomiting, anorexia, withholding of food, and the catabolic effects of infection) seem obvious. Diarrhoeal disease episodes are often implicated in growth faltering and under-nutrition in young children in areas where diarrhoea is endemic. Individuals with diarrhoea have excessive faecal losses of nutrients and dietary energy. Chronic diarrhoea can lead to clinically significant intestinal malabsorption with loss of fat and fat-soluble vitamins in the stools (Matia 1992; Guerrant et al 1992). A South American study estimated that the average two to eight diarrhoeal episodes per year experienced by children in developing countries in their first three years of life, causes a cumulative negative effect on body height of 2.5 to 10 cm at 36 months of age. Nutritional supplementation was able to completely offset this negative effect (Lutter et al 1989).

At the community level, diarrhoea does not always lead to restricted growth because young children are capable of ‘catch-up’ growth. However, this does not always occur or is incompletely achieved because of continuing or recurrent intestinal infections, other infections or parasitic infestations, and poor feeding practices (Guerrant et al 1992). These can include stopping of breast-feeding or the withholding of food. Therefore, the importance of diarrhoea as a major cause of malnutrition in children under five years in developing countries has been questioned (Briend 1990). It also seems paradoxical that diarrhoeal prevalence rates and general infectious diseases morbidity have fallen in some places including West Africa (Poskitt et al 1999) and in north-west Australia (Rousham & Gracey 1997) while there have been no detectable improvements in the growth of young children.

The links between diarrhoea and malnutrition are quite complex and may differ from place to place depending on geography, climate, infecting micro-organisms and their transmission (Gracey 1996). Malnutrition generally causes more prolonged diarrhoea and more severe episodes while in some studies there has been a higher incidence of diarrhoea (Lima et al 1992). Malnutrition and impaired immunity were risk factors for diarrhoeal episodes in a community-based study in Bangladesh (Baqui et al 1993). A community-based prospective case-control study, conducted in an Indian village showed that being underweight increased the risk of diarrhoea becoming persistent. The study concluded that prevention of malnutrition and intensive management of acute diarrhoea in malnourished children should help reduce the risk of diarrhoea becoming persistent, that is lasting for 14 days or longer (Bhandari et al 1989).
The relationship between diarrhoea and malnutrition is particularly complex. Distinguishing between cause and effect in this relationship (i.e. between diarrhoea and malnutrition) is difficult because it is potentially bi-directional (Cousens 1991). A hypothetical model shows how, if 'A' causes 'B' and the risk of 'A' varies among individuals, commonly used epidemiological methods may lead to the 'observation' that 'B' causes 'A'. However, this model says nothing about the real association of diarrhoea and malnutrition, which has been studied by many researchers. The results indicate that, with non-uniform diarrhoea rates in a study population, the approach commonly used to investigate this association is unable to determine whether poor nutritional status precedes diarrhoea or vice versa. If diarrhoea rates in the community are heterogeneous, it may appear that poor nutritional status results in increased diarrhoea when this, in fact, is not the case (Cousens 1991).

It was shown many years ago that Aboriginal children with chronic diarrhoea have bacterial contamination of their upper small intestinal contents (Gracey & Stone 1972). This so-called 'contaminated small bowel syndrome' (Gracey 1979) can be associated with intestinal mucosal damage and malabsorption and is sometimes termed 'tropical enteropathy' or 'environmental enteropathy' (Baker & Mathan 1972; Lindenbaum 1973; Tomkins 1981; Fagundes-Neto et al 1984; Rolston & Mathan 1990; Solomons & Bulux 1993; Brewster et al 1997; Menzies et al 1999). This is probably due to living in highly contaminated environments and may be made worse by poor nutrition and reduced gastric acid secretion (Gracey 1981). This may be associated with increased intestinal permeability and losses of nutrients in the faeces.

Impaired intestinal absorption of the sugar, D-xylose, has been shown in populations in tropics and can be used as a marker of intestinal mucosal damage (Hill et al 1981). The measurement of lactose:hannose (L:R) concentrations in urine after the sugars are given by mouth can be used as a non-invasive test of the small bowel mucosal barrier and absorptive function (Bjaranson et al 1995) and reflects mucosal damage in the small intestine (Ford et al 1985). Altered intestinal permeability has been associated with growth faltering in Gambian infants (Lunn et al 1991). Ratios of L:R were much higher in hospitalised Aboriginal children with diarrhoea in Darwin than in Aboriginal children without diarrhoea. The ratios were lower in non-Aboriginal children whether they had diarrhoea or not (Kukuruzovic et al 1999). Unfortunately, the L:R test requires a five-hour collection of urine which severely limits its practical usefulness in remote areas and primary care settings, particularly in girls.

In combination with inadequate and/or contaminated weaning diets (Motarjemi et al 1993), these factors could help explain why some children with diarrhoea, particularly those with persistent diarrhoea, have difficulty in putting on weight for many weeks or months.
Chapter 6 — Involving communities in promoting good nutrition

Involving communities in promoting good nutrition

Nutritional problems in Aboriginal and Torres Strait Islander communities may be very complex and are not due solely to lack of knowledge about contemporary foods (Lee et al 1995b). Such problems will not be alleviated by nutrition education alone but require social and political action within the framework of community development programs (Lee et al 1995b). Previous policies aimed at improving Aboriginal and Torres Strait Islander nutrition were well intentioned but often inappropriate. Reviews of more recent programs have shown that it is important that programs address community priorities and engage communities in all aspects of development, implementation and evaluation and that they use culturally effective structures and processes. Aboriginal and Torres Strait Islander people live in urban, rural and remote locations around the country and programs need to be accessible and relevant to people in all of these areas.

Health programs in Aboriginal communities must address structural and environmental factors and decrease dependency on non-Indigenous organisations (Lee et al 1995b). Cultural and socio-political considerations, housing, environment and public works, education and employment also need to be addressed as they are major contributors to the poor health of Aboriginal people (National Aboriginal Health Strategy Working Party 1989).

6.1 Approaches to Aboriginal nutrition before 1990

Past approaches to nutritional problems in Aboriginal and Torres Strait Islander peoples, although well meant, have suffered from:

- insufficient appreciation of cultural, socio-economic and environmental factors;
- a lack of community ownership and self determination;
- a paternalistic approach to the problem;
- limited appreciation of the Aboriginal holistic approach to well-being;
- disregard for Aboriginal attitudes towards infant feeding and family food distribution; and
- programs being short and poorly funded.

Lessons learnt about approaches to Indigenous nutrition programs from those initiatives undertaken before 1990 include the following.

- Nutrition education programs need to recognise factors including the greater appeal of a ‘familiar’ diet, peer group pressure, budgeting considerations and poor self-esteem supported by ‘the psychology of the history of community failure’ (Sykes 1977).
• While past nutrition programs tended to focus on children, it would appear to be more appropriate to work with Aboriginal adults, particularly elders and key community members (Lee 1992). It has been argued that the traditional self-management of health problems within the extended family groups needs to be revived (White 1977), particularly as the family represents the only security Aboriginal people are likely to know in present-day Australia (Wadsworth 1984).

• The role of Aboriginal women in the health system has been overlooked and trivialised and should be recognised in health programs (Bell 1983; Gale 1983). Early reports indicated that mothers did have a ‘working’ knowledge of European foods (Tilley 1974) and could repeat European nutrition concepts (Stacy 1975). There appeared to be no lack of European nutrition knowledge, rather a lack of internalisation and lack of opportunity to act on that knowledge.

• The potential impact of the community foodstore has frequently been overlooked in medically orientated nutrition programs. ‘The store manager is a very important person. If he is unwilling to help by stocking and encouraging people to buy better foods, little will change’ (Rowley & Rowley 1978).

• Cross-cultural communication is essential to the development of effective health programs (Lester 1974). Certain qualities and communication skills would appear to be necessary in non-Indigenous health professionals working in a cross-cultural situation (Hamilton 1974). However, the transient nature of non-Indigenous health professionals working in Indigenous health in relatively remote areas has generally precluded the development of long-term individual relationships (Rebgetz 1985).

• Non-Indigenous health professionals need to be aware of several specific traditional values and beliefs that may affect the effectiveness and the evaluation of health and nutrition programs in remote communities. Such beliefs may preclude some forms of biological sampling in traditionally orientated communities.

• A holistic approach to health, incorporating Aboriginal and Torres Strait Islander beliefs concerning the inter-relationship of individual health and concepts of the role of land, religion and social relationships should be fostered (DCSH 1987).

6.2 Evaluation of Aboriginal nutrition programs

Programs aimed at improving the general nutritional status of Aboriginal communities have rarely been systematically applied or evaluated (Sykes 1977; Patterson 1987). There have been few well-conducted controlled intervention trials to examine the effectiveness of interventions to improve nutritional health in Aboriginal populations (Butlin et al, unpublished).

Claims that death rates and ill-health amongst Aboriginal and Torres Strait Islander peoples have improved since the establishment of a preventive health program by the Queensland Government (Musgrave 1984) have been publicly criticised (Grunseit 1984; Thomson 1984). Similarly, improvements in growth rates of Aboriginal children in Sydney have been said to be the result of the ‘many factors initiated by
the Aboriginal Medical Service' particularly 'nutrition and the emphasis on preventive medicine' (Brand et al. 1978). Simple association with changing health indicators is not necessarily proof of the efficacy of specific programs. Controls and specific evaluation techniques need to be applied.

Changes in dietary intake and health indicators of nutritional status would appear to be the most appropriate measure in evaluating the success of nutrition programs, as improvements in morbidity data will occur only in the long term. Evaluation of intervention projects usually requires a comparative measurement of pre- and post-intervention change (Lee et al. 1995b).

Several recent reports have noted that there is still a lack of well-evaluated nutrition/health programs for Aboriginal and Torres Strait Islander people (Butlin et al., unpublished; Couzos et al. 1998). Without evaluation results, limited information is available to assist health service providers to decide on effective nutrition projects for their community. The resource, Thinking, Listening, Looking, Understanding and Acting as You Go Along: Steps to Evaluating Indigenous Health Promotion Projects (Colin & Garrow 1996) assists health workers to develop a culturally relevant evaluation in the context of sound health promotion principles outlined in the Ottawa Charter (WHO 1986) and recommendations of the National Aboriginal Health Strategy (National Aboriginal Health Strategy Working Party 1989).

6.3 Promoting the health of Aboriginal and Torres Strait Islander peoples

Existing national programs such as community housing, community education and community anti-alcohol programs have provided potentially useful tools to assist Aboriginal and Torres Strait Island communities to improve their health. However, there has been a lack of emphasis on enabling communities to use programs in ways that fit with their local perceptions and needs. Experience shows that, to succeed, such programs must use culturally effective structures and processes to enable significant involvement and control by Indigenous people (NHMRC 1997a). There is overwhelming evidence that people are more committed to initiating and maintaining changes that they helped to design or adapt to their own purposes and circumstances (Green & Kreuter 1990).

The medical model that has been used to define causes of illness and to guide the development of interventions is not appropriate for Aboriginal and Torres Strait Island people. A holistic understanding of health and its causes takes into account the interaction between health and well-being of individuals and their living conditions (NHMRC 1997a). This concept is now being accepted as a better model.

The history of Indigenous people in Australia continues to determine their health status today. Government policies that have aimed for segregation, protection, assimilation, and/or self-determination have failed to deliver the essential requirements for long-term improvements in Indigenous health — access to safe water; adequate housing; nutritious affordable food; appropriate storage systems; and well-maintained sewage and garbage disposal (Gray et al. 1995).
Reviews of health programs

A number of reviews of health programs for Aboriginal and Torres Strait Islanders have been undertaken.

Promoting the Health of Indigenous Australians

The NHMRC’s Health Advancement Standing Committee undertook a comprehensive review and analysis of past and current health promotion initiatives in Australia. A specific consultation with Aboriginal and Torres Strait Islander peoples was established as a major component of the review. The consultation with Aboriginal and Torres Strait Islander health personnel confirmed the need for (NHMRC 1997a):

- health policy that focuses on improving the health of Australia’s Indigenous population (in addition to providing high quality health-care services);
- partnerships among Indigenous people and organisations, with non-Indigenous people, with the mainstream health system, and with sectors other than health;
- monitoring and surveillance to identify problems and to measure progress;
- research that identifies the reasons that problems are occurring and that helps to select appropriate solutions;
- workforce development to ensure that the people who are responsible for delivering programs have access to training, ongoing education, career paths, and professional support networks;
- effective systems to deliver programs at national, State/Territory and community levels;
- active engagement with other sectors (e.g., housing, education, local government);
- evaluation to identify ‘what works’ and the extent of overall effort to promote health;
- a formal, transparent process to identify priorities and to allocate resources; and
- mechanisms and forums for effective community participation.

As part of the review, nine case studies were undertaken with the aim of highlighting the range of positive actions that are occurring and to identify some principles of good practice. In every case, Aboriginal or Torres Strait Islander health workers played key roles in working with communities to identify concerns and to establish whether people wanted to take action. Agreement to act came from within communities rather than from ‘above’ or outside. The programs were designed to suit the needs and circumstances of individual communities and were managed by Aboriginal/Torres Strait Islander health workers or health teams. Aboriginal and
Torres Strait Islander health workers play a pivotal role in getting people to participate in programs, liaising between the community and the health system and acting as interpreters.

The partnership between Indigenous and non-Indigenous health workers appeared to have been particularly important in each case, with each respecting and needing the other's support, knowledge and skills and with each being willing to share these. All the programs used statistics in their development and evaluation. The use of statistics was introduced and explained by the Aboriginal and/or Torres Strait Islander health workers to community members.

Key aspects to the success of the case studies were:

- development of effective working partnerships— Aboriginal and Torres Strait Islander health workers worked in partnership with their communities and in partnership with non-Indigenous health workers or organisations;

- support— the projects received considerable 'support-in-kind' including salaries, cars and funds for tools, the production of resources, hire of equipment and venues;

- community input— all resources were produced with input from the community so that people felt a sense of ownership;

- key roles played by Aboriginal and Torres Strait Islander health workers— their knowledge of community issues ensured that the real problems facing people in their everyday lives were considered when the program was being developed. The Indigenous health workers have ensured that there has been effective communication among all the groups involved; and

- culturally effective evaluation

Another positive outcome of one of the case studies was the recognition and growing respect that has been shown towards the elders for their wisdom and energy.

Barriers to good nutritional health identified in the nutrition promotions included:

- the high cost of food and the limited choice;

- salad, green vegetables and fruit are expensive and difficult to keep fresh; and

- Aboriginal and Torres Strait Islander people did not see their health as a priority, tending not to ask for help until faced with a crisis — many Aboriginal and Torres Strait Islander people associate hospitals and medical attention with death, which deters them from attending mainstream health-care services.
Food and Nutrition Programs for Aboriginal and Torres Strait Islander Peoples

A critical review of the evidence for successful food supply and nutrition programs for Aboriginal and Torres Strait Islander people commissioned by the Commonwealth Office of Aboriginal and Torres Strait Islander Health Services (Butlin et al, unpublished) found that few nutrition programs were adequately evaluated.

Projects which did or had the potential to provide good quality evidence shared several features:

• community involvement and support in all stages of the project;
• supporting the empowerment of the community rather than imposing priorities on the community;
• multi-faceted interventions;
• monitoring and providing feedback on progress to participants; and
• modifying strategies according to need or case manage so that few strategic options are excluded.

Projects that provided the best examples of effective practice commenced with examination of the food supply system with interventions based on the outcomes of community consultations. These included some level of intersectoral input into the project.

Gaps in food and nutrition programs included:

• few public health programs specifically targeted urban communities;
• there was little evidence that mainstream nutrition programs addressed the nutritional needs of Aboriginal and Torres Strait Islander peoples; and
• little published information was available on programs and projects targeting Aboriginal and Torres Strait Islander communities.

Sharing Good Tucker Stories: A Guide for Aboriginal and Torres Strait Islander Communities

*Sharing Good Tucker Stories: A Guide for Aboriginal and Torres Strait Islander Communities* (Bear-Wingfield 1996) describes eleven food and nutrition programs. The community studies suggest that the way to good nutritional health for Aboriginal and Torres Strait Islander people is ensuring that:

• good food is available at affordable prices and people choose to buy these foods;
• people undertake regular enjoyable physical activity and control their weight; and
• local culture is affirmed and strengthened and that people feel proud of themselves and their community.
Aboriginal food and nutrition policies

The Northern Territory (Territory Health Services 1995), Queensland (Queensland Health 1995) and Western Australian (Office of Aboriginal Health 1998) health departments in consultation with Aboriginal health workers, communities and community organisations have developed Food and Nutrition Policies/Strategic Plans specifically addressing the poor nutritional health status of Aboriginal people (see Appendix 2).

A range of strategies and approaches have been used for implementation of the Queensland policy since its development in 1995. The Western Australian policy now has an intersectoral advisory group which is developing the Program Plan. The Northern Territory is funded and has been implemented for five years.

All policies identified priority areas and included goals (objectives) and potential strategies. Priorities identified in all Policies included: food supply; nutrition programs/education; and food and nutrition information systems. These policies reflect the complexity of the problem, the need for an intersectoral approach to improve the nutritional health of Aboriginal people, and contemporary approaches to this problem.

Aboriginal and Torres Strait Islander peoples have been identified as subgroups for consideration within the overall nutrition policy in some States.
Minjilang — a successful health and nutrition project

- A successful health and nutrition project was initiated by the people of a remote Aboriginal community (Minjilang) in the Northern Territory (Lee et al. 1994a; Lee et al. 1995b; Yarmirr & Bonson 1996). The project aimed to assess and reduce the risk factors for coronary heart disease and related conditions in the community. Dietary intake was assessed by the store-turnover method (see Appendix 1). Biochemical, anthropometric and haematological indicators of health and nutritional status were measured before the intervention and at three-monthly intervals during the intervention year.

- Intervention strategies were planned in consultation with the community to correct observed nutritional problems. Physical activity was encouraged. Traditional and contemporary beliefs and attitudes related to health and nutrition were used to build on existing knowledge. Specific strategies included direction of the project by community leaders, the employment of an Aboriginal research assistant and community members, prompt feedback of individual and community results following each period of clinical and dietary monitoring, the provision and promotion of a wide variety of nutritious foods in the store, and the use of ‘shelf-talkers’ to aid recognition of ‘target’ foods.

- Throughout the study, the intervention strategies were modified in the light of community reactions to results and progress. It was concluded that when community members are themselves involved in all stages of the development, implementation and evaluation of a nutrition-intervention project, improvements in nutritional health are possible (Lee et al. 1994a).

- After the intervention, there were significant improvements in levels of cholesterol, folate, vitamin B₆ and vitamin C. Blood pressure levels and weight of participants had also improved. These improvements corresponded with a decrease in dietary intake of sugar and saturated fat and an increase in micronutrient density.

- A three-year follow-up evaluation showed that the program produced lasting improvements in dietary intake of most target foods (including fruit, vegetables and wholegrain bread) and nutrients (including folate, vitamin C and vitamin B₆). Sugar intake fell in both communities before the program, but the additional decrease in sugar consumption during the program at Minjilang ‘rebounded’ in the next year. Dietary improvements in the comparison community were delayed and smaller than Minjilang (Lee et al. 1995b).

- Although an effective partnership between community members and those providing technical support was critical to success, the ‘ownership’ of the program remained firmly within the community. Members of the community were involved actively in all stages of development, implementation and evaluation. Such community control and participation is essential to strengthen community ‘competence’ and is critical to the fulfilment of self-determination for Aboriginal people.

- Effective strategies were those targeted to community needs. These strategies brought about structural change to make healthier food choices easier by providing and promoting a wide range of good quality nutritious foods. Community members were also assisted to make informed health decisions and develop the confidence to take long-term control over factors impinging on their health.
SECTION II

MATERNAL AND CHILD HEALTH
Birthweight is probably the most important factor affecting neonatal mortality, postnatal infant mortality and infant and childhood morbidity (Kramer 1987). Recent evidence suggests that low birthweight, that is, a birthweight of less than 2,500 g, has long-term effects on the body, being associated with increased risk of Type 2 diabetes, high blood pressure and cardiovascular disease in adulthood (Barker 1995) (see Subsection 1.2).

Birthweight is influenced by duration of gestation and intrauterine growth rate. Low birthweight can be caused by either a short gestation period (<37 weeks) or intrauterine growth restriction (also termed ‘small-for-dates’ or small-for-gestational-age’), or a combination of both (Kramer 1987).

Nutritional determinants of intrauterine growth restriction include low maternal dietary energy (kilojoules) intake, inadequate weight gain during pregnancy and low pre-pregnancy weight. The causes of preterm delivery are less well understood (Kramer 1987). Low birthweight due to preterm delivery is related primarily to reproductive history (Sayers & Powers 1997). Preterm delivery does not always result in a low birthweight baby. Table 7.1 shows factors associated with low birthweight grouped by the type of low birthweight that they affect.

**Table 7.1 Established determinants of intrauterine growth restriction and preterm delivery**

<table>
<thead>
<tr>
<th>Intrauterine growth restriction</th>
<th>Preterm delivery</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Direct</strong></td>
<td></td>
</tr>
<tr>
<td>Infant sex</td>
<td>Pre-pregnancy weight</td>
</tr>
<tr>
<td>Race/ethnic origin</td>
<td>Prior preterm delivery</td>
</tr>
<tr>
<td>Maternal height and pre-pregnancy weight</td>
<td>Prior spontaneous abortion</td>
</tr>
<tr>
<td>Paternal height and weight</td>
<td>In utero diethylstilboestrol exposure</td>
</tr>
<tr>
<td>Maternal birthweight</td>
<td>Bacterial vaginosis</td>
</tr>
<tr>
<td>Parity</td>
<td>Maternal diabetes</td>
</tr>
<tr>
<td>Prior low birthweight infant</td>
<td>Cigarette smoking</td>
</tr>
<tr>
<td>Gestational weight gain</td>
<td></td>
</tr>
<tr>
<td>Energy intake</td>
<td></td>
</tr>
<tr>
<td>General morbidity</td>
<td></td>
</tr>
<tr>
<td>Maternal infections</td>
<td></td>
</tr>
<tr>
<td>Cigarette smoking</td>
<td></td>
</tr>
<tr>
<td>Alcohol consumption</td>
<td></td>
</tr>
<tr>
<td>Tobacco chewing</td>
<td></td>
</tr>
<tr>
<td><strong>Indirect</strong></td>
<td></td>
</tr>
<tr>
<td>Maternal age</td>
<td>Maternal age</td>
</tr>
<tr>
<td>Socio-economic status</td>
<td>Socio-economic status</td>
</tr>
</tbody>
</table>

**Sources:** Kramer et al (1992); Sayers & Powers (1997).

Of the nutritional factors shown in Table 7.1, only low maternal pre-pregnancy weight is associated with preterm delivery while intrauterine growth restriction is associated with low maternal height and maternal pre-pregnancy weight and poor
weight gain during pregnancy. Studies into the effects of diabetes during pregnancy have shown an increased rate of preterm delivery — three-fold in a group of Canadian women (Kramer et al 1992) and four-fold in Australian Aboriginal women (Sayers & Powers 1997).

7.1 Extent of the problem

In 1994–1996, the proportion of low birthweight among babies born to Aboriginal and Torres Strait Islander mothers was almost twice that of babies born to non-Indigenous mothers (12.4 per cent compared with 6.2 per cent) (Day et al 1999) (Figure 7.1). There is still some discussion about how much of this may be due to preterm delivery rather than intrauterine growth restriction (Humphrey 1996; O’Connor & Bush 1996).

Figure 7.1 Low birthweight births by maternal identification of racial status, by State/Territory, 1994-1996

The reported prevalence of Aboriginal low birthweight is varied, ranging from less than 7 per cent in Victoria (ABS & AIHW 1997) to more than 20 per cent in parts of northern Australia (Markey et al 1998). Figure 7.2 shows the prevalence of low birthweight Indigenous babies by Aboriginal and Torres Strait Islander Commission (ATSIC) region compared to all Australian births.

Figure 7.2  Proportion of low birthweight babies born to Indigenous mothers, by ATSIC region, 1991-1996

Note: Unknown and interstate births have been excluded. All-Australian births are from 1994.

Low birthweight is only one part of the birthweight distribution. As shown in Figure 7.3, there are also differences in weight between Indigenous and non-Indigenous infants in the area above 2,500 g. There is a greater proportion of Indigenous infants than non-Indigenous infants in the 2,500–2,999 g category and a smaller proportion with weights over 3,500 g. Improving the birthweight of Indigenous infants requires shifting the entire distribution up, not solely improving the weights of those in the low birthweight range. This means that programs would generally need to include all pregnant women and not only target those at high risk of having low birthweight infants.

There is a trend towards higher birthweights in Aboriginal babies. Between 1986 and 1995, the proportion of low birthweight babies in the Northern Territory decreased while the proportion of non-Indigenous babies of low birthweight did not change significantly (Markey et al 1998). It is unlikely that this improvement arises from more Aboriginal women identifying themselves as such, because absolute numbers of low birthweight babies also fell over this period (Markey et al 1998).

The Torres Strait ATSIC region has the lowest proportion of low birthweight infants (see Figure 7.2). Among Aboriginal infants, the prevalence of low birthweight is lowest in the metropolitan areas and increases with remoteness. The prevalence of birthweights 4,000 g or higher decreases with remoteness. This is consistent with an overall downward shift in the distribution of birthweight with increasing remoteness. The pattern in Torres Strait Islander babies is different. The prevalence of both low and higher birthweight is greatest in the metropolitan areas and lowest in the remote locations. By contrast, there is little variation across Queensland in the birthweight patterns in non-Indigenous infants (Figure 7.4).
Figure 7.4 Prevalence of high and low birthweight babies in infants of Indigenous mothers, Queensland, 1996-1998

![Prevalence of high and low birthweight babies](image)

Note: ‘Capital city’ includes other metropolitan; ‘Rural’ includes rural centres and areas; and ‘Remote’ includes remote centres and areas. These data are preliminary and may be subject to change. People identifying as both Aboriginal and Torres Strait Islander have been excluded.

Source: Perinatal Data Collection, Queensland Health, special tabulation 2000.

As birthweight is inversely related to mortality it might be assumed that the higher overall weights in the Torres Strait Islander babies are a sign of better survival compared to Aboriginal babies. However, birthweight is only one factor influencing mortality. After allowing for differences in the birthweight patterns, Torres Strait Islander babies have a higher neonatal mortality rate than Aboriginal babies (Coory 2000). This suggests that different strategies are needed to reduce neonatal mortality in the two Indigenous groups.

### 7.2 Risks and associated factors

There are numerous factors associated with increased risk of low birthweight in Indigenous babies. Factors associated with a higher birthweight include maternal height (>165 cm), increased parity and maternal diabetes (Blair 1996).

Aboriginal mothers often have multiple risk factors that may contribute to low birthweight and impaired growth of their babies in the first two years of life (Gracey et al 1992). These include anaemia, genitourinary tract infections, poorly controlled blood pressure, proteinuria, inadequate maternal weight gain during pregnancy, young maternal age, cigarette smoking and alcohol consumption. Social class differences have also been demonstrated to be important determinants of health including prematurity and low birthweight (James et al 1997).

A prospective study of 49 Aboriginal mothers and their babies in the Kimberley region of Western Australia showed that satisfactory birthweight and growth in the first two years of life were associated with maternal health during pregnancy, regular antenatal supervision and avoidance of alcohol consumption or cigarette smoking, as
well as personal and family hygiene (Gracey et al 1992). Other studies have found that increased risk of low birthweight exists within communities that have Indigenous populations, lower socio-economic status, that are in the tropics, are remote from main population centres (Dugdale et al 1994), and have poor antenatal attendance (Algert et al 1993; de Costa & Child 1996).

**Under-nutrition in pregnancy**

An important study of risk factors for low birthweight in Aboriginal babies in Darwin showed that infants born to mothers with a body mass index of less than 18.5 (see Table 11.1) had five times the odds of having low birthweight and more than double the odds of intrauterine growth restriction (Sayers & Powers 1997). Low birthweight and intrauterine growth restriction were much less likely in infants of women with a body mass index greater than 25.5. Because pre-pregnancy weights were not available, it is not possible to comment on the relative contributions of pre-pregnancy weight and weight gain during pregnancy. The combination of these odds with the prevalence of maternal malnutrition suggests that 28 per cent of low birthweight and 15 per cent of intrauterine growth restriction could be attributed to continuing poor nutrition of Aboriginal women in the Darwin health region (Sayers & Powers 1997).

The effect of under-nutrition during pregnancy varies according to the stage of development of the fetus. For example, under-nutrition during early pregnancy restricts fetal growth so that the baby is small yet has normal body proportions. By contrast, under-nutrition in late pregnancy alters the body proportions of the fetus and the relative sizes of internal organs.

The effects of under-nutrition before and after birth are also quite different. A developing fetus exposed to under-nutrition is born small and tends to stay small throughout adult life. That is, it stops growing at the normal chronological age and so does not ‘catch-up’ during childhood. Babies who are exposed to under-nutrition after they are born tend to have delayed growth and development. They may continue to grow after the normal chronological age and so may have catch-up growth incorporated into their eventual, attained adult body size.

**Maternal malnutrition**

Malnutrition before and during pregnancy can adversely affect the outcomes of pregnancy. A meta-analysis of 25 datasets about 111,000 births world-wide showed that attained weight indicators from pre-pregnancy through nine (lunar) months of pregnancy were inversely associated with high rates of low birthweight and intrauterine growth restriction (Kelly et al 1996). In women of below average pre-pregnancy weight, the strongest predictor of intrauterine growth restriction was provided by attained maternal weight at seven months. From a practical point of view, a single measurement of attained weight at five or seven lunar months (16–20 or 24–28 weeks) is the most useful screening tool for low birthweight and intrauterine growth restriction in most primary health-care settings. This should be used as a warning of the need for appropriate intervention.
Other individual studies have shown different impacts of maternal health and nutrition on weight at birth. A study from the Central African Republic showed that maternal pre-pregnant weight, representing long-term nutritional status, accounted for 13 per cent of the variance in birthweight. By comparison, weight gain explained only 5.6 per cent of the variance (Andersson & Bergstrom 1997).

The timing of low weight gain, if it occurs, appears to be important. Low weight gain in the second trimester is associated with approximately double the risk of intrauterine growth restriction and low weight gain in the third trimester with a slightly lesser risk while low weight gain in the first trimester was not influential (Strauss & Dietz 1999). This means that there should be more awareness of the importance of maternal weight gain in later pregnancy as a risk factor for intrauterine growth restriction. Even in well-nourished populations, poor nutritional status of mothers can predispose to growth restriction in utero and low weight at birth (Kirchengast & Hartmann 1998). Women who are shorter also have an increased risk of delivering low birthweight babies (Abikusno & Novotny 1996–97; Lawoyin 1997).

A reason that the risks of maternal malnutrition on birthweight appear to be so different in published studies is that there are so many factors that can influence birthweight, apart from the mother’s anthropometric characteristics or weight gain during pregnancy (Table 7.1). Standards of antenatal care given (Gracey et al 1992), and maternal glucose, insulin and lipid metabolism (Catalano et al 1998) are also important influences. Maternal anaemia is important and must be prevented and/or treated during pregnancy. It is also very important to improve the nutritional status of girls so that full height potential is achieved (Lawoyin 1997) and to discourage cigarette smoking.

Intervention studies using protein/energy supplementation

The above observations indicate that certain interventions in pregnant women might lead to improvements in fetal growth, although results have not always been those expected. Studies into protein and energy in pregnant women comparing the effect of a supplement versus no supplement or placebo have been undertaken in a number of countries. Overall, the results are somewhat disappointing. Considering only randomised controlled studies, Kramer (1993) concluded that a balanced supplement of protein and energy caused an increase of 21 g per week in maternal weight gain, an increase of 30 g in birthweight, a non-significant increase of 0.2 cm in birth length and no difference in head circumference. Despite this, there was a reduction in both intrauterine growth restriction and preterm birth, both having odds ratios of about 0.8 but neither were statistically significant. The mean net increase in energy intake achieved was estimated to be 1 MJ/day or less.

Two of the three randomised trials of nutritional advice during pregnancy did not report birthweight outcomes but reported a mean increase of less than 500 kJ/day in maternal energy intake. The third trial found a small increase of 15 g in birthweight in infants of women receiving advice. By contrast, the infants of women randomised to a high protein, isoenergetic supplement were significantly lighter than control.
infants (−64 g), and their mothers gained less weight than the control mothers (Kramer 1993).

Earlier observational studies had suggested that birth outcomes such as perinatal mortality would be better if overweight women restricted their weight gain during pregnancy (Naeye 1979). However this was not found in trials testing this theory. Kramer (2000) examined trials of restricting the intake of obese pregnant women to between 1,200 and 2,000 kcal/day. There was a statistically significant reduction in birthweight of 153 g in the women who were dieted. There was no reduction in the incidence of pregnancy-induced hypertension or pre-eclampsia although there was a non-significant reduction in the preterm delivery rate.

To summarise, increasing maternal intake by about 1MJ using a balanced diet containing a range of nutrients will cause a small increase in infant birthweight, probably in the vicinity of 30 g. At present, there is no evidence that increasing intake in underweight women will have a greater effect on increasing birthweight than for normal weight women. Advising obese women to restrict their intake, or any pregnant women to increase protein alone will decrease the birthweight of their infants.

Maternal age

The role of underweight in affecting birthweights among Aboriginals may seem surprising as there is a high prevalence of obesity in some sections of the Indigenous population. However, 60 per cent of deliveries occur in women aged less than 25 years (Day et al 1999) and this group also has the highest prevalence of underweight. There is a wide variation in the weight status of Indigenous women in different parts of Australia which makes generalisations risky.

Socio-economic status

Low birthweight is also associated with low socio-economic status (Villar & Belizan 1982). This is an important factor as Indigenous Australians are seriously disadvantaged in comparison with other Australians. Low birthweight is prevalent in low-income families in the United States and contributes to low length-for-age (eg reduced stature or stunting) in later childhood. There is a link between low birthweight and impaired growth even through adolescence and into adulthood but it is possible that ‘low birthweight may be a proxy for other factors that also affect nutritional status and growth’ (Gayle et al 1987). This is likely to be so for Aboriginal children who often live in impoverished and unhygienic circumstances and who have high rates of infections and parasitic diseases which are likely to be major causes of their impaired growth (Gracey et al 1983; Martorell & Habicht 1986).

Access to services

In a recent study, Najman et al (1994) examined the obstetric outcomes of Aboriginal women living in urban centres, having access to conventional medical services and living in an environment similar to that of non-Aboriginals of low socio-
Cigarette smoking in pregnancy

Cigarette smoking is another important negative factor on intrauterine growth and birthweight (Salmeron et al 1997). Infants born to mothers in the Darwin health region who smoked more than half a packet of cigarettes a day had almost three times the risk of having low birthweight and more than one and a half times the odds of intrauterine growth restriction (Sayers & Powers 1997). The perinatal collection in the Northern Territory now includes information on smoking.

The high proportion of women with unknown smoking status (Figure 7.5) shows that there are still some problems with collecting this information. Infants of women who smoked at 36 weeks of pregnancy were smaller than the infants of non-smokers. It is also clear that although smoking is an important difference, it is not the only factor leading to different average birthweights between the infants of Indigenous and non-Indigenous women in the Northern Territory.

Alcohol consumption in pregnancy

Alcohol is teratogenic, that is, it can cause birth defects in the developing embryo or fetus (Warren & Bast 1988; Jacobson et al 1993). Clinically, the possible outcomes of alcohol during pregnancy include subtle neurological disturbances, spontaneous
abortion, preterm delivery, stillbirth and major malformations (NHMRC 1992b). Fetal alcohol syndrome is a major consequence of alcohol use in pregnancy. The main features of this syndrome are:

- pre- and postnatal growth restriction;
- neurological abnormalities (intellectual impairment, developmental delay and behavioural disorders); and
- typical facial features such as flattening of the mid-facial area, under-developed jaw and thin upper lip (Day 1992; Jacobson et al 1993).

Microcephaly (a small head in relation to the rest of the body) and congenital heart disease are also common.

Opinion has been divided as to whether there is a safe level of alcohol consumption during pregnancy (NHMRC 1992b). The NHMRC guidelines, which are currently under review, recommend that women who are pregnant or who might soon become pregnant:

- may consider not drinking at all;
- if they do drink, should reduce their drinking to a minimum and not drink on a regular basis;
- should not have more than one standard drink on any one day; and
- should never become intoxicated.

Low consumption (one or two standard drinks per week) has been associated with an increase in spontaneous abortion (Kline et al 1980) and drinking 20 g of alcohol (two standard drinks) or more during mid-pregnancy and binge drinking of five or more drinks prior to recognition of pregnancy have been associated with effects on the child's intelligence and with learning problems at seven years of age (Steissguth et al 1990). On the other hand, low to moderate alcohol use (two to four standard drinks daily) before or during pregnancy may have no serious effects (Walpole et al 1990).

The apparent variation of response of the fetus to exposure to alcohol in utero is poorly understood but the NHMRC, and several other authorities, have considered that abstinence from alcohol consumption during pregnancy should be recommended (NHMRC 1992b).

### 7.3 Health consequences

As discussed above and in Subsection 1.2, the health consequences of low birthweight affect both short and long-term development. In the neonatal period, low birthweight infants have a much higher risk of death than infants of normal birthweight. Babies with intrauterine growth restriction have structural and metabolic abnormalities that might restrict their future health and make them more prone to particular conditions including Type 2 diabetes and cardiovascular disease. They have abnormal glucose-insulin metabolism, elevated serum triglycerides and
high levels of plasma hormones and cortisol (Barker & Fall 1993). They may also have disproportionate reduction in the size of some organs, including the liver.

Differences in low birthweight rates account for the higher neonatal death rates that occur in some groups, particularly those with socio-economic disadvantages (McCormick 1985). Infant mortality is similar or better among low birthweight Aboriginal babies than among low birthweight non-Aboriginal babies. However, as birthweight increases so does the risk-ratio of Aboriginal to non-Aboriginal mortality (Kliwer & Stanley 1993; Mercer et al 1994).

Prospectively collected measurements showed that Aboriginal infants of low birthweight were lighter and shorter throughout the first five years of life than other Aboriginal infants and children and showed incomplete ‘catch-up’ growth over this period (Gracey et al 1989). This is similar to experience with Aboriginal children in Cape York (far north Queensland) who had poor ‘catch-up’ in later years in attempting to compensate for growth restriction which occurred after three to twelve months of age (Cox 1979).

Infants who are small in terms of birthweight for their gestational age are also at high risk of sudden infant death syndrome (SIDS). The SIDS rate in the Aboriginal population has been found to be 3.7 times that in the non-Aboriginal population (Alessandri et al 1994).

### 7.4 What can be done about it?

As the risk factors and health outcomes for preterm delivery and intrauterine growth restriction differ, it is important to determine the causes of low birthweight so that appropriate preventive strategies can be developed and implemented (Kramer 1987). Opinions differ about the extent to which low birthweight in Indigenous babies reflects poor intrauterine growth or preterm delivery.

Poor utilisation of antenatal services by Aboriginal women in urban and rural areas has been found to be a very important cause of poor pregnancy outcomes (National Aboriginal Health Strategy Working Party 1989; de Costa & Child 1996) and is related to a combination of poorer health education and cultural barriers. The high cultural significance of ‘Women’s Business’ must be taken into account when trying to provide appropriate and acceptable obstetric services to Indigenous women (O’Connor & Bush 1996). Among other things, it has been proposed that the following could help provide better services for pregnant Aboriginal and Torres Strait Islander women:

- expanded outreach services from tertiary, regional and district hospitals; and
- more specialist obstetricians and gynaecologists working and living in regional and remote areas (O’Connor & Bush 1996).

**Strong Women – Strong Babies – Strong Culture**

The 1994 to 1996 ‘Strong Women – Strong Babies – Strong Culture’ program (SW-SB-SC) focused upon improving birthweight, with aims to reduce the occurrence of poor nutrition and infection during pregnancy (Mackerras 1998). The program,
which was conducted in the Northern Territory and Western Australia, is strongly Aboriginal-orientated in its design and style of presentation. It concentrates on maternal nutrition and uses a family cultural model which was developed by Aboriginal women and health workers. Prenatal advice and services which have evolved in this program are provided in culturally sensitive and appropriate ways through senior Aboriginal women who are important role models in their communities (Fejo & Rae 1996).

Successful outcomes of the SW-SB-SC program were:

- the development and introduction of the ‘Strong Women’s Story’ (an illustrated narrative), which provides information about the nutritional status and reproductive health of women;
- the modification of antenatal care services (a combination of a bicultural approach — sensitive to the needs of Aboriginal women — with high quality health care); and
- improved health status of pregnant women and their infants (Fejo & Hobson 1998).

The first workshop to train SW-SB-SC workers was held in August 1993. There was a statistically significant and important decline in the prevalence of low birthweight after the SW-SB-SC program started. This decline was much larger than the secular trend seen in Aboriginal births in the rest of the Top End and the Northern Territory over the same time. The proportion of low birthweight infants declined between 1990–91 and 1994–95 by 8.4 per cent from 19.8 per cent to 11.3 per cent in the three pilot communities. By contrast, in the rural Top End regions, there was a 1.5 per cent decline in the prevalence of low birthweight from 17.4 per cent in 1990–91 to 15.9 per cent in 1994–95 (Mackerras 1998).

Figure 7.6 Prevalence of low birthweight in SW-SB-SC pilot communities compared with Aboriginal births in rural regions in the Northern Territory

Source: Northern Territory Midwives Collection (Mackerras 1998).

The trend data show that the improvement did not occur before the introduction of the program. Ongoing evaluation will indicate whether these effects can be reproduced when the program is expanded to other communities.
### What is a healthy diet for pregnant women?

Suggested numbers of sample serves from each food group for women

<table>
<thead>
<tr>
<th>Age Group</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Women (19–60 yrs)</td>
<td>4–6</td>
<td>4–7</td>
<td>2–3</td>
<td>2–3</td>
<td>1–1½</td>
<td>0–2½</td>
</tr>
<tr>
<td>Pregnant</td>
<td>4–6</td>
<td>5–6</td>
<td>4</td>
<td>2</td>
<td>1½</td>
<td>0–2½</td>
</tr>
<tr>
<td>Breast-feeding</td>
<td>5–7</td>
<td>7</td>
<td>5</td>
<td>2</td>
<td>2</td>
<td>0–2½</td>
</tr>
<tr>
<td>60+ yrs</td>
<td>3–5</td>
<td>4–6</td>
<td>2–3</td>
<td>2–3</td>
<td>1½</td>
<td>0–2</td>
</tr>
</tbody>
</table>

1. A sample serve of **bread, cereal, rice, pasta, noodles** is: 2 slices of bread or 1 medium bread roll; 1 cup cooked, rice, pasta or noodles; 1 cup porridge or $1\frac{1}{3}$ cup breakfast cereal flakes or $1\frac{1}{2}$ cup muesli.

2. A sample serve of **vegetables** is: 75 g or $1\frac{1}{2}$ cup cooked vegetables; $1\frac{1}{2}$ cup baked beans; 1 cup salad vegetables or 1 potato.

3. A sample serve of **fruit** is: 1 medium piece (eg apple, banana, orange); 2 small pieces (eg apricots, kiwi fruits, plums); 1 cup diced pieces or canned fruit; $1\frac{1}{2}$ cup fruit juice; dried fruit (eg 4 dried apricot halves or $1\frac{1}{2}$ tablespoons sultanas).

4. A sample serve of **milk, yoghurt, cheese** is: 250 mL fresh, long-life or reconstituted dried milk; $1\frac{1}{2}$ cup evaporated milk; 40 g (2 slices) cheese; 200 g (1 small carton) yoghurt; 250 mL (1 cup) custard.

5. A sample serve of **meat, poultry, fish, eggs, nuts or legumes** is: 65–100 g cooked meat or chicken (eg $1\frac{1}{2}$ cup mince, 2 small chops or 2 slices roast meat); $1\frac{1}{2}$ cup baked beans; 80–120 g cooked fish fillet; 2 small eggs; $1\frac{1}{3}$ cup peanuts or almonds; $1\frac{1}{3}$ cup sunflower seeds or sesame seeds.

6. A sample serve of **extra foods** is the amount of food that provides 600 kilojoules. Some examples are: 4 plain sweet biscuits; 25 g chocolate; 12 hot chips; $1\frac{1}{3}$ meat pie or pastie; 200 mL wine; * 600 mL light beer; * 1 can soft drink

* Alcohol is not recommended for pregnant or breast-feeding women.

Folate deficiency in the mother can cause birth defects such as spina bifida. Pregnant women should include sources of folate, such as cereals, orange juice, spinach and other leafy green vegetables, liver, dried beans or yeast spreads (such as Marmite™, Promite™ or Vegemite™) in their diet. This is especially important in the month before conception and the first three months of pregnancy.

Pregnancy is a major drain on iron reserves. Dietary sources of iron include offal (such as liver and kidney), meat, eggs, fish, poultry and leafy green vegetables.

Only offal from animals that have been butchered according to Australian health regulations should be consumed. Offal from native animals may contain parasites or micro-organisms (ie Salmonella).

**Sources:** DHFS (1998); NHMRC (1992b), NHMRC (1993).
Adequate nutrition is essential for normal growth during infant life and childhood as well as in the prenatal period. Initially an infant will be either breast-fed or bottle-fed. The introduction of solids\(^5\) at four to six months to complement milk feeds (whether from the bottle or the breast) is important. Solids should be given in adequate quantities on a consistent basis to provide additional nutrients and not used as an intermittent condiment.

### 8.1 Breast-feeding

Breast-milk is a balanced, low-residue food, which is readily utilised and meets the particular needs of infants in the early months of life. It provides proteins, fats and carbohydrates at levels and in forms uniquely adapted to the infant's metabolic capacities and growth requirements. It also contains biologically active components that promote growth and development by assisting the digestion and assimilation of nutrients, and actively protects the infant from disease such as gastroenteritis and respiratory tract infections (NHMRC 1995).

It has been suggested that if the prevalence of exclusive breast-feeding to three months was increased from 60 to 80 per cent, at least $11.5 million could be saved each year in health-care costs in Australia (Drane 1997).

**Health consequences**

Breast-feeding is associated with reduced infant and child mortality (Jain 1996) and is increasingly recognised as fundamental for long-term health (James et al 1997). The benefits are no longer considered to be restricted to infants in less technically developed countries (for example the prevention of diarrhoea from contaminated nursing bottles and the reduction in rates of other acute infectious disease during infancy). Breast-fed infants have reduced risk or severity of (Cunningham et al 1991):

- physiological reflux;
- pyloric stenosis;
- respiratory illness;
- gastrointestinal tract disease;
- inflammatory bowel disease;
- some childhood cancers;

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\(^5\) The term ‘introduction of solids’ is used by the NHMRC in its *Dietary Guidelines for Children and Adolescents* (NHMRC 1995) and is less ambiguous than discussion of ‘weaning foods’.
• delayed onset of coeliac disease;
• otitis media (middle ear infection);
• urinary tract infections;
• bacteraemia and meningitis;
• SIDS; and
• necrotising enterocolitis in infants delivered preterm.

Babies breast-fed for the first 13 weeks (three months) of life have substantially reduced rates of gastrointestinal illness and, to a lesser extent, respiratory illness. This benefit has been found to continue up to one year of age (Howie et al 1990). Mothers need to breast-feed for at least three months to confer these advantages on their infants (Howie et al 1990). If mothers consider they have insufficient milk and wish to wean their babies completely, they should be advised that there are advantages in continuing to give some breast-milk for at least three months.

Breast-feeding is beneficial to both infants and their mothers. Health benefits to the breast-feeding mother include some protection against premenopausal breast cancer, ovarian cancer and osteoporosis, and hastening uterine involution after birth (NHMRC 1996). There may also be a beneficial effect on widening the later birth spacing interval (Zhu et al 1999).

Despite widespread and prolonged breast-feeding by Aboriginal mothers in remote areas, their infants have poor growth patterns after six months (Gracey et al 1983) and recurrent infections. Although breast-feeding helps protect against infections such as gastroenteritis, it cannot be expected to completely prevent such infections in contaminated environments. Continued breast-feeding is beneficial for Aboriginal infants and their health would probably be much worse if they were bottle-fed in unhygienic living conditions (Hitchcock 1989).

Rates of breast-feeding

Interpreting data on breast-feeding rates is difficult because the definition of breast-feeding may be restricted to babies who are breast-fed exclusively, or it may include babies who receive only a single daily feed of breast-milk (Cunningham et al 1991). The 1994 NATSIS defined breast-feeding to include ‘any time spent weaning and where this was only part of the child’s dietary intake’ (ABS 1996a). Another problem associated with studies on breast-feeding is that most studies tend only to study women attending a particular maternity or paediatric primary care facility. That population is not necessarily representative of the wider breast-feeding population (Rutishauser & Carlin 1992).

The 1995 NHIS showed that Indigenous mothers breast-fed for longer than their non-Indigenous counterparts (ABS & AIHW 1999). Although this result is based on a small number of Indigenous mothers and does not include mothers (Indigenous or non-Indigenous) from remote areas, it is consistent with the result of the 1994 NATSIS.
Figure 8.1  
Duration of breast-feeding in non-remote mothers, 1995

Note: Percentages based on number of children breast-fed.

Questions on breast-feeding history were asked about children under 13 years old in the 1994 NATSIS. Breast-feeding was found to be most common in the Northern Territory, where it was reported that about 90 per cent of children under the age of 13 had been, or were currently being, breast-fed. The proportions of children under 13 years of age who reported having been breast-fed were highest across northern Australia and the west coast, and lowest in much of the south-east (ABS & AIHW 1997).

Table 8.1  
Breast-feeding of Indigenous children under 13 years of age, by State/Territory

<table>
<thead>
<tr>
<th>Whether breast-fed</th>
<th>NSW</th>
<th>Vic</th>
<th>Qld</th>
<th>SA</th>
<th>WA</th>
<th>Tas</th>
<th>NT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Males (proportion %)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Currently</td>
<td>2.6</td>
<td>7.1</td>
<td>1.3</td>
<td>5.8</td>
<td>6.3</td>
<td>2.2</td>
<td>9.1</td>
</tr>
<tr>
<td>Was breast-fed</td>
<td>56.8</td>
<td>53.6</td>
<td>70.5</td>
<td>54.1</td>
<td>71.4</td>
<td>69.6</td>
<td>80.5</td>
</tr>
<tr>
<td>Not breast-fed</td>
<td>38.2</td>
<td>34.5</td>
<td>26.4</td>
<td>35.6</td>
<td>21.0</td>
<td>26.5</td>
<td>9.7</td>
</tr>
<tr>
<td>Do not know/not stated</td>
<td>2.4</td>
<td>4.8</td>
<td>1.8</td>
<td>4.5</td>
<td>1.2</td>
<td>1.7</td>
<td>0.7</td>
</tr>
<tr>
<td>Females (proportion %)</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Currently</td>
<td>1.5</td>
<td>9.8</td>
<td>2.6</td>
<td>4.6</td>
<td>8.0</td>
<td>3.2</td>
<td>5.6</td>
</tr>
<tr>
<td>Was breast-fed</td>
<td>59.1</td>
<td>52.6</td>
<td>69.7</td>
<td>55.8</td>
<td>66.9</td>
<td>66.5</td>
<td>84.3</td>
</tr>
<tr>
<td>Not breast-fed</td>
<td>38.7</td>
<td>36.3</td>
<td>27.0</td>
<td>34.2</td>
<td>23.5</td>
<td>30.1</td>
<td>9.4</td>
</tr>
<tr>
<td>Do not know/not stated</td>
<td>0.7</td>
<td>1.4</td>
<td>0.7</td>
<td>5.4</td>
<td>1.6</td>
<td>0.2</td>
<td>0.8</td>
</tr>
</tbody>
</table>

Source: ABS (1996a).

Over 70 per cent of Indigenous babies in the Northern Territory were breast-fed for six months or more. This proportion is much higher than that reported for other States/Territories. Indigenous babies in rural areas were more likely to be breast-fed for longer than six months than those in urban areas (ABS 1996a ).
Table 8.2  Breast-feeding in persons aged under 13 years, by area of residence, 1994

<table>
<thead>
<tr>
<th>Whether breast-fed</th>
<th>Capital city (%)</th>
<th>Other urban (%)</th>
<th>Rural (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Less than 3 months</td>
<td>17.4</td>
<td>11.6</td>
<td>8.5</td>
</tr>
<tr>
<td>3 months to &lt; 6 months</td>
<td>12.1</td>
<td>13.6</td>
<td>8.4</td>
</tr>
<tr>
<td>6 months to &lt; 12 months</td>
<td>13.2</td>
<td>15.9</td>
<td>13.7</td>
</tr>
<tr>
<td>12 months or more</td>
<td>19.1</td>
<td>23.1</td>
<td>42.1</td>
</tr>
<tr>
<td>Currently breast-feeding</td>
<td>4.0</td>
<td>3.2</td>
<td>6.2</td>
</tr>
<tr>
<td>Not stated</td>
<td>0.1</td>
<td>0.5</td>
<td>1.0</td>
</tr>
<tr>
<td>Subtotal</td>
<td>65.9</td>
<td>67.8</td>
<td>79.8</td>
</tr>
<tr>
<td>Was not breast-fed</td>
<td>33.3</td>
<td>30.4</td>
<td>18.2</td>
</tr>
<tr>
<td>Don’t know/not stated</td>
<td>0.9</td>
<td>1.8</td>
<td>2.0</td>
</tr>
</tbody>
</table>

Source: ABS (1996a).

Factors influencing rates of breast-feeding

Many factors influence the pattern and duration of breast-feeding. In general, older, better educated women who are relatively well off socio-economically, live in a stable relationship, and have support for breast-feeding from friends and family continue to breast-feed longer. The use of combined oral contraceptives and cigarette smoking are both associated with a shorter duration of breast-feeding (Rutishauser & Carlin 1992).

Socio-economic status, including education as well as income, is the most potent factor affecting the prevalence of breast-feeding (Lund-Adams & Heywood 1995). The 1989–90 NHS supports the positive relationship between higher socio-economic status of women and longer duration of breast-feeding (Jain 1996). The NATSISS also found that women of higher socio-economic status are more likely to breast-feed and to breast-feed for longer than women from lower socio-economic groups. For example, babies from households with an annual income of $25,000 or more were more likely to be breast-fed and Indigenous babies in a household with one or more persons working were more likely to be breast-fed and for longer than those in a household where no-one was working (ABS 1996a).

Urban Aboriginal women breast-fed for approximately the same length of time as non-Aboriginal women of low socio-economic status (Phillips & Dibley 1983; Hitchcock & Coy 1989). One study found no difference in breast-feeding rates between Aboriginal and non-Indigenous women of low socio-economic backgrounds (Cox 1981).

Consistent with findings of Australian infant feeding surveys (Cox & Turnbull 1994; Lund-Adams & Heywood 1995), the most commonly reported reason for ceasing breast-feeding among Aboriginal mothers was the anxiety over inadequate or no milk (39 per cent). Most studies have concluded that this response is due to a lack of understanding about breast-feeding, the relative isolation of new mothers, and the lack of professional and social support (Lund-Adams & Heywood 1995). Other reasons given by Aboriginal mothers were: interferes with lifestyle (22 per cent);
anxiety about infant’s illness (17 per cent); and sore breasts or nipples (9 per cent) (Phillips & Dibley 1983).

Focus group discussions with Aboriginal people in Melbourne identified that most Aboriginal women wanted and expected to breast-feed. They perceived their community was supportive of breast-feeding, though they often lacked people to turn to for advice and support. Disruption of the passing on of knowledge of traditional healthy infant feeding practices between generations is a cultural loss that has occurred among Aboriginal communities. Factors which influenced their choice to bottle-feed in recent years included the embarrassment of feeding in public, perceptions that breast-feeding is painful and inconvenient, and the belief that feeding formulas are as good as breast-milk (Holmes et al 1997). Breast-feeding promotion in Aboriginal communities needs to include strategies to raise confidence and self-esteem, address feelings of shame, and foster a return to traditional supportive attitudes towards pregnant women and mothers with babies (Holmes et al 1997).

Promotion of breast-feeding

Formal Commonwealth Government commitment to protecting, promoting and supporting breast-feeding is shown by the 1992 Australian Agreement on Marketing in Australia of Infant Formulas which gives effect to the principles of the WHO’s International Code of Marketing of Breast Milk Substitutes (WHO 1981; DHHCS 1993). Under the Agreement, manufacturers and importers of infant formula may not advertise or in any other way promote infant formulas to the general public. The Government also supports Baby Friendly Hospital Initiatives and community initiatives promoted by UNICEF and the International Paediatric Association (Lund-Adams & Heywood 1995; UNICEF, undated).

The inclusion of a guideline on breast-feeding in the Dietary Guidelines for Australians (NHMRC 1992a) and Dietary Guidelines for Children and Adolescents (NHMRC 1995) and the inclusion of breast-feeding in Australia’s national goals and targets (Nutbeam et al 1993) all indicate support for the importance of breast-feeding to the health of infants.

Health professionals and voluntary health workers can provide invaluable assistance to new mothers when they start breast-feeding. Factual information, sympathetic support, demonstration of practical skills and locally relevant strategies for problem solving can create a positive environment for breast-feeding (NHMRC 1996). The Infant Feeding Guidelines for Health Workers (NHMRC 1996) address some of these issues. The Guidelines aim to help health workers understand how to deal with problems which may lead to early and inappropriate weaning that includes foods that are not satisfactory for infants and which may be contaminated.

Breast-feeding support programs in Australia

A review of current interventions and identification of current best practice by community-based Aboriginal and Torres Strait Islander health service providers in promoting and supporting breast-feeding and appropriate infant nutrition, acknowledged that projects generally lacked the benefit of formal evaluation (Engeler...
et al 1998). Therefore, the assessment of individual services and projects was
difficult. This needs to be addressed in future. The review summarises programs that
support Indigenous mothers in breast-feeding and provides appropriate infant
nutrition and includes ‘self-assessed’ strengths and weaknesses of the project.

Common themes identified were that (Engler et al 1998):

- breast-feeding and infant nutrition are not seen as a priority issue by all providers;
- variations in perspective between urban providers, where in general breast-
  feeding rates are lower, and those in remote rural areas are marked;
- resources, combined with social and political action, are key factors;
- multiple approaches are needed;
- sustainable funding and the importance of pro-active services are required;
- competence of health staff to diagnose, manage and refer is often constrained by
  a lack of expertise or workplace support to exercise skills;
- infant nutrition issues need to be placed within the wider context of women’s
  lives — the effect of lost parenting skills in the ‘stolen generation’ is noted;
- there is a need for health policies to address erroneous assumptions about
  technical aspects and community dynamics;
- simply providing information will not solve the problem; and
- there is a need for both research and evaluation of programs.

It was the general view that the core elements of effective programs are that:

- the project should ideally be initiated and designed with support by the target
  community in partnership between community members and those providing
  technical support;
- ownership of the program remains firmly within the community;
- community members are involved actively in all stages of development,
  implementation and evaluation; and
- strategies should be targeted specifically to community needs.

The recommendations were developed to sustain an environment that supports
Aboriginal and Torres Strait Islander mothers to breast-feed and to provide
appropriate food for their babies. The recommendations address issues on policy and
information, staff expertise, service management, research and community and
family support.

Focus group discussions with Aboriginal people in Melbourne identified that many
women felt ill at ease at hospital antenatal classes and requested classes at their own
health service (Holmes et al 1997).
Breast-feeding

Why breast-feeding?

• Breast-milk is a balanced food that meets the needs of infants in the early months of life, providing all the necessary nutrients for proper growth.
• Breast-feeding for the first three months of the baby’s life will help protect the baby from illnesses, such as gastroenteritis and respiratory tract infections.

Changes to the mother’s diet during breast-feeding

• During the period that she is breast-feeding, a woman needs to increase her daily intake of nutrients. The Table on page 95 shows approximate numbers of serves from each food group required during breast-feeding.
• Cigarette smoking can affect the milk supply and may cause gastrointestinal upsets in the infant so mothers should be advised to give up smoking. If this is not possible, they should reduce smoking to as little as possible and should not smoke in the hour before feeding, or during feeding, to reduce harmful effects.
• Breast-feeding mothers should be advised not to drink alcohol. If alcohol is consumed, it should be limited to a maximum of one or two drinks a day and consumed after breast-feeding. The level of alcohol in the breast-milk is the same as the level in the mother’s blood.

Difficulties with breast-feeding

The most common reasons for stopping breast-feeding are that the mother feels she does not have enough milk, that she has sore nipples or that the baby demands too many feeds. Some women also find breast-feeding in public embarrassing.
• Babies are getting enough milk if:
  — they are fully breast-fed (no other fluids or solids) and having six to eight very wet nappies of pale, inoffensive smelling urine in a 24 hour period;
  — as young infants they have some greenish/yellow bowel motions daily;
  — they are alert with bright eyes, moist lips, good skin tone;
  — they have appropriate weight gain when averaged out over a four week period;
  — they are fed according to need rather than schedule (although some sleepy infants may need reminding).
• Nipple soreness and damage is usually a sign of poor attachment and sucking. The diagram below illustrates good and poor attachment of the baby to the breast.

Sources: NHMRC (1996); DHFS (1998); WHO (1993).
8.2 Bottle-feeding

There are several circumstances where breast-feeding may not be possible or feasible for the mother or may be hazardous to the infant. Examples include:

- severe maternal illness and infections (including HIV);
- maternal alcohol or drug consumption; and
- maternal separation or death.

Some mothers may choose to bottle-feed their babies from an early age. This choice may be made for personal reasons, because of competing demands on the mother or because of attitudes in society to breast-feeding in public places. Women who are thinking of bottle-feeding their baby for these reasons should be advised that there are health advantages in continuing to give some breast-milk for at least three months.

They should also be advised of the negative aspects of bottle-feeding and of the necessity for careful attention to hygiene. The greatest risk arising from bottle-feeding is gastrointestinal infection which may lead to loss of fluids and nutrients and, therefore, to growth faltering, failure to thrive and under-nutrition. This, in turn, can predispose infants to further infectious episodes including diarrhoeal disease. Bottle-fed babies are more prone to other infections as well, particularly those affecting the respiratory tract. A major reason for gastrointestinal infections is the contamination of milks when they are prepared or the contamination of feeding bottles, particularly in unhygienic conditions and in warm or hot ambient temperatures. Diseased animals (such as dogs) can also be a source of infection.

Other important negative considerations relevant to bottle-feeding include:

- the use of powdered milks rather than formulas designed specifically for the nourishment of infants, which may result in under-nutrition;
- the incorrect mixing of infant formula;
- the high cost of infant formulas;
- the possibility of allergic illnesses induced by foreign proteins (e.g., cow's milk or soy); and
- the risk of dental caries if babies are given sweetened drinks.
Bottle-feeding

Why bottle-feeding?

- Bottle-feeding is essential in cases of maternal separation and can be useful if the mother is seriously ill.
- If the mother regularly consumes alcohol or drugs (both of which pass into breast-milk), it is better for her baby if she changes to bottle-feeding.
- Women who are known to be infected with HIV, Hepatitis C or other blood-borne viruses should not breast-feed their babies.
- Some women are unable to breast-feed for medical reasons.
- Some women are not comfortable with breast-feeding.
- Women who are not in any of the above circumstances should be advised of the advantages of breast-feeding.

Correct bottle-feeding

- It is important to use a formula specially designed for feeding babies and infants. Powdered milk does not have all the nutrients the baby needs.
- Infant formula must be made up following the instructions. If the mixture is too weak, it will not support normal growth. Clean water should be used, otherwise there is a risk of infection. It is safest to make up one bottle at a time, in the bottle. The bottle should not be kept warm for more than 10 minutes.
- Baby bottles should be rinsed in cold water after use, washed in detergent and hot water and rinsed again. They can be sterilised by boiling them in a large saucepan for five minutes. Infant formula should be kept in a covered container. Poor hygiene can lead to high rates of infections in infants, particularly diarrhoea and respiratory tract infections.
- Special-purpose formulas, such as those that are low-lactose or lactose-free, should only be used when this is advised by a health professional. Similarly, milks that are based on particular ingredients, such as soy-based formulas, should only be used on professional advice. These special formulas are more expensive and can have side effects including allergies.
- Babies should not be given baby bottles containing sweetened drinks as this can cause dental caries. Tooth decay can also be caused by sweetening the teat of the bottle or leaving the bottle propped in the baby’s mouth (see Chapter 15). The use of dummies should also be discouraged.
8.3 Introducing solids

The nutritional and immunological effects of prolonged breast-feeding are particularly important in communities with a high prevalence of infectious diseases such as diarrhoea and respiratory tract infections. However, the introduction of solids at four to six months to complement milk feeds is also important. In groups where the nutritional status of the mothers may be marginal, it is probably wise to promote the addition of solids at around four to five months rather than delaying this until six months. Solids should be given in adequate quantities on a consistent basis to provide additional nutrients and not used as an intermittent condiment.

Studies have shown that if children are fed solids in adequate quantities at four to six months they grow normally even in microbiologically contaminated circumstances (Engeler et al 1998).

A traditional practice among Aboriginals is to wait until a child demands food before breast-feeding or giving other foods (Harrison 1986). This tradition will not adversely affect a well nourished child. However lack of hunger and apathy are common results of mild malnutrition (Waterlow 1994). Hence, children who have even mild growth failure may not be very hungry and need to be encouraged to eat. They would be disadvantaged if their mothers waited for signs of hunger before feeding them.
Introducing solids

When should solids be introduced?

When an infant is aged between four and six months solids should be introduced into their diet. At this age:

- the infant’s appetite and nutritional requirements are generally no longer satisfied by breast-milk or infant formula alone — stores of several nutrients, such as iron, are often falling in exclusively milk-fed infants (both breast-fed and formula-fed) (see page 49 for examples of foods that are rich in iron);
- the development of feeding behaviour has progressed from sucking to biting (and subsequently chewing by seven to nine months of age); and
- the infant’s digestive system is maturing.

What foods should be introduced?

- Introduction of foods generally starts with iron-enriched infant cereals, then fruits, vegetables, meats, poultry and fish are added gradually as the infant becomes accustomed to them. There are no rules about the order of introduction.
- The first foods should be soft and smooth (ie commercial baby food or cereal, mashed banana, cooked carrot, potato or pumpkin). The infant will quickly learn to cope with different textures and will accept food that has been mashed with a fork or minced. Once the infant is able to hold things, finger foods such as pieces of fruit or bread can be offered. Other foods can be chopped into small pieces.
- Commercial baby foods are a useful alternative to food prepared at home, particularly those in jars, which can be closed to protect the food.
- Feeding bottles should only be used for breast-milk or infant formula (see Chapter 15). Comfort sucking on a bottle can become a habit. Lidded feeding cups should be used for any other liquids from six months of age.

How much food does the infant need?

The amount of food an infant or child needs varies from day to day and from child to child. Variables influencing the child’s appetite include age, gender, whether the child is recovering from low birthweight or an illness (and what type of illness) and injury.

Strategies for improving and monitoring food intake include:

- establishing routines for meal times and snacks where the child sits down to eat in the company of others;
- establishing habits that will help ensure variety and nutritional adequacy;
- making a snack box available to the child. This can contain healthy snack foods (ie pieces of fruit, vegetables, cheese, small sandwiches);
- giving small amounts of food rather than large servings and having extra available if the child wants more; and
- trying to serve the child healthy foods that they enjoy. Remember that likes and dislikes change over time.

Chapter 9 — Childhood growth

Childhood growth

Normal growth results from the combined effects of favourable genetic and environmental influences. Genetic factors include parental size and the physical size and growth patterns of particular ethnic or racial groups of origin. Positive environmental factors include good maternal health during pregnancy, healthy feeding and weaning practices, and the absence of infections and parasitic infestations.

When these factors are compromised, growth of the fetus, infant and young child is likely to be inadequate. If the environmental factors that affect growth are persistently negative (e.g., because of living in overcrowded, unhygienic conditions, with repeated infections and poor nutrition) then growth performance is likely to be chronically or persistently sub-standard and this can lead to ‘failure-to-thrive’ or clinically evident under-nutrition or malnutrition, sometimes with important nutritional complications such as specific nutrient deficiencies and anaemia. Under-nutrition in infancy and early childhood, often preceded by low birthweight, can lead to permanent growth restriction and stunting (Gayle et al. 1987).

9.1 Assessment of growth

In healthy populations, infants and children are able to reach their growth potential. This depends on the provision of adequate food, the use of infant feeding practices that are appropriate for age and development, and good hygiene.

Growth can be measured for two quite different purposes:
- to track the growth of individual children; or
- to describe how a population is growing.

When tracking individual children, sequential measurements on each child are needed and the results are generally plotted on a separate chart for each child. When monitoring an individual child, the shape of the child’s growth should follow the shape of the curves on the chart. Whether the child is above or below various cutpoints is rarely important. When describing the status of growth in a population, each child, or a sub-sample of children, only needs to be measured once. Usually the results are presented as means or the proportion above or below one or more cutpoints. The results of a survey can be also presented visually by plotting all children on a single chart (e.g., Figures 9.12 and 9.13).

Growth references

To know how a child is growing, the child’s height (or length) and weight are compared to curves determined by the National Center for Health Statistics (NCHS) in the United States. The curves show the range in child size that can be expected at any age in a well-growing population. These have been endorsed by the WHO as a number of studies in less developed countries have shown that pre-
Nutrition in Aboriginal and Torres Strait Islander Peoples

Pubertal children from high socio-economic groups of most ethnicities have similar growth to American children (Habicht et al. 1974; Graciter & Gentry 1981; Dibley et al. 1987). The data are based on fairly old surveys and more recent work indicates that secular trends with increasing height are still occurring in North America, Australia, the Mediterranean and Asia (Ulijaszek 1994). This means that the current reference curves probably underestimate growth potential of pre-adolescent children. Little is known about the ethnic influences on the growth of adolescent children (Martorell et al. 1994).

The NCHS reference curves have been endorsed by the NHMRC for use in Australia. The curves can be represented in a number of different ways. Charts using centiles (or percentiles) and those using Z-scores (or standard deviations) are currently in use in Australia. Older charts using percent-of-the-median are no longer used.

Figures 9.1 to 9.4 show curves based on the centiles of the distribution. Separate charts are available for boys and girls in the ranges birth to three years and two to eighteen years. Weight and length (or height) are on the same chart. Note that the length in the two to three years range on Figure 9.1 is greater than the stature for two to three years range on Figure 9.3 because Figure 9.1 assumes that the child is measured lying down and Figure 9.3 that the child is measured standing up.

Figures 9.5 to 9.8 show curves based on Z-scores that are used in remote Aboriginal communities in the Northern Territory and some parts of South Australia. There are separate charts for boys and girls in just the birth to three years age range. Length (or height) and head circumference are plotted on different graphs. The dip in the length/height graph at age two years shows the change due to measuring a child standing up rather than lying down. The middle line (or median) for each of the graphs in Figures 9.5 to 9.8 is the same as the equivalent middle lines in Figures 9.1 to 9.4. The other lines in Figures 9.5 to 9.8 show +2 and –2 standard deviations of the distribution, rather than the centiles shown in Figures 9.1 to 9.4.

The charts in Figures 9.1 to 9.8 show age along one of the axes and so they plot weight-for-age or height-for-age. Low values of weight-for-age are referred to as underweight and low values of height-for-age are referred to as stunting. These charts tend to be used in monitoring individual children. Another useful index is to plot weight against height instead of age. Reference curves for weight-for-height are available for girls up to 137 cm and boys up to 145 cm. Low values of weight-for-height are referred to as wasting. Measuring underweight alone does not reveal whether the child is suffering from stunting, wasting or both.

Various cutpoints are used to define underweight, stunting or wasting. UNICEF requests all counties to report on the proportion of the under-five population having weight-for-height, height-for-age and weight-for-age Z-scores of –2. These data are not available for either the general Australian population or for the Aboriginal and Torres Strait Islander population. It is important to remember that 2.3 per cent of children are expected to fall below these cutpoints. A public health problem only exists when the proportion in these groups is higher than 2.3 per cent (Gorstein et al. 1994). Although cutpoints can be useful when describing the results of a population survey, they are often misused when monitoring the growth of individual children.
Figure 9.1 NCHS growth chart (girls, birth to 36 months) for weight and length

Source: Adapted from Hamill et al (1979).
Figure 9.2  NCHS growth chart (boys, birth to 36 months) for weight and length

Source: Adapted from Hamill et al (1979).
Figure 9.3 NCHS growth chart (girls, 2 to 18 years) for weight and stature (height)

Source: Adapted from Hamill et al. (1979).
Figure 9.4  NCHS growth chart (boys, 2 to 18 years) for weight and stature (height)

Source: Adapted from Hamill et al (1979).
Figure 9.5  Growth chart for use in communities in the Northern Territory — weight for girls

Figure 9.6  Growth chart for use in communities in the Northern Territory — length/height and head circumference for girls
Figure 9.7  Growth chart for use in communities in the Northern Territory — weight for boys

Figure 9.8  Growth chart for use in communities in the Northern Territory — length/height and head circumference for boys
Describing child size

- The spread around the central point, the median, can be described either as centiles (percentiles) or as Z-scores (standard deviations). Centiles have been used in the past but the extreme centiles, such as the 97th or 3rd centile are calculated from a small subset of the data and so are inaccurate (Cole 1990). This combined with the fact that the centiles are not normally distributed and so statistics such as t-tests cannot be done on them (Shann 1993; Gorstein et al 1994) has lead to the recommendation that Z-scores be used both for charts for individual monitoring and for analysis of population surveys (Ulijaszek 1994).

- Centiles rank the population from bottom to top. The 50th centile is the median: 50 per cent of the population are above it and 50 per cent below. The 25th centile is the point at which 25 per cent of the population have values lower and 75 per cent higher. The 75th centile is the opposite: 75 per cent of the population have values below the 75th centile and 25 per cent have higher values.

- If the spread is the same on both sides of the median, then the distance between the median and the 25th centile is the same as the distance between the median and the 75th centile. It is clear from Figures 9.1 to 9.4 that the spread for height-for-age is even while the spread for weight-for-age is uneven. Weight-for-height also has an uneven spread around the median.

- Z-scores rank the population out from the centre using the standard deviation as the unit. A child who is on the median has a Z-score of 0 because he/she is no different from the median. The standard deviation is different at every age because the spread slowly increases with age.

- Someone who is one standard deviation unit below his age median has a Z-score of −1.0, someone who is two standard units above his median for age has a Z-score of +2.0. As weight-for-age and weight-for-height have a different spread on each side of the median, different values of the standard deviation were determined to allow calculation of the Z-scores. This is why the reference curves are called ‘normalised’.

- Theoretically, there is a relationship between Z-scores and centiles and so the centile lines can be derived from the Z-scores. In practice the particular centile lines on the charts may have been derived by a separate process from the Z-scores. However, the skewness of the data make the practicalities of applying this simple idea to drawing up charts fairly complex (Cole 1990).

- The theoretical equivalences of the Z-scores and the centiles can be looked up in a table available in any statistics text. The EpiInfo computer package has a module that will calculate the Z-scores and centiles (and percent-of-the-median). Some key equivalences are:

<table>
<thead>
<tr>
<th>Z-score</th>
<th>Centile</th>
</tr>
</thead>
<tbody>
<tr>
<td>+2.0</td>
<td>97.7</td>
</tr>
<tr>
<td>+1.64</td>
<td>95</td>
</tr>
<tr>
<td>+1.28</td>
<td>90</td>
</tr>
<tr>
<td>+0.675</td>
<td>75</td>
</tr>
<tr>
<td>0</td>
<td>50</td>
</tr>
<tr>
<td>−0.64</td>
<td>25</td>
</tr>
<tr>
<td>−1.28</td>
<td>10</td>
</tr>
<tr>
<td>−1.64</td>
<td>5</td>
</tr>
<tr>
<td>−2.0</td>
<td>2.3</td>
</tr>
</tbody>
</table>
Monitoring growth of individual children

When tracking the growth of individual children, it is necessary to have more than one measurement to assess how growth is progressing. This allows an assessment of the velocity, or speed, of growth. For an individual child, the exact place on the graph is not so important. The critical factor is whether the line of the child's growth has the same slope as the lines on the chart. It is a common mistake to think that a child who is between the lines must be growing well and that a child who is below the bottom line has a growth problem. The same principles apply in monitoring the growth of any child whether they were of low birthweight, delivered preterm, both or neither.

Figures 9.9 and 9.10 both show the growth of the same two children, Figure 9.9 using Z-scores and Figure 9.10 using centiles. Although Child A is growing between the lines, he is the child that the health clinic should be concerned about, not Child B. The clinic should have found out what the problem was at about 9 to 11 months of age. Child B's growth is following the lines on the chart very well and his mother should be told that she is looking after him very well.

If Child A had received early attention, then he would not have needed to put on much weight to get back to his previous track. At about two years of age he needs to put on about 1.5 kg, more than 10 per cent of his current weight. He needs to eat a lot of food over a long time to do this and to keep growing at the same time.

It is clear that the clinic can make the same assessment about the children's progress even though one of these charts has lines based on centiles and the other has lines based on Z-scores.

Figure 9.9  Growth chart showing unsatisfactory (child A) and acceptable (child B) growth, Z-scores chart
A child's growth path in about the first two months of life is mainly related to readjustments, with large babies tending to grow more slowly and small babies growing fast (Ulijaszek 1994). Many preterm babies who are small or light at birth tend to have catch-up growth within the first few years of life. A common observation in Northern Australia is that Aboriginal infants grow faster than the reference curve. After this age, growth should vary around a track that follows the shape of the curve. The variation is due to things such as seasonal variation in growth (Ulijaszek 1994).

One important exception relates to the derivation of the reference curve from a largely artificially fed population. It seems that between about six and twelve months of age, the track of a breast-fed infant who is receiving the recommended amounts of solids, should drop slightly and then rise again after 12 months (WHO Working Group on Infant Growth 1995). The drop appears to be to a Z-score of -0.5 at 12 months at the population level. This is considerably less than the drop seen in many Aboriginal communities where a drop to about -1.5 Z-scores may occur by 12 months and there is no subsequent catch-up (Ruben & Walker 1995; Muller et al 1995; Rousham & Gracey 1997; Walker 1996).

The greater weight in offspring of diabetic women is due to adiposity rather than greater length (Blank et al 1995). Unlike large babies of non-diabetic women, the large infants of women who had diabetes during pregnancy commonly lose their excess adiposity in the first year of life, but subsequently regain it and become overweight by about five years of age (Silverman et al 1996). This means that when the child's growth is monitored it will cross centile lines on a weight-for-age or weight-for-height chart during the first year and this may be misinterpreted as
growth failure. This may be particularly important for Torres Strait Islanders who have the highest rates of diabetes during pregnancy (see Subsection 7.1).

Sometimes there is confusion about plotting the growth of babies who were born preterm. This can be done in several ways. One method is to ignore the fact that delivery was preterm and plot the infant’s postnatal age. If this is done, preterm babies will often fall below the bottom line of the chart. The main point is to check that the slope of the baby’s growth follows the slope of the lines on the chart. It should be explained to the mother why her baby seems so small and that it is the slope that is important. For example, Child B in Figures 9.9 and 9.10 could be a preterm baby who is growing well.

Another way to plot the growth of a baby born preterm is to correct for the degree of preterm delivery. For example, if a child was born four weeks early, then he would not be plotted on the chart until he was one month old. At this point he would be plotted as a baby aged zero months not as a baby aged one month. This method plots the child against post-conception age (minus nine months). A baby plotted this way will often fall between the lines and this may be more easily understood by some people. However it is still important to look at the slope of growth, not the exact place on the chart when monitoring growth.

Growth velocity
As discussed, children’s weight and length are usually plotted on the standard growth charts which show the size attained. However, slight reductions in the velocity (speed) of growth may not be very obvious to the naked eye. Figure 9.11 shows a fairly typical velocity pattern for many children in remote areas if weight change is plotted as grams/month instead of kilograms. In the age range six to twelve months, weight velocity is lower than the expected rate but it returns to the expected rate in the second year. This means that the child never catches up the missing weight (and length).

Velocity charts are more difficult to understand than growth charts because the velocity declines in the first year when the child is actually putting on weight. Hence they are not generally used in health clinics. In addition, children would not generally be weighed often enough to make velocity a more useful assessment than attained size. From a clinical viewpoint, growth velocity is a more sensitive indicator of actual health than attained weight or height in a child (Gorstein et al 1994). However, the inaccuracies and difficulties in measuring height or length mean that this is rarely practical.
Differences in growth

As discussed, recent studies in a number of countries, including Australia, have shown that exclusively breast-fed infants who are given solids at four to six months in accordance with recommendations, grow differently from formula-fed infants (Hitchcock & Coy 1989; Dewey et al 1995). Compared to the currently used NCHS charts, breast-fed infants grow faster in the first four to six months of life, and have an average Z-score of +0.1 at six months. They grow more slowly in the second half of infancy and have an average Z-score of −0.5 at 12 months. The populations studied to date have not included many low birthweight infants and so the lower bounds of the breast-fed population data are not yet certain (WHO Working Group on Infant Growth 1995).

As more work is needed in a greater variety of populations with long duration of breast-feeding, revised charts are not expected for a number of years. Hence, it could appear that the current charts may underestimate the occurrence of growth failure in the first six months of life and overestimate it at around 12 months. (WHO Working Group on Infant Growth 1995). However, this cannot explain the low growth in Aboriginal populations with prolonged breast-feeding rates (Ruben & Walker 1995; Mullan et al 1995; Rousham & Gracey 1997; Walker 1996).

9.2 Extent of the problem

The second use of the reference growth charts is to describe the growth of the population. As the charts are approximately correct, it is possible to say that when the growth of a population of pre-pubertal children deviates substantially from the reference, this is caused by malnutrition, not by genetic differences. Describing
population growth as Z-scores is preferable, as these tend to be normally distributed and so statistics such as t-tests can be done. In addition, as the Z-scores adjust for the differences in size that are due to age and sex, using Z-scores as the summary allows information from small groups to be collated.

School-aged children

Children aged five years and older were measured in the NATSIS and the results presented as Z-scores (Table 9.1). Overall, for the group aged five to nine years, the children were shorter than expected but had an average weight that was almost exactly on the median (i.e. a Z-score of 0). This means that they were relatively heavy for their height which is shown in the mean weight-for-height Z-score of about +0.5. These findings were generally true for boys and girls for each year of age. However, when place of residence was examined, the pattern was quite different. Children living in the capital cities had less deficit in height than children living in other urban or rural locations. Children living in capital cities were, on average, heavier for their age than the expected average whereas children in other locations were lighter for their age than the expected average. Even so, the mean weight-for-height in all locations was above the expected average with children in capital cities being markedly above the expected average. However, the standard deviations are extremely wide. This indicates that, even though the average is high, there is an unexpectedly high proportion of children who are light for height. The extent to which the wide standard deviations are due to the method of ascertaining age in the NATSIS is currently unknown.

Table 9.1  Mean Z-scores for height-for-age, weight-for-age and weight-for-height for Indigenous children aged 5-9 years, by place of residence, 1994

<table>
<thead>
<tr>
<th></th>
<th>Height-for-age</th>
<th>Weight-for-age</th>
<th>Weight-for-height</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
</tr>
<tr>
<td>Boys</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overall</td>
<td>-0.47</td>
<td>1.6</td>
<td>-0.03</td>
</tr>
<tr>
<td>Capital city</td>
<td>-0.29</td>
<td>1.3</td>
<td>0.35</td>
</tr>
<tr>
<td>Other urban</td>
<td>-0.54</td>
<td>1.7</td>
<td>-0.11</td>
</tr>
<tr>
<td>Rural</td>
<td>-0.53</td>
<td>1.6</td>
<td>-0.3</td>
</tr>
<tr>
<td>Girls</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overall</td>
<td>-0.38</td>
<td>1.5</td>
<td>0.00</td>
</tr>
<tr>
<td>Capital city</td>
<td>-0.19</td>
<td>1.5</td>
<td>0.42</td>
</tr>
<tr>
<td>Other urban</td>
<td>-0.41</td>
<td>1.4</td>
<td>-0.13</td>
</tr>
<tr>
<td>Rural</td>
<td>-0.53</td>
<td>1.6</td>
<td>-0.20</td>
</tr>
</tbody>
</table>

Notes: As age was collected in completed years, Z-scores were calculated by adding six months (e.g. for all five-year old children, a value of five years and six months was used). Weight-for-height is for children 77-137 cm only. SD = standard deviation.

One problem with assessing child size is that the NCHS charts do not have weight-for-height references for children taller than 137 cm. This is because puberty occurs at different ages in different populations and exerts a major influence on the size of the child. To get round this problem, it has become customary to calculate body mass index. It is most important that the body mass index of a child is compared to age- and sex-specific reference values and not to the categories defined for adults. This is because both weight and height, and therefore body mass index, are dependent on age and sex during childhood and adolescence. Comparisons can be made with different sets of reference values such as American values for 6–24 years (Must & Dallal 1991) or Australian values from the 1985 Australian Council on Health, Physical Education and Recreation (ACHPER) survey for the range 7–15 years (Lazarus et al 1995; Harvey & Althaus 1993).

The body mass index of Indigenous children aged 7–15 years in the NATSIS was compared to the 1985 Australian survey using the 15th and 85th centiles as cutpoints (Table 9.2). Theoretically, Z-scores would be preferable but the Z-scores for body mass index from the 1985 survey have not been published. This comparison is still based on the assumption that puberty occurs at the same age in the general Australian and Indigenous populations. Overall the proportion in the two groups is higher than the 15 per cent expected by definition. This varies by location. In the capital cities, the expected proportion fall below the 15th centile while there is double the expected prevalence of high values. By contrast, the distribution in the rural areas is very spread out with an excess of both low and high body mass indices. Although the age group is slightly older than that shown in Table 9.1, the pattern by location of residence is the same with urban children being heavier in relation to their height than rural children.

<table>
<thead>
<tr>
<th></th>
<th>Boys</th>
<th></th>
<th>Girls</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;15th centile (%)</td>
<td>&gt;85th centile (%)</td>
<td>&lt;15th centile (%)</td>
<td>&gt;85th centile (%)</td>
</tr>
<tr>
<td>Overall</td>
<td>24</td>
<td>28</td>
<td>22</td>
<td>28</td>
</tr>
<tr>
<td>Capital city</td>
<td>16</td>
<td>28</td>
<td>16</td>
<td>30</td>
</tr>
<tr>
<td>Other urban</td>
<td>26</td>
<td>31</td>
<td>23</td>
<td>29</td>
</tr>
<tr>
<td>Rural</td>
<td>30</td>
<td>24</td>
<td>26</td>
<td>25</td>
</tr>
<tr>
<td>Expected value</td>
<td>15</td>
<td>15</td>
<td>15</td>
<td>15</td>
</tr>
</tbody>
</table>

Source: Cunningham & Mackerras (1998)

Both ways of analysing weight in relation to height show that there is a very wide range. A particularly notable and unexpected feature was that the proportion of children between the 85–95th and 5–15th centiles tended to be less than the proportion above the 95th or below the 5th centiles whereas twice the proportion would have been expected (Cunningham & Mackerras 1998).
This national survey indicates that there is enormous variability in the growth of Indigenous children around the country. Health promotion programs need to consider the local situation. These data also indicate that stunting is an important problem even in this age group. The extent of the stunting problem would not have been guessed if only weight data were collected. Stunting generally commences much earlier in life.

Preschool-aged children

Children younger than five years were not measured in the NATSIS. The information that is available comes almost exclusively from studies done in remote areas. No recent studies reporting the growth of preschool-aged Indigenous children in the capital cities could be found. Most studies show that growth in remote areas is good in the first four to six months. After this age, growth frequently slows at around 12 months (average below -1.0 Z-score) and remains there. Growth in height and head circumference is also slowed (Skull et al 1997). There seems to be little catch-up growth. This is consistent with the observations from the NATSIS showing that children aged five to nine years living in rural areas are shorter than expected.

In some remote and rural areas, a substantial proportion of Indigenous preschool children over six months have an unacceptable level of malnutrition. The minimum prevalence of malnutrition (defined as either weight-for-height or height-for-age below the 3rd centile) was 20 per cent in children aged less than two years in the Darwin region (Ruben & Walker 1995). Muller et al (1995) reported lower prevalence but also noted that the prevalence varied by season in the Katherine region of the Northern Territory.

There are improvements in some locations. Dugdale et al (1994) reported that the median weights-for-age of Aboriginal infants at birth, three, six, nine and twelve months living at Cherbourg had improved consistently since the 1950s and had almost achieved the international reference medians.

Torres Strait Islander children

There is little specific information about the growth of Torres Strait Islander children. Figures 9.12 and 9.13 show the results of a survey done on one island (Vlack et al 1993). As most of the children lie between the 3rd and 97th centile, these results suggest that growth failure is not occurring in that population and there was no indication of general faltering of growth in the second half of infancy. In fact, these results show a larger than expected proportion of children over the 97th centile for weight.
Figure 9.12 Weight-for-age, Torres Strait Island children, 1992

Note: Sexes combined for simplicity of presentation.

Figure 9.13 Height-for-age, Torres Strait Island children, 1992

Note: Sexes combined for simplicity of presentation.
9.3 Risks and associated factors

At a population level, growth failure in young children is due to inadequate energy and nutrient intake in relation to needs. However, this does not mean that faulty diet is the only cause, or always the major cause. Infection increases dietary energy needs, and repeated infections during childhood are a common cause of growth failure in many less developed countries. Repeated infections are also common in many Aboriginal communities. Inadequate dietary intake causes weight loss and/or growth failure, and results in low nutritional reserves (Tomkins & Watson 1989) and eventually malnutrition (Figure 9.14).

Although it is still accepted that malnutrition exacerbates the severity of diarrhoeal disease, it is no longer always accepted that, at the population level, diarrhoeal diseases predispose to poor growth owing to the potential for catch-up growth. Several studies in the Gambia (Poskitt et al 1999) and the Kimberley (Rousham & Gracey 1998) have shown a reduction in diarrhoeal disease and general morbidity rates without any concomitant improvement in growth. This has lead to a downplaying of the importance of infection and more focus on inadequate introduction of solids and malabsorption due to gut damage.

Figure 9.14 Some factors that cause childhood malnutrition

Source: Adapted from Tomkins & Watson (1989) and other sources.
With malnutrition and vitamin A deficiency there can be progressive damage to mucosal surfaces, such as the gut, with lowered resistance to microbial colonisation and invasion by pathogens. If the two major defence mechanisms are compromised, diseases have potentially increased incidence, severity and duration. The disease process exacerbates the loss of nutrients, by both the metabolic response and by physical loss from the intestine. Concurrently, many diseases result in loss of appetite causing further decreases in dietary intake (Waterlow 1994). This cycle begins in utero and can have detrimental effects on adult health status with poor maternal and infant diet contributing to immune deficiency and making the infant more susceptible to the deferred effects of infection (Figure 9.15).

**Figure 9.15 Early influences on health in later life**

![Diagram showing early influences on health in later life](image)


**9.4 Health consequences**

Psychomotor development is delayed in populations with a high prevalence of childhood malnutrition. Such groups also suffer from many other social disadvantages so it is difficult to disentangle the effects of malnutrition *per se* from the effects of general social deprivation. Malnourished children have lower levels of activity which reduces environmental exploration (Grantham-McGregor 1992). When severe malnutrition occurs in the first two years of life in the presence of
sociocultural deprivation it appears to have a detrimental effect on mental development which lasts at least through childhood. It also appears that the role of stimulation may be critical (Grantham-McGregor et al 1991). Sociocultural stimulation should therefore be a part of the treatment of malnourished children.

A study of Aboriginal primary school children, using a simple, apparently non-culture-specific test, found no evidence of delayed visual-motor developments (Dugdale et al 1975). The teacher's assessments of poor school performance were related to nutritional status, school absenteeism and measures of personal hygiene.

In most children, 'energy malnutrition' usually reflects a total shortage of food causing a deficiency in both energy and all other nutrients. Energy malnutrition in early childhood is associated with growth restriction, impaired neurological development, anaemia and increased susceptibility to infection. The interactions of infections and nutritional status are complex. Three key factors are: anorexia (loss of appetite), malabsorption, and catabolic losses (Keusch 1990). Fever increases the resting metabolic rate by 15 to 40 per cent and also causes anorexia. The combined result of these and other metabolic alterations is wasting. This can be remedied by catch-up growth during convalescence, which can exceed seven times the normal growth rate. This rate of catch-up growth would theoretically require 14 per cent more energy and 8 per cent more protein than normal requirements.

Monotony of the diet can provoke nutrient deficiency, causing anorexia which in turn leads to further weight loss, making the child susceptible to infection and further anorexia (Golden 1991). Anorexia with infection seems to be a marker for metabolic abnormalities or systemic infectious complications, and may be mediated by cytokines.

The growth of Child A in Figures 9.9 and 9.10 is a commonly observed growth pattern in remote areas during the first two years of life. Growth appears to be satisfactory in the first months of life but then slows for about a year and improves at around 18 to 24 months of age. This type of pattern is usually associated with repeated infections and the non-introduction of adequate quantities of complementary foods at four to six months (Gracey & Sullivan 1989; Hurley 1993). Poor countries with high mortality rates also tend to have a high prevalence rates of stunted children. Stunting starts early in infancy in developing countries with growth rates often returning to normal, but without full catch-up growth, after the age of three years which leaves the child with a height deficit. Children recovering from malnutrition do not start to catch-up in height until after they have achieved at least 85 per cent of expected weight-for-height, with the most stunted children growing the fastest (Waterlow 1994).

Malnutrition in the first two to three years of childhood either irrevocably reduces the child's growth potential, or full catch-up growth is possible but may not occur in the environment in which these children live because of recurrent infections with intestinal malabsorption or insufficient quantities of additional food to allow catch-up growth to occur. The relationship of bone age to height age is crucial, since the bone age (maturity) of stunted children is as much delayed as height growth. This means that malnourished children may retain their capacity for full catch-up.
Indeed, studies of growth following malnutrition, immigration, adoption or deprivation have documented this potential for almost complete catch-up (Golden 1994).

9.5 What can be done about it?

The achievement of normal nutrition and growth during infancy and childhood is extraordinarily complex and is often very difficult in impoverished, unhygienic circumstances. This is despite it being accepted as the norm in communities where standards of living and hygiene are high, where food shortages are non-existent and where maternal knowledge and practices about infant and child nutrition and hygiene are well founded.

As discussed in Chapter 7, breast-feeding for at least four to six months is one of the most important factors in supporting the growth of infants. Bottle-feeding has serious risks such as contamination of the infant’s feeds or bottles with harmful pathogens. Infant feeding formulae can also easily be made up at the wrong concentration or with unbalanced non-human milks, such as unfortified cow’s milk. The introduction of solids in sufficient quantities at four to six months of age is as important as breast-feeding for ensuring good child growth (see Subsection 8.3).

Monitoring of a child’s growth should examine the pattern of the growth in relation to the reference curves. Intervention with the mother should occur when the growth trajectory starts to flatten in comparison to the curves. Intervention should not be delayed until the child has dropped below the bottom line on the chart.

In some parts of the country, it is common either for malnourished Aboriginal children to be admitted to hospital for rehabilitation, or for malnourished children admitted for other reasons to be kept in hospital for rehabilitation. The rapid weight gain of up to 20 times normal during nutritional rehabilitation is only proportional to energy intake if there is no infection or specific nutrient deficiencies. One of the consequences of the latter is an inability to utilise an excess of energy intake, leading to anorexia (Monckeberg 1990). A dietary protein intake of 3 g/kg/day and energy of 400–600 kilojoule/kg/day is generally recommended during recovery from stress or infection (Feigin 1977; Scrimshaw 1992). It has been alleged that Aboriginal children frequently lose the weight gained in hospital after discharge. The long-term effects of this weight cycling are unknown. It would seem more sensible to have community-based programs directed at early detection and intervention than to use the hospital system for intensive crisis care.

Access to curative health care can clearly affect the malnutrition-morbidity synergism. However, improved health care can reduce childhood mortality without improvements in nutritional status, as illustrated by community health research units in Africa and the experience of Aboriginals in the Northern Territory (Walker et al 1988; Pison et al 1993; Walker 1994). Therefore, approaches to eradicating malnutrition and its consequences need to be multi-faceted and include improved physical infrastructure, better food supplies, health and nutrition education and access to clinical care.
Management of malnutrition

The slow onset of under-nutrition means that it will also take time to reverse its effects.

A child who is 12 months behind in height growth, will have to grow at twice the rate of a normal child of their current height-age for at least 12 months to catch up — this is faster than twice the rate of a normal child of his chronological age because the rate of normal growth decreases with age. The older the child is and the further behind in growth, the longer they will have to maintain a faster rate of growth for full catch-up.

The following points are important in the management of malnutrition in children:

• children showing growth poor growth need to eat substantial amounts of extra food for as many months as the poor growth has been evident on the growth chart;

• starting this intervention early when the growth line flattens is much more likely to lead to achieving the potential for adult size than waiting till the child’s line drops below the bottom line on the chart before intervening;

• having a program directed at helping young children (one to three years) is more likely to be effective in the long term than having a program directed as school-age children;

• the most effective action would be to prevent poor growth by encouraging the introduction of adequate quantities of solid food as well as breast-milk or infant formula at the recommended times.

Source: Golden (1994).

Programs in Australia

The Fitzroy Crossing ‘Failure to Thrive’ (FTT) project, undertaken in the late 1980s, examined the effect of interventions (health education addressing hygiene, parasites, nutrition, environmental health, scabies, breast-feeding, contraception and diarrhoea) over a two-year period on the prevalence of FTT in two (rural and remote) communities. Results indicated that FTT was delayed on average by about two months, but the effects were not able to be solely attributed to the intervention. The study showed that health education only marginally improved growth and health in these communities and that there was a need for community ownership and empowerment in all aspects of daily life for significant improvements in health status to occur (Sullivan & Gracey 1990).

The Ngunyti Tjitji Pirni (NTP) project in Western Australia aims to improve the health of families by educating and empowering Aboriginal mothers through strategies to maintain health and treat minor illnesses at home as well as providing early intervention and referral when problems are identified (Engeler et al 1998).

The program involved maintenance of a health record system for participating women and children and the provision of health education. Family nutrition was emphasised in morning and afternoon teas. Cooking demonstrations focused on healthy foods and how to prepare them. A library of health promotion material on
these subjects was available for health workers and clients. Evaluation of the three year program showed:

- improved provision of immunisation and earlier medical and hospital referrals for children;
- mothers liked regular home visits and health, nutrition and immunisation advice given by health workers; and
- those who participated in playgroup services considered the sessions to be good for them and their children.

**Dietary guidelines for children and adolescents**

The *Australian Dietary Guidelines for Children and Adolescents* were developed in response to concerns that the *Dietary Guidelines for Australians* (NHMRC 1992a) were for adults and thus not appropriate for children and adolescents. The *Dietary Guidelines for Children and Adolescents* were designed for the general population of healthy children (ages 0–18 years), and to emphasise the development of healthy eating habits. Although many Indigenous children have poorer health, including nutritional status, these recommendations remain pertinent to improving the nutritional status of Indigenous children (see Appendix 3).

The differences between guidelines for children and adults make it hard to give simple messages and care should be taken to ensure that the appropriate guidelines are followed. Anecdotal reports from paediatricians in Australia indicate that some non-Indigenous people apply the adults’ low-fat message to their children’s diets so zealously that their children fail to grow properly. Table 9.3 shows the gradual progression from fat levels recommended for infants to those recommended for adults, highlighting the differences between the guidelines.

**Table 9.3 Quantitative guidelines for Australian children and adolescents**

<table>
<thead>
<tr>
<th>Age (approx)</th>
<th>% energy from fat</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–6 months</td>
<td>50 per cent</td>
<td>Fat supplies half the energy content of breast-milk</td>
</tr>
<tr>
<td>6–24 months</td>
<td>40 per cent</td>
<td>As complementary foods become more important, the contribution of carbohydrate to energy increases. Low-fat and skim milks should not be given to children of this age</td>
</tr>
<tr>
<td>2–5 years</td>
<td>35–40 per cent</td>
<td>Increasing importance of solid foods in the diet. Low fat and skim milk should not be given to children under 5 years of age</td>
</tr>
<tr>
<td>5–14 years</td>
<td>35 per cent</td>
<td>And no more than 10 per cent of energy coming from saturated fat</td>
</tr>
<tr>
<td>15 years and older</td>
<td>30 per cent</td>
<td>And not more than 10 per cent of energy coming from saturated fat, ie the same guideline as for adults.</td>
</tr>
</tbody>
</table>

Iron-deficiency anaemia occurs when the amount of iron in the body is less than that required for normal formation of haemoglobin, iron enzymes, and other functioning iron compounds (US Department of Health and Human Services 1988; US National Research Council 1989). Iron deficiency remains the most common nutrient deficiency in the world (NHMR 1992a).

There are numerous causes of iron deficiency. In infants and children, because rapid growth imposes large iron needs and the bioavailability of iron in the infants diet is low (Dallman et al 1980; MacPhail & Bothwell 1989), inappropriate nutrition is likely to play a major role (Lovric et al 1972). In women of child-bearing age, iron loss resulting from pregnancy or heavy menstrual periods may, on a long-term basis, not be matched by dietary iron. In men and in post-menopausal women the major cause of iron deficiency is pathological bleeding. Most commonly, this occurs from the gastrointestinal tract (eg peptic ulcers, gastritis, haemorrhoids, tumours, hookworm infestation).

Iron-deficiency anaemia is usually diagnosed by measuring the concentration of plasma iron, together with the concentration of the iron-binding protein, transferrin. A low level of plasma iron and an elevated level of transferrin are characteristic of iron deficiency. Concentrations of serum ferritin below 10 micrograms/Litre indicate iron deficiency. Iron-deficiency anaemia is characterised by red cells becoming smaller than normal and having a low concentration of haemoglobin (Roeser 1990).

Low iron stores represent a state of vulnerability for progression to iron deficiency and iron-deficiency anaemia. Low serum ferritin indicates a lack of iron stores in the liver and is the first sign of iron depletion (Cook & Finch 1979; de Maeyer 1989; Gibson 1990). However, levels of haemoglobin, transferrin, and serum ferritin, are not only indicators of iron status. For example, serum ferritin may be high when there is an infection or inflammatory disease. Serum transferrin may be low due to inflammation (Cook & Finch 1979). Haemoglobin may be low when there is high exposure to environmental lead or gastrointestinal bleeding due to parasites and, in some tropical and developing countries because of vitamin A deficiency and low oxygen tension at high altitudes (Gibson 1990). Hence, several measures are usually required to determine the prevalence of iron deficiency in a population.

Iron absorption is affected by many factors. Haem iron is present in meats, poultry and fish and is more efficiently absorbed than inorganic (non-haem) iron, which is found in plant as well as animal foods. Ascorbic acid facilitates the absorption of non-haem iron, but dietary fibre, phytates and certain trace elements may diminish it (Roeser 1990).
10.1 Extent of the problem

There is little contemporary population-based data on iron deficiency. Based on dated rural and remote surveys, the prevalence of iron deficiency in Aboriginal groups is much higher than in the general population (Jose & Welch 1970).

Chronic infections are common in AboriginaIs and as these cause an increase in ferritin levels, studies which do not account for this may underestimate the extent of iron deficiency in children. Harris et al (1988) and Lawrence et al (1992) comment that intestinal parasites were not common in their study populations, and it was therefore difficult to explain the high levels of iron deficiency observed.

Surveys done from the 1950s to 1980s showed that iron deficiency and anaemia were prevalent in Aboriginal people of both sexes, especially children (see Cobiac & Baghurst 1993, for review). Work done in western New South Wales (Harris et al 1988) and in the Top End of the Northern Territory (Watson & Tozer 1986) showed that the rate of anaemia (11 to 12 per cent) in Aboriginal children was much higher than the estimated 3 per cent in non-Aboriginal children (Lovric 1970; Lovric et al 1972).

In recent years, the prevalence of documented anaemia in pregnancy has been increasing in both Aboriginal and non-Aboriginal mothers in the Northern Territory. This may be due to improved access to antenatal care and surveillance rather than an increase in true prevalence (Markey et al 1998). Figure 10.1 shows the prevalence of anaemia in Aboriginal women compared to that in non-Aboriginal women.

Figure 10.1 Proportion of mothers who were diagnosed with anaemia during their pregnancy, Northern Territory, 1986-1995


10.2 Risks and associated factors

Several factors have been reported to be associated with poor iron status in infants and young children including low birthweight (Friel et al 1990), low socio-economic status (Vazques-Seoanne et al 1985), early introduction of cow's milk (before 12 months) (Tunnessen & Oski 1987), excessive intake of cow's milk after 12 months (Mills 1990), late introduction of solids (Simes et al 1984; Calvo et al...
1992), and low total iron intake (Hercberg et al 1987). Also, over reliance on breast-feeding as a source of nutrition in older infants, who should also be having weaning foods, may contribute to low dietary iron intakes.

Pre-term birth has also been linked to anaemia in the mother although some of the same studies did not find that low ferritin levels had the same effect (Cook et al 1994). Hence, it is not clear whether these findings are due to low haemoglobin, blood volume expansion or iron deficiency per se.

Tea drinking reduces the absorption of non-haem iron (de Maeyer 1989). A number of studies report that young Aboriginal children often drink tea and this may be one reason why the anaemia rates are so high (Jose & Welch 1970; Lawrence et al 1992).

Figure 10.2 Absorption of iron from different breakfasts

![Graph showing iron absorption from different breakfasts](image)

Note: The continental breakfast consisted of coffee, tea or chocolate milk (150 mL), two wheat rolls with margarine (12 mg), one with orange marmalade (10 g) and the other with cheese (15 g). Additions to this were: cornflakes (21 g) and milk (250 mL); boiled egg (60 g); scrambled egg (60 g) and fried bacon (50 g before cooking); orange juice 150 mL. The values represent absorption in subjects having 40 per cent absorption from oral reference doses of ferrous ascorbate (3 mg Fe).


Hookworm infections are present in some coastal and inland communities in the north of Australia (Hopkins et al 1997). Hookworm disease can cause iron-deficiency anaemia resulting from gastrointestinal blood loss caused by worms attaching to the small-intestinal mucosa. The severity of disease is influenced by the total worm burden, the level of dietary iron intake and the level of physiological iron loss. A survey of a community in the north of Western Australia showed that hookworm infections were endemic in Aboriginals, and were likely to contribute to the high prevalence of iron deficiency and anaemia that were observed in the Aboriginal population, particularly in young children and in women. Inadequate dietary iron intake contributed to the high levels of iron deficiency (50 per cent) as 31 per cent of Aboriginal women aged over 14 years who were hookworm-negative had iron-deficiency anaemia (Hopkins et al 1997).
Intestinal parasites

Some gastrointestinal worms and parasites can cause acute or chronic diarrhoea, malabsorption and/or anaemia. In some instances the anaemia is of the iron-deficiency type, usually when it is associated with gastrointestinal blood loss. However, in other instances anaemia might be of some other type, such as megaloblastic or macrocytic (Charters 1983).

- **Hookworms** (*Ancylostoma duodenale* or *Necatur americanus*) are well known to cause gastrointestinal blood loss and iron-deficiency anaemia as a result. Infestations with hookworms occur in northern Australia, particularly in areas of heavy rainfall.

- **Whipworms** (*Trichuris trichiura*) can occur in persons who are symptomless. They can also cause eosinophilia, dysentery, abdominal discomfort or pain, anorexia and, especially in children, anal prolapse. Heavy infestations can cause blood loss, iron deficiency and microcytic anaemia.

- **Strongyloidiasis** (*S. stercoralis*) can cause severe diarrhoea and malabsorption.

- **Giardia intestinalis** (*G. lamblia*) can cause acute or chronic diarrhoea which, if sufficiently severe and prolonged, can cause growth faltering and failure-to-thrive. Chronic diarrhoea in these circumstances can be associated with anaemia which is usually normochromic or macrocytic rather than microcytic which is the characteristic pattern in iron deficiency.

- Dogs can carry a ‘dog type’ *Giardia* or types that are able to infect humans and dogs. In Aboriginal communities, where dogs are just as likely to swallow *Giardia* cysts of either the ‘dog’ or ‘human’ type, the ‘dog’ type predominates but the animals are effective mechanical carriers of the ‘human’ type of the parasite (Thompson 1998).

- **Ancylostoma caninum**, the commonest nematode parasite of dogs, is not associated with chronic blood loss and anaemia in humans but it is possible that human infections from diseased animals can cause eosinophilic enteritis as there have been several reports from tropical Australia (Schad 1994).

- The iron status of Indigenous children who are infected with intestinal worms or parasites is also affected by other factors such as the presence of co-existing infections and their dietary iron intake.
10.3 Health consequences

Iron-deficiency anaemia in infants and children is associated with lower scores on tests of development, learning and school achievement (Oski et al 1983; Haas & Wilson Fairchild 1989; Lozoff et al 1991). Prospective studies in Costa Rica and Chile have demonstrated that the effects of iron-deficiency anaemia on intelligence may be irreversible (Walter et al 1989; Lozoff et al 1991). In adults, diminished levels of serum iron have been linked to significant reductions in work productivity (Basta et al 1979; Cook et al 1994) and mental performance (Soemantri et al 1985).

10.4 What can be done about it?

It is important to prevent and, if needed, to adequately treat iron deficiency in pregnancy. A diet rich in iron is always the preferred basis of prevention and should be combined with any iron supplement administration if that is needed. High absorbable iron foods are those that are rich in haem iron. These include cooked lean beef, cooked skinless chicken breast and cooked fish. Low absorbable iron foods include iron-fortified breakfast cereal, and cooked or canned beans or lentils. Spinach and broccoli contain reasonable amounts of iron but it is mostly poorly absorbed. Iron absorption boosters include vitamin C-rich foods and fruit juices. Tea, coffee and fibre are inhibitors of iron absorption.

Similar dietary principles apply for promoting iron absorption in infants aged six to twelve months and in older children. That is, high-iron foods such as beef, lamb and liver as well as medium-iron food sources such as pork, chicken and fish should be offered. Food combinations can be used to promote iron absorption. For example iron absorption from cereals, vegetables, legumes, eggs or nut pastes can be improved by giving meat and vitamin C as fruit or juice. Vegetables such as pumpkin, potato, zucchini and broccoli can be mixed with meats until they are a smooth consistency. Well mashed foods are useful and palatable for infants often from about six months of age. After seven to nine months of age most infants can manage a thicker consistency such as that of minced meat. Coarse textured and finger foods can be introduced later. As discussed, tea reduces iron absorption and should not be given to young children.

In the early 1970s, iron-deficiency anaemia was reduced by 50 per cent when white bread fortified with iron and vitamin B1 and niacin was made available to an Aboriginal population for approximately six months (Kamien et al 1975). The authors acknowledged that the reduction in iron-deficiency anaemia which subsequently occurred in children may have been due to other factors. However, the results indicate that fortification of bread may have health benefits to the community.

Intestinal parasites are common in the wet tropics (Holt et al 1980) and regular de-worming is often carried out. A combination of mebendazole and iron was found to be the most effective way to reduce anaemia in Arnhem Land (Fraser 1996). An annual single dose of mebendazole failed to prevent anaemia in the study population, but there was a significant weight improvement in children with failure-to-thrive and among apparently healthy children.
A recent study from Indonesia found that twice-weekly supplementation of iron was as effective as daily supplementation (Schultink et al 1995) and this has also been shown in an Aboriginal community (Kruske et al 1999). This may be simply due to improved patient compliance or it may be that the mucosal block engendered by a high dose prevents absorption for several days, and thus much of the daily iron is wasted.

As discussed in Chapter 3, the prevalence of iron-deficiency anaemia and other anaemias (particularly macrocytic anaemia due to folate deficiency) needs to be assessed amongst contemporary Indigenous groups. In many community studies in remote areas (such as at Minjilang [Lee et al 1994a]) iron-deficiency anaemia has not been found to be a problem even amongst adult women. This may be due to a high iron intake due to a more traditional meat-based diet (Lee 1992). However, iron deficiency may remain more of a problem amongst children, particularly in urban areas.

Medical aspects of the treatment of iron deficiency and/or iron-deficiency anaemia, including the use of iron-containing medications and injectable iron, are outside the scope of this publication.
Iron deficiency is the most common nutritional deficiency in Australia, and in the world as a whole. Not everyone who has iron deficiency is anaemic, with anaemia only occurring towards the end of the process of falling iron stores.

What causes iron deficiency?

- **Children** — the most likely cause of iron deficiency in children is an inadequate amount of iron in the diet, coupled with the extra requirement for iron because of growth. The dietary patterns that most often lead to iron deficiency are prolonged exclusive breast-feeding, delayed introduction of solids and over-use of cow’s milk and cow’s milk substitutes (e.g., powdered milk).

- **Adolescents** — In this age group, the combination of the extra iron requirements of the growth spurt, poor eating habits and onset of menstruation combine to produce a high prevalence of iron deficiency.

- **Women** — Causes of iron deficiency in women are usually dietary or through iron loss in menstruation, pregnancy or breast-feeding.

- **Other adults** — Dietary deficiency and chronic diseases, including intestinal disease and cancers, can cause iron deficiency in men and in women beyond their reproductive years.

Absorption of iron

- **All meat sources promote the absorption of iron from other foods and even small amounts of meat or fish may improve the availability of iron from other foods, such as rice.**

- **Drinks are not a source of iron in themselves but the drinks chosen to accompany a meal may influence the amount of iron available to the body. Fruit juices or drinks with vitamin C can improve iron absorption. Tea and, to a lesser extent, coffee reduce the amount of iron available for absorption.**
### Iron content of common children’s foods

<table>
<thead>
<tr>
<th>High absorbable iron foods</th>
<th>Total iron</th>
<th>Absorbed iron</th>
<th>Requirement (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lean beef, 90g</td>
<td>4.9</td>
<td>2.0</td>
<td>1.0</td>
</tr>
<tr>
<td>Chicken breast (baked), 120g</td>
<td>3.4</td>
<td>1.5</td>
<td>0.5</td>
</tr>
<tr>
<td>Ham, 60g</td>
<td>2.0</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>Baked beans, 120g</td>
<td>15.0</td>
<td>10.0</td>
<td>2.0</td>
</tr>
<tr>
<td>Boiled spinach, 70g</td>
<td>4.9</td>
<td>3.4</td>
<td>1.5</td>
</tr>
<tr>
<td>Iron-fortified cereal, 30g</td>
<td>15.0</td>
<td>10.0</td>
<td>2.5</td>
</tr>
<tr>
<td>Weetbix™, 30g</td>
<td>15.0</td>
<td>10.0</td>
<td>2.5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Low absorbable iron foods</th>
<th>Total iron</th>
<th>Absorbed iron</th>
<th>Requirement (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cooked broccoli, 60g</td>
<td>2.0</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>Cooked English spinach, 60g</td>
<td>1.0</td>
<td>0.5</td>
<td>0.5</td>
</tr>
<tr>
<td>L medium</td>
<td>1.0</td>
<td>0.5</td>
<td>0.5</td>
</tr>
<tr>
<td>Boiled egg, 1 medium</td>
<td>1.0</td>
<td>0.5</td>
<td>0.5</td>
</tr>
<tr>
<td>Cooked/canned legumes, 120g</td>
<td>3.4</td>
<td>2.0</td>
<td>1.0</td>
</tr>
<tr>
<td>Iron-fortified cereal, 40g</td>
<td>3.4</td>
<td>2.0</td>
<td>1.0</td>
</tr>
</tbody>
</table>

### Iron content of common adult foods

<table>
<thead>
<tr>
<th>High absorbable iron foods</th>
<th>Total iron</th>
<th>Absorbed iron</th>
<th>Requirement (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lean beef, 125g</td>
<td>4.9</td>
<td>2.0</td>
<td>1.0</td>
</tr>
<tr>
<td>Chicken breast (skinless), 125g</td>
<td>3.4</td>
<td>1.5</td>
<td>0.5</td>
</tr>
<tr>
<td>Fish, 125g</td>
<td>2.0</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>Baked beans, 120g</td>
<td>15.0</td>
<td>10.0</td>
<td>2.5</td>
</tr>
<tr>
<td>Boiled spinach, 70g</td>
<td>4.9</td>
<td>3.4</td>
<td>1.5</td>
</tr>
<tr>
<td>Iron-fortified cereal, 30g</td>
<td>15.0</td>
<td>10.0</td>
<td>2.5</td>
</tr>
<tr>
<td>Weetbix™, 30g</td>
<td>15.0</td>
<td>10.0</td>
<td>2.5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Low absorbable iron foods</th>
<th>Total iron</th>
<th>Absorbed iron</th>
<th>Requirement (mg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cooked broccoli, 60g</td>
<td>2.0</td>
<td>1.0</td>
<td>1.0</td>
</tr>
<tr>
<td>Cooked English spinach, 60g</td>
<td>1.0</td>
<td>0.5</td>
<td>0.5</td>
</tr>
<tr>
<td>L medium</td>
<td>1.0</td>
<td>0.5</td>
<td>0.5</td>
</tr>
<tr>
<td>Boiled egg, 1 medium</td>
<td>1.0</td>
<td>0.5</td>
<td>0.5</td>
</tr>
<tr>
<td>Cooked/canned legumes, 120g</td>
<td>3.4</td>
<td>2.0</td>
<td>1.0</td>
</tr>
<tr>
<td>Iron-fortified cereal, 40g</td>
<td>3.4</td>
<td>2.0</td>
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</tr>
</tbody>
</table>

SECTION III

NUTRITION-RELATED CONDITIONS
Obesity is a complex medical disorder which is significant to populations worldwide. The prevalence of excess weight is increasing in Australia as a whole (NHMRC 1997c). The NATSI showed variability in weight-for-height among the Indigenous population for all geographic locations but found that Indigenous adults were more likely to be obese than their non-Indigenous counterparts (Cunningham & MacKerras 1998).

Overweight and obesity are terms that describe body weight (ie mass) in relation to ‘normal’ values for males and females. Conventionally, these terms are related to body mass index (or BMI) in adults. This is a simple index which is calculated as follows:

\[
\text{BMI} = \frac{\text{body weight (in kg)}}{\text{height (in metres)}^2}
\]

This ratio provides an approximation of adiposity and it has a strong association with morbidity and mortality (WHO Expert Committee on Physical Status 1995). In Australia it has been customary to accept the body mass index range of 20 to 25 as indicating healthy weight range, 25 to 30 indicating ‘overweight’ and a body mass index in excess of 30 reflecting ‘obesity’. The WHO definitions are given in Table 11.1.

Table 11.1  WHO definitions of body mass index for adults

<table>
<thead>
<tr>
<th>Category</th>
<th>Body mass index range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Underweight</td>
<td>&lt;16</td>
</tr>
<tr>
<td>Severe thinness (WHO grade 3)</td>
<td>16 to &lt;17</td>
</tr>
<tr>
<td>Moderate thinness (WHO grade 2)</td>
<td>17 to &lt;18.5</td>
</tr>
<tr>
<td>Mild thinness (WHO grade 1)</td>
<td>18.5 to &lt;20</td>
</tr>
<tr>
<td>Normal or acceptable weight</td>
<td>20 to &lt;25</td>
</tr>
<tr>
<td>Overweight</td>
<td></td>
</tr>
<tr>
<td>Overweight (WHO grade 1)</td>
<td>25 to &lt;30</td>
</tr>
<tr>
<td>Obese (WHO grade 2)</td>
<td>30 to &lt;40</td>
</tr>
<tr>
<td>Obese (WHO grade 3)</td>
<td>40 +</td>
</tr>
</tbody>
</table>


However, this simple approach, though practical, does not accurately define adiposity and associated risks in all persons. The body mass index is not valid at the extremes of life (including infancy and childhood) and in those who are very fit and...
Nutrition in Aboriginal and Torres Strait Islander Peoples

11.1 Impact of changed lifestyles

As discussed in Chapter 3, before European contact and colonisation of Australia, Aboriginal people were believed to be slim and not to suffer from cardiovascular disease and diabetes (Kirk 1981). A group of Aboriginal people newly emerged from the Great Sandy Desert in Western Australia about 30 years ago were described as ‘slimly built, sinewy feather-weights’ whose average height was 167.1 cm. Only three of the 22 men examined weighed more than 56 kg (Elphinstone 1971). In the mid-1960s a cross-sectional study of more than 550 Aboriginal people (adults and subjects down to infancy) showed that they were particularly lean (Abbie 1967). However, it should be noted that subjects were physically active and had a diet derived from wild animals (lean meat low in saturated fats) and uncultivated plant foods (Abbie 1967).

By the early 1970s, it was considered that ‘modern Aboriginals appear to be affected to the same extent as modern [non-Indigenous] Australians by diseases of the cardiovascular system if they survive to and beyond middle age’ despite there being ‘no data for incidence in respect to most chronic progressive diseases in the Aboriginal population’ (Moodie 1973). It is recognised now that these diseases have become major causes of morbidity and deaths in Aboriginal adults.

11.2 Extent of the problem

The NATSIS included measurements of height and weight on those persons aged five years and older who agreed to these measurements being done. Among adults aged 18 years or more, about 25 per cent of Indigenous males and 28 per cent of Indigenous females were classified as obese (body mass index of 30 or more), compared with about 19 per cent of all Australian males and females aged 19 years and over in the NNS. Torres Strait Islander adults in the NATSIS tended to have a higher body mass index than Aboriginal adults (Cunningham & Mackerras 1998).
Figure 11.1 Distribution of overweight or obese Indigenous males, 18 years and over, 1994


Figure 11.2 Distribution of overweight or obese Indigenous females, 18 years and over, 1994

The body mass index distributions of adults (18 years and older) were calculated and divided into the four categories described by the NHMRC (Mackerras & Cunningham 1996). The percentage distribution of body mass index by sex, race and location of residence is shown in Table 11.2.

### Table 11.2 Body mass index distribution of Indigenous adults, 1994

<table>
<thead>
<tr>
<th>BMI group</th>
<th>Race</th>
<th>Geographical location</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>All Indigenous (%)</td>
<td>Capital city (%)</td>
</tr>
<tr>
<td></td>
<td>Aboriginal (%)</td>
<td>Other urban (%)</td>
</tr>
<tr>
<td></td>
<td>TSI (%)</td>
<td>Rural (%)</td>
</tr>
<tr>
<td>Males</td>
<td></td>
<td></td>
</tr>
<tr>
<td>&lt;20</td>
<td>6.1 6.4 1.6</td>
<td>4.3 5.9 7.9</td>
</tr>
<tr>
<td>20 to 25</td>
<td>25.4 25.7 21.6</td>
<td>27.5 24.6 24.6</td>
</tr>
<tr>
<td>&gt;25 to 30</td>
<td>27.8 27.7 25.6</td>
<td>31.1 28.7 23.8</td>
</tr>
<tr>
<td>&gt;30</td>
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<td>21.2 20.0 15.1</td>
</tr>
<tr>
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<td>15.9 20.8 28.6</td>
</tr>
<tr>
<td>Females</td>
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<td></td>
</tr>
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<td>11.4 9.0 7.6</td>
</tr>
<tr>
<td>20 to 25</td>
<td>21.2 21.8 12.5</td>
<td>22.3 21.8 19.3</td>
</tr>
<tr>
<td>&gt;25 to 30</td>
<td>20.4 20.3 22.6</td>
<td>22.9 21.5 16.5</td>
</tr>
<tr>
<td>&gt;30</td>
<td>20.3 19.1 39.0</td>
<td>21.9 20.9 17.9</td>
</tr>
<tr>
<td>Not measured</td>
<td>29.0 29.4 22.2</td>
<td>21.6 26.9 38.7</td>
</tr>
</tbody>
</table>

Notes: Persons identifying as both Aboriginal and Torres Strait Islanders (TSI) are excluded. ‘Rural’ applies to persons living in communities of less than 1,000 residents.


It is important to appreciate that overweight in Aboriginal people is not necessarily an ‘adult problem’ and that it probably has its origins in childhood or, perhaps, during intrauterine life (Dorner & Plagemann 1994). A study of 100 Australian Aboriginal children and adolescents in the Kimberley region in Western Australia in the late 1980s found that the prevalence of overweight in children aged seven to eighteen years was 2.7 per cent. Five years later the prevalence rate was 17.6 per cent (Braun et al 1996).
Central obesity

It is characteristic that adiposity in Aboriginal people has a central or ‘android’ pattern of distribution. This is the pattern that is usually associated with obesity in males. There tends to be little difference in this central or abdominal distribution of fat between Aboriginal men and women (Rutishauser & McKay 1986; O’Dea 1987; O’Dea et al 1990). The central distribution of obesity is associated with a high risk of development of Type 2 diabetes (Bjorntorp 1988a) and cardiovascular disease (Gensini et al 1998). Central obesity is also part of the metabolic Syndrome X (see Subsection 1.2).

A survey involving more than 300 Aboriginals and more than 550 non-Aboriginals aged one year or over living in the more settled south-eastern part of Australia found central obesity to be most prevalent among the Aboriginal women (Guest et al 1993a). Waist:hip ratios (a marker used to assess central obesity) were significantly greater among Aboriginal females aged 25–64 years than among their non-Aboriginal counterparts. In males, the mean ratio in Aboriginals and non-Aboriginals was the same. For females aged 20–49 years, 75 per cent of Aboriginals had a waist:hip ratio higher than the WHO cut-off point for increased risk of cardiovascular disease compared with a rate of 43 per cent in non-Aboriginals.

11.3 Health consequences

Obesity is closely associated with the risk factors for the main causes of morbidity and mortality in the Indigenous population — Type 2 diabetes (see Chapter 12), cardiovascular disease (see Chapter 13) and renal disease (see Chapter 14). The complications of obesity include cardiovascular disease (including high blood pressure which itself is a risk factor for cardiovascular disease and renal disease), metabolic disorders (such as Type 2 diabetes), respiratory disorders, gastrointestinal disease, Syndrome X and complications of pregnancy (Jung 1997) (Table 11.3)

Table 11.3 Morbidity associated with obesity

<table>
<thead>
<tr>
<th>Cardiovascular</th>
<th>Neurology</th>
<th>Urological</th>
</tr>
</thead>
<tbody>
<tr>
<td>Coronary heart disease</td>
<td>Nerve entrapment</td>
<td>Prostate cancer</td>
</tr>
<tr>
<td>High blood pressure</td>
<td>Hyperlipidaemia</td>
<td>Stress incontinence</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>Insulin resistance</td>
<td>Orthopaedic</td>
</tr>
<tr>
<td>Varicose veins</td>
<td>Diabetes</td>
<td>Osteoarthritis</td>
</tr>
<tr>
<td>Deep venous thrombosis</td>
<td>Menstrual irregularities</td>
<td>Gout</td>
</tr>
<tr>
<td>Respiratory</td>
<td>Renal</td>
<td>Endocrine</td>
</tr>
<tr>
<td>Breathlessness</td>
<td>Proteinuria</td>
<td>Growth hormone alterations</td>
</tr>
<tr>
<td>Sleep apnoea</td>
<td></td>
<td>Lowered prolactin response</td>
</tr>
<tr>
<td>Hypoventilation syndrome</td>
<td></td>
<td>Altered sex hormones</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>Breast</td>
<td>Pregnancy</td>
</tr>
<tr>
<td>Hiatus hernia</td>
<td>Breast cancer</td>
<td>Obstetric complications</td>
</tr>
<tr>
<td>Gallbladder disease</td>
<td>Male gynaecomastia</td>
<td>Caesarean surgery</td>
</tr>
<tr>
<td>Fatty liver, cirrhosis</td>
<td>Endometrial cancer</td>
<td>Macrosomia (large babies)</td>
</tr>
<tr>
<td>Haemorrhoids</td>
<td>Cervical cancer</td>
<td></td>
</tr>
<tr>
<td>Colorectal cancer</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Source: Jung (1997).
Diabetes
The risk of developing Type 2 diabetes rises continuously with increasing obesity and is approximately five to ten times greater in those with a body mass index greater than 30 than in those with a body mass index below 25 (Perry et al 1995; Shaten et al 1993). As well, overweight/obesity increases the predisposition of persons with diabetes to cardiovascular disease (Health & AIHW 1999a). The risk of diabetes has been shown to be markedly increased by even moderate overweight in Aboriginals in the first prospective study examining this question in Aboriginals (Daniel et al 1999).

Cardiovascular disease
Obesity has been associated with a higher risk of high blood pressure (Yong et al 1993; Huang et al 1998) and diabetes (see above) both of which are risk factors for coronary heart disease and stroke (Health & AIHW 1999b). Central obesity has been associated with greater cardiovascular risk than overall obesity (Rowley et al 1997; Prineas et al 1993).

11.4 What can be done about it?
Improved nutrition and the control of obesity are crucial to controlling the major causes of ill-health and death in Aboriginal and Torres Strait Islander peoples. A focus on primary prevention of overweight and obesity should target children and young people. This fits closely with promotion of healthy lifestyles (such as healthy eating and drinking, regular physical activity, no cigarette smoking) which will help reduce obesity but will also minimise the risks from related disorders such as cardiovascular disease and Type 2 diabetes.

Excessive energy intake (facilitated by the consumption of high energy-dense diets) is likely to be the most important factor increasing the risk of obesity. Reduction of dietary energy (kilojoule) intake can achieve weight loss in the short term, but compliance is difficult and the weight that has been lost is often regained gradually. Diet, behavioural change and regular physical activity are all important in maintaining long-term weight loss. It is important to realise that modest weight losses (3 to 5 kg) can have substantial health benefits and patients should be encouraged to set realistic, achievable goals. More substantial reductions in weight (10 kg) can lead to a broader range of health benefits in people who are obese (Table 11.4).
Table 11.4 Health benefits associated with sustained reduction in obesity by 10 kg

| Mortality              | 20 to 25% fall in total mortality  
|                       | 30 to 40% fall in diabetes-related deaths  
|                       | 40 to 50% fall in obesity-related cancer deaths  
| Blood pressure        | Fall of 10 mmHg systolic pressure  
|                       | Fall of 20 mmHg diastolic pressure  
| Angina                | Reduced symptoms by more than 90%  
|                       | 33% increased exercise tolerance  
| Lipids                | Fall by 10% in total cholesterol  
|                       | Fall by 15% in low density lipoprotein cholesterol  
|                       | Fall by 30% in triglycerides  
|                       | Increase by 8% in high density lipoprotein cholesterol  
| Diabetes              | Reduces risk of developing diabetes by > 50%  
|                       | Fall of 30 to 50% in fasting blood glucose  
|                       | Fall of 15% in glycosylated haemoglobin  

Source: Jung (1997).

The Homelands movement

It has been suggested that the ‘homelands movement’ with a return to traditional diet and lifestyle can have healthy influences on Aboriginal communities, including a reduction in overweight/obesity (McDermott et al 1998). Living in small isolated groups may promote health for Aboriginals if traditional lifestyles are followed but many other environmental factors have to be taken into account, including the regular availability of fresh fruit, vegetables and other nutritious foods. All 77 members of a very isolated community in the Great Sandy Desert (in Western Australia) took part in a recent survey which showed that children and 14 per cent of adults were undernourished. However, despite moving from a more settled mission environment several years previously, 22 per cent of adults were overweight and 40 per cent of women and 13 per cent of the men were obese. Central obesity was present in 90 per cent of the obese women and 48 per cent of the obese men (Gracey et al 1996).

Healthy Weight Program

The Healthy Weight Program, developed in Queensland, is a community-based weight management program for overweight Aboriginal and Torres Strait Islander adults. Aboriginal and Torres Strait Islander health workers are trained as facilitators in order to run the program in their communities. The aim of the program is for its participants to lose an achievable amount of weight. It covers topics such as healthy shopping, low-fat cooking and physical activity (Tropical Public Health Unit 1997).
An evaluation of the Healthy Weight Program in Queensland was conducted in 1999. Screening data was used to assess participant outcomes including weight loss and loss from waist and hip. Data was available on 260 participants. Of those who completed the first nine weeks of the program:

- 66 per cent of females and 79 per cent of males lost weight;
- 68 per cent of females and 71 per cent of males lost centimetres from their waists; and
- 70 per cent of females and 79 per cent of males lost centimetres from their hips.

The evaluation also indicated that the facilitators benefited greatly from the program in terms of improved nutrition knowledge, cooking skills and organisation and presentation skills (Dunn 1999).
Regular physical activity

Think of movement as an opportunity, not an inconvenience

- Underlying all education about increased physical activity for health must be a change in attitude towards movement.
- If all movement is regarded as an opportunity to improve health rather than as a time-wasting inconvenience, the benefits of modern technology can be enjoyed without the negative health consequences.

Be active every day in as many ways as you can

- The increase in effort-saving technology in modern societies has coincided with busier lifestyles. We not only have less need to be active, but seem to have less time.
- Small increases in daily activity can come from small changes carried out throughout the day. Make a habit of walking or cycling instead of driving or riding in a car; doing some gardening; going for a walk when you’re talking to a friend.
- It is important to remember that some activity is better than none, and more is better than a little.

Put together at least 30 minutes of moderate-intensity physical activity most days

- Improvements in body weight and blood pressure can result from putting together shorter amounts of moderate-intensity activities totalling a minimum of 30 minutes a day on most days, or doing 30 minutes continuously.
- A good example of moderate-intensity activity is brisk walking at a pace where you are able to comfortably talk but not sing. Other examples include digging in the garden, or medium-paced swimming or cycling.
- Moderate-intensity activity should be carried out for a minimum of about 10 minutes at a time without stopping. It is important to remember that the 30 minutes total need not be continuous. Three 10 minute walks are as good as one 30 minute walk.

Also enjoy some regular, strong activity for extra health and fitness

- Research has shown that able-bodied people can get added health and fitness benefits (beyond those achieved through increasing daily movement or regular moderate-intensity activity), by carrying out some regular strong physical activity.
- ‘Strong physical activity’ means that the activity makes you huff and puff and talking in full sentences between breaths is difficult. This can come from active sports such as football and basketball, or from fun activities such as aerobics or line dancing. For best results, this type of activity should be carried out for a minimum of around 30 minutes on three to four days a week.
- Although there’s no age barrier to carrying out strong activity, medical advice is recommended for those who have been inactive, who have heart disease or close relatives with heart disease, or who have major health problems. Strong activity in pregnancy is not recommended without strict medical supervision.
- Warm-up, cool-down, stretching and a gradual build-up from an inactive level are also recommended with strong physical activity.

Source: Adapted from Health (1999).
DIABETES

Diabetes is a chronic disease, characterised by hyperglycaemia or high levels of glucose in both the fasting and fed states. It is caused by deficient insulin production and/or resistance to its action. Over the course of the disease, diabetes can lead to a variety of complications including coronary heart disease, stroke, blindness, kidney problems and lower limb amputations (Health & AIHW 1999a). Diabetes can also lead to pregnancy-related complications, both for the mother and the fetus or newborn baby.

Because the common feature of diabetes is elevated blood glucose concentration, in the past it was considered to be a single disease. However, it is now clear that diabetes is caused by many different mechanisms and four major types of diabetes are recognised (Alberti & Zimmet 1998). In addition, impaired glucose tolerance defines a level of blood glucose that is intermediate between normal and diabetes. Because fasting blood glucose levels are in the normal range, impaired glucose tolerance can only be identified using an oral glucose tolerance test.

By far the most common form of diabetes worldwide is Type 2 diabetes (or non-insulin-dependent diabetes mellitus [NIDDM]), which is highly prevalent in the Australian Indigenous population. Type 2 diabetes results from a combination of abnormalities of insulin action and insulin secretion. Prospective studies indicate that prior to the onset of Type 2 diabetes, individuals develop insulin resistance (often secondary to overweight and/or physical inactivity), and that diabetes only develops when a defect in insulin secretion occurs as well ('beta cell exhaustion'). Thus, resistance to the action of insulin to lower blood glucose results in over-stimulation of the pancreatic beta-cells, and sets the scene for their eventual failure in individuals susceptible to Type 2 diabetes.

12.1 Extent of the problem

Type 2 diabetes constitutes 85–90 per cent of all diabetes in developed countries and in those countries it is predominantly a disease of middle age or older people (WHO 1994). Type 2 diabetes is more prevalent among populations that have migrated from rural to urban areas. Increasing industrialisation and westernisation and changes in diet and energy expenditure are associated with increases in the prevalence of Type 2 diabetes (see Subsection 1.2) (McCarty & Zimmet 1994; McCarty et al 1996). Risk may be increased by stresses occurring across the lifespan: under-nutrition in early life; and over-nutrition, physical inactivity, and obesity in early adult life. In populations in transition, which include Australian Aboriginals, Type 2 diabetes occurs at much earlier ages as well as being much more prevalent (O'Dea 1992).
Type 2 diabetes is a major cause of morbidity and mortality among Indigenous Australians. Epidemiological investigations suggest that the overall prevalence of diabetes among Indigenous adults is between 10 per cent and 30 per cent, at least two to four times that of the non-Indigenous population (de Courten et al. 1998). Studies of diabetes in a central Australian Aboriginal community with a long period of acculturation showed a prevalence 15 times higher (20 to 50 year age group) (O'Dea et al. 1993) than in the Australian population (Glatthaar et al. 1985; Guest & O'Dea 1992). Pooled data from south-eastern and central Australian Indigenous people, aged 20 to 49 years, also show a 12 per cent prevalence of Type 2 diabetes, compared with 1 per cent in a Victorian country town sample of non-Indigenous people in the same age range (Guest & O'Dea 1992).

Onset of Type 2 diabetes occurs at a far earlier age than in the general population (O'Dea et al. 1993). A recent study of Indigenous children and adolescents has documented a high prevalence of Type 2 diabetes (2.7 per cent), and of risk factors for Type 2 diabetes (Braun et al. 1996).

In the 1994 NATSIS, more than 4 per cent of Indigenous persons reported diabetes as a long-term illness (ABS 1996b). The rate is one-third higher among Indigenous females than males. After the age of 45 years, one in five people reported that they had the disease (ABS & AIHW 1997). The survey also indicated that diabetes is more prevalent among Indigenous people living in rural areas. An oversample of Indigenous individuals during the 1995 NHS has also provided similar estimates (ABS & AIHW 1999). However, results from both the NATSIS and the NHS are based on self-reported data, and therefore may underestimate true levels of diabetes.

An increasing trend in diabetes mortality has been noted among Indigenous Australians. Between 1985 and 1994, diabetes mortality rose sharply, at an annual rate of 9.6 per cent among males and 5.4 per cent among females (Anderson et al. 1996). Diabetes contributed to 3–5 per cent of excess deaths in Indigenous people (aged 15 to 65 years) compared to the general population in the Northern Territory (Cunningham & Condon 1996) and there was a very substantial increase in age-standardised death rates related to diabetes in Aboriginals in Western Australia from 1985–1989 to 1990–1994 (Health Department of Western Australia 1997). It should be noted that much of the apparent rise in diabetes-related mortality may be secondary to increased awareness of diabetes among medical practitioners. Also, mortality data underestimate the extent of diabetes-related deaths in a population as these are based on underlying cause of death. Since 1997, deaths have been coded for the underlying cause and the associated causes (ABS 1999c) but this breakdown has not yet been published for Aboriginals and Torres Strait Islanders.
12.2 Risks and associated factors

Prospective studies in adults show that impaired glucose tolerance, hyperinsulinaemia, obesity, abdominal fat distribution and a family history of diabetes are risk factors for Type 2 diabetes (Haffner et al 1990; Stern et al 1993). Other factors such as diet, level of physical activity and westernisation are also likely to contribute.

Obesity

The risk of developing Type 2 diabetes rises continuously with increasing obesity, and is approximately 5 to 10 times greater in those classified as obese (body mass index 30 and over) than in those with an acceptable weight (body mass index under 25) (Perry et al 1995; Shaten et al 1993). Early onset obesity is therefore of particular concern.

Obesity, particularly central obesity, may be influenced by total dietary energy intake (Guest & O’D ea 1992) and diet therefore plays a crucial role in the development of Type 2 diabetes. Research into the long-term effects of diet is hindered by difficulties in accurately measuring dietary intake (see Appendix 1). High saturated fat intake is considered to be an important dietary determinant of Type 2 diabetes (Marshall et al 1994), although this has not been confirmed by other studies (de Courten et al 1997).

Physical inactivity

Several studies indicate that physical activity plays a protective role against the development of diabetes (Helmrich et al 1991; M anson et al 1991; 1992; Perry et al 1995). After other risk factors have been accounted for, people who undertake regular physical activity have a 30–60 per cent lower risk of developing diabetes than
those who do not. The effect appears to be somewhat weaker in females, and in those who are not overweight (i.e., it is most beneficial for those most at risk of Type 2 diabetes). The beneficial impact of physical activity on diabetes risk is probably mediated by effects on both energy expenditure and insulin sensitivity.

**Low birthweight**

As discussed in Subsection 1.2, studies linking low birthweight with disease later in life suggest an increased lifetime risk for Type 2 diabetes (Hales & Barker 1992). The association is independent of gestational age, gender, adult body mass index, waist-to-hip ratio, and social class at birth and in adulthood (Rewers & Hamman 1995). The risk also extends to impaired glucose tolerance and insulin resistance (Phillips et al 1994).

**Impaired glucose tolerance**

Impaired glucose tolerance is considered a strong risk factor for diabetes. Approximately 33 per cent of people with impaired glucose tolerance will develop Type 2 diabetes over the next five to ten years (WHO 1994).

**Gestational diabetes (see also page 158 to 161)**

The hormonal changes of pregnancy increase insulin resistance. Women at risk of diabetes may develop impaired glucose tolerance (gestational diabetes) during pregnancy. Gestational diabetes is associated with increased risk of diabetes in women in the future. Women who have been diagnosed with gestational diabetes should be regularly screened for diabetes and actively counselled about their weight, diet, and levels of physical activity.

**12.3 Health consequences**

Phillips et al (1995) reported on the first longitudinal study of a cohort of Aboriginal people (Central Australia) with diabetes which examined direct causes of death from this disease. The single most common cause of death was renal disease, although the proportion of deaths directly attributed to diabetic nephropathy is not known. Other main causes of deaths attributed to diabetes included infections (septicaemia) (21 per cent) and coronary heart disease (14 per cent) (Table 12.1).
Table 12.1  Immediate causes of death in central Australian Aboriginal adults with diabetes, 1984-1991

<table>
<thead>
<tr>
<th>Category</th>
<th>Men</th>
<th>Median age (yrs)</th>
<th>Women</th>
<th>Median age (yrs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infection</td>
<td>15</td>
<td>53</td>
<td>12</td>
<td>58</td>
</tr>
<tr>
<td>Renal disease</td>
<td>9</td>
<td>55</td>
<td>20</td>
<td>60</td>
</tr>
<tr>
<td>Coronary heart disease</td>
<td>9</td>
<td>61</td>
<td>9</td>
<td>52</td>
</tr>
<tr>
<td>Other heart disease</td>
<td>7</td>
<td>65</td>
<td>7</td>
<td>57</td>
</tr>
<tr>
<td>Cerebrovascular disease</td>
<td>7</td>
<td>75</td>
<td>7</td>
<td>53</td>
</tr>
<tr>
<td>Neoplasm</td>
<td>6</td>
<td>61</td>
<td>7</td>
<td>64</td>
</tr>
<tr>
<td>Other</td>
<td>7</td>
<td>60</td>
<td>8</td>
<td>57</td>
</tr>
</tbody>
</table>


**Cardiovascular disease**

There is strong evidence that diabetes is an independent risk factor for cardiovascular disease particularly in combination with other risk factors such as high blood pressure and cigarette smoking (Knuiman et al 1986; Masarei et al 1986; McCann et al 1994). Diabetes can accelerate the rate of progression of coronary heart disease, particularly in those who have high blood pressure and are treated with insulin (Krolewski et al 1991). Diabetes magnifies the effect of conventional risk factors like hyperlipidaemia, high blood pressure and cigarette smoking (McCann et al 1994). Proteinuria may reflect this added risk by being a marker of vascular disease as well as of diabetic nephropathy (Deckert et al 1989).

**Nephropathy**

Diabetic nephropathy (renal disease) is the main cause of end-stage renal disease in western societies and is a dominant reason for various types of renal replacement therapy, including dialysis and transplantation (Brady & Brenner 1998). Microalbuminuria is the first manifestation of injury to the glomerular filtration barrier and predicts the development of overt nephropathy. Dipstick-positive proteinuria typically develops five to ten years after the onset of microalbuminuria, that is, 10 to 15 years after the onset of diabetes. By then, high blood pressure and progressive loss of renal function are well established (Brady & Brenner 1998).

Diabetes was about three times more likely to be reported as a comorbidity in Aboriginal patients registering for dialysis than in their non-Indigenous counterparts (Disney et al 1997). In Central Australia, it is estimated that renal disease is the immediate cause of death in 22 per cent of Indigenous people with diabetes (Phillips et al 1995; Hoy et al 1996a).
Neuropathy

Diabetic neuropathy (damage to the nerves) can affect every part of the nervous system except, perhaps, the brain. It rarely causes death but it is a major cause of morbidity particularly because of peripheral neuropathy, diabetic foot ulcers and gangrenous damage which can be complicated by peripheral vascular disease (Foster 1998).

Retinopathy

Diabetic retinopathy is a major cause of blindness (Foster 1998). Its causation and pathology can be complex and its effective management requires specialist opinion and treatment, coordinated with all other health professionals involved in the patient's care.

Early development of risk factors

Risk factors for Type 2 diabetes may develop rapidly and without clinical warnings in Aboriginal people. A 1979 study showed that changes in diet and lifestyle (i.e., changing from growing fruit, vegetables and livestock to dependency on store foods) can lead to substantial increases in body mass index, increased prevalence of diabetes, a fall in the age of onset and increased prevalence of hyperlipidaemias in a relatively short period of time (11 years) (O’Dea et al 1982; Gracey 1995).

Another study has shown that hyperinsulinaemia and impaired glucose tolerance may be present for many years before diabetes becomes detectable by ‘routine’ screening procedures or as a result of clinical symptoms (White et al 1990). Follow-up after five years showed significant progression of risk factors for diabetes. The percentage of subjects who were overweight had increased substantially. At a mean age of 18.5 years 8.1 per cent of the group had impaired glucose tolerance, 2.7 per cent had diabetes, and almost 22 per cent had high plasma cholesterol concentrations (Braun et al 1996).

These observations indicate that risk factors for diabetes can be present in a significant proportion of apparently symptomless Aboriginal children and adolescents and that this situation can deteriorate quickly within the space of a few years. Obviously, greater attention is required for secondary prevention of diabetes including screening programs, earlier detection, and earlier and more effective management including diet and physical activity.

Gestational diabetes

About 4 to 6 per cent of women not previously known to have diabetes develop hyperglycaemia during pregnancy. This percentage is higher in Indigenous women and in other high-risk populations than in the total population (see below). In most cases of gestational diabetes, the hyperglycaemia resolves soon after the delivery of the baby. However, in some cases it may continue, leading to a diagnosis of diabetes.
Compared to those without diabetes, mothers with diabetes experience a significantly higher incidence of pregnancy-induced hypertension and preterm birth (Mello et al 1997), urinary tract infection, pre-eclampsia and uterine bleeding (McMahon et al 1998; Martinez-Frias et al 1998). Gestational diabetes also carries health risks for the infant, and even when the mother's blood glucose levels return to normal after the pregnancy, the mother remains at high risk of developing diabetes later in life (Sullivan et al 1998).

Children born to mothers with diabetes are also at a high risk of developing macrosomia (large body size), fetal malformations, fetal distress, and neonatal complications including hypoglycaemia, respiratory distress and jaundice (Kamath et al 1998; Martinez-Frias et al 1998; American Diabetes Association 1993). The infants of mothers with pre-gestational diabetes or gestational diabetes may also develop insulin resistance and impaired glucose tolerance early in life (Plagemann et al 1997).

Longitudinal studies in Pima Indians (an indigenous population in the United States) indicate that the offspring of mothers who were diabetic during pregnancy had a much higher risk of becoming obese as adolescents and developing Type 2 diabetes as young adults than the offspring of mothers who developed diabetes after that pregnancy (Pettitt et al 1987; 1993). This effect remained after adjustment for possible confounders such as the diabetic status of the father, and the mother's body mass index. Interestingly, these researchers have recently reported that the adverse impact of the diabetic pregnancy on the risk of premature obesity and Type 2 diabetes in the offspring is halved if the mothers breast-feed their infants for at least two months (Pettitt et al 1997).

There may also be increased perinatal and infant mortality in babies born of mothers with gestational diabetes (Blank et al 1995; Sullivan et al 1998).

Management guidelines for gestational diabetes have been published recently (Hoffman et al 1998).

Prevalence of gestational diabetes in the Indigenous population

The prevalence of gestational diabetes is reported on the perinatal collection forms in Queensland and the Northern Territory. It is substantially higher in Aboriginals than non-Aboriginals at all ages, although in the Northern Territory the rates in Aboriginals and Asians are similar (Markey et al 1996). In Queensland, the only location with a substantial Torres Strait Islander population, the gestational diabetes rate is higher at most ages in this group than in Aboriginals. Some of these differences may be due to different screening rates in pregnant women, but they are consistent with other data reporting differences between racial groups (Zimmet et al 1991).
### Table 12.2 Confinements with gestational diabetes by age and ethnicity, Northern Territory (1992–1995) and Queensland (1996–1998)

#### Northern Territory

<table>
<thead>
<tr>
<th>Age</th>
<th>Aboriginal (%)</th>
<th>Caucasian (%)</th>
<th>Asian (%)</th>
<th>(%)</th>
</tr>
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<td>25–29</td>
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<td>3.2</td>
<td>6.8</td>
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<td>30–34</td>
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<td>35</td>
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</tbody>
</table>

#### Queensland

<table>
<thead>
<tr>
<th>Age</th>
<th>Aboriginal (%)</th>
<th>Torres Strait Islanders (%)</th>
<th>Non-Indigenous (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;20</td>
<td>1.7</td>
<td>1.4</td>
<td>0.8</td>
</tr>
<tr>
<td>20–24</td>
<td>3.1</td>
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<td>1.5</td>
</tr>
<tr>
<td>25–29</td>
<td>5.6</td>
<td>7.8</td>
<td>2.3</td>
</tr>
<tr>
<td>30–34</td>
<td>9.1</td>
<td>13.3</td>
<td>3.3</td>
</tr>
<tr>
<td>35+</td>
<td>14.7</td>
<td>27.8</td>
<td>5.1</td>
</tr>
</tbody>
</table>

**Notes:** Northern Territory data for Aboriginals include a small number of Torres Strait Islanders. ‘Other’ and ‘not stated’ races have been excluded. Queensland data are preliminary and may be subject to change. People identifying as both Aboriginal and Torres Strait Islanders or with race not stated have been excluded.

**Sources:** Markey et al (1996); Perinatal Data Collection, Queensland Health, special tabulation 2000.

The higher prevalence rates suggest that the cost-benefit calculations of screening Aboriginals and Torres Strait Islanders for gestational diabetes may be greater than for lower risk groups. Screening for gestational diabetes is inconsistent across the country with adequate testing during pregnancy being undertaken in only about 50 per cent of the general population (Moses & Colagiuri 1997).
Gestational diabetes

What is gestational diabetes?

• Gestational diabetes occurs in some women during pregnancy. Sometimes the mother has diabetes before she is pregnant but does not discover this until her blood sugar levels are tested in pregnancy. As the baby grows more insulin is needed. Some woman are unable to make any more insulin so their blood sugar can no longer be controlled. Gestational diabetes can be dangerous so the mother is tested for glucose in her blood after a sugar drink (glucose tolerance test), usually at 24 to 28 weeks.

What effects does gestational diabetes have on the mother and baby?

• After the birth, the diabetes in some mothers goes away but over the next few years, many women with past gestational diabetes develop full diabetes.
• The higher sugar to the baby can lead to a bigger baby, which can lead to a more difficult and damaging delivery for the baby or mother. The higher sugar can also make the baby sick so that the baby needs special treatment after birth.
• Although the baby may develop diabetes later in life, gestational diabetes does not necessarily cause babies to have diabetes.

Treatment of gestational diabetes

It is established that careful treatment of gestational diabetes protects babies from damage. Treatment includes:

• having the right balance of food for the diabetes, the pregnancy, and the baby;
• undertaking regular physical activity (ie walking, swimming);
• self-testing of blood sugar and laboratory blood tests if the glucose level is too high;
• careful and close watching of the growth of the baby;
• self-administered insulin injections around the abdomen, if diet and physical activity are not enough to keep the blood sugar level right;
• delivering the baby at the right time and controlling the blood glucose level;
• checking the blood sugar of the baby after birth; and
• checking the mother by six weeks after the birth to see if the diabetes has gone away.

Dietary treatment of gestational diabetes

The principles of treating gestational diabetes by diet are the same as for treating Type 2 diabetes. The diet should include:

• low quantities of saturated fat;
• plenty of vegetables and fruit;
• lean meat and fish;
• wholegrain cereals;
• low-fat dairy products; and
• unsaturated fats (olive oil, other vegetable oils and spreads).

Frequently the goal is weight control, so bulky foods should be included and fats, sugars and processed foods limited.
12.4 What can be done about it?

Insulin resistance is affected by lifestyle. Nutritionally improved diets focused on reduced dietary intake of fat, refined carbohydrate and total energy, in conjunction with weight loss and regular physical activity have been shown to directly improve insulin action. The modification of diet and physical activity can help to correct abnormal metabolism associated with diabetes. Therefore, lifestyle modifications should be the base for intervention programs, with the targeting of young people in particular (O’Dea 1992; Rowley et al 1997).

Prevention

Prevention of excessive weight gain throughout life is probably the most important means of preventing Type 2 diabetes. This can be achieved through:

- regular physical activity incorporated into daily routines:
  - sport and outdoor activities for children; and
  - sport, walking, cycling, swimming for adults;
- diet rich in bulky foods:
  - fresh vegetables and fruit;
  - lean meat and fish; and
  - whole grain cereals, legumes;
- diet low in energy-dense foods:
  - refined carbohydrates; and
  - foods high in fats.

Interventions should be targeted at children and adolescents if the aim is primary prevention of obesity.

Management

It has been shown that the main metabolic abnormalities of Type 2 diabetes (postprandial glucose clearance, fasting insulin levels, insulin responses to a glucose load) could be corrected or greatly improved in a small group of diabetic Aboriginals who reverted to a more traditional hunting and food gathering lifestyle for several weeks (O’Dea 1984). The participants in this study also had reductions in major risk factors for cardiovascular disease including elevated blood pressure and dyslipidaemia. There were numerous factors associated with the temporary transition to traditional lifestyle which could have contributed to this improvement in health status: the diet (low fat, low energy, slowly digested carbohydrate, high bulk), regular physical activity, weight loss.
In a study of more than 500 subjects with impaired glucose tolerance in Da Qing, China, diet and/or physical activity interventions led to a significant reduction in the incidence of diabetes over a six-year period among those with impaired glucose tolerance (Pan et al 1997). Subjects were advised to increase their daily physical activity level by one to two units which was equivalent to 30 minutes to 1 hour of mild activity (slow walking), 20–40 minutes of moderate activity (brisk walking), 10–20 mins of strenuous activity (running), or 5–10 mins of very strenuous activity (vigorous sport).

These observations have important implications for the prevention, control and improved management of diabetes in Indigenous people. Increased and regular physical activity and weight reduction and/or control programs can be very helpful but they have to be acceptable, even attractive, and they must be locally relevant. Consumption of diets that are high in fibre, low in fat and with appropriate amounts of dietary energy, should be encouraged and carbohydrate content should contain less highly refined sources and greater proportions of slowly digested complex carbohydrate. Simple culturally appropriate messages are important, and the positive model of traditional lifestyle should be highlighted: lean meat, and plenty of fresh vegetables and fruit, with little processed food; and regular physical activity as part of daily routines. It is also important to appreciate that dietary and lifestyle modification should be made to complement holistic approaches to the control and minimisation of other serious and endemic related disorders in the Aboriginal and Torres Strait Islander population. These include overweight/obesity, high blood pressure, cardiovascular disease and renal disease.

**Education**

Diabetes education for Aboriginal people generally focuses on community awareness, a healthy lifestyle approach and personal management (London & Guthridge 1998). In an exploration of contemporary beliefs about diabetes within a remote community, diabetes and its treatment were found to be viewed within a broad holistic context with health maintained by hunting and gathering traditional foods and stable relationships with family and community members. ‘Diet’ and ‘exercise’ were seen as western health methods. Education programs need to be culturally relevant to the particular community and its setting and may need to be modified for individual communities and different regions around Australia.

Education programs involving Aboriginal health workers have focused upon determining appropriate methods that take into account Aboriginal teaching styles and Aboriginal culture and traditional sensitivities. Programs have been developed to help Aboriginal health workers in the management and treatment of Type 2 diabetes and to provide health education to address the needs of individual Aboriginal communities. Strategies include the role of a healthy diet and physical activity in diabetes prevention and management. Another attempt to improve the standards of Aboriginal and Torres Strait Islander health care has resulted in a review of diabetes guidelines (Kimberley Aboriginal Medical Services Council 1998).
Strategies are being developed in Aboriginal communities which will focus more attention on community-based approaches to help prevent diabetes. These include altering attitudes to food and physical activity, community development and more active involvement in decision making in issues that affect health (e.g., changes in food supplies to Aboriginal communities and in management of community stores) (Scrimgeour et al. 1994; George 1996; Zakrevsky et al. 1996; Simmons et al. 1997). Some successful community-based diabetes programs are summarised below.

**Diabetes programs in Australia**

The Looma Healthy Lifestyle Program

Looma is an Aboriginal community about 100 km from Derby in Western Australia. The Looma Diabetes Program (later changed to Looma Healthy Lifestyle Program) was established in 1993 and is a community-based intervention program to raise awareness of diabetes and its complications, to prevent and reduce the severity of diabetic complications, and to prevent and treat obesity and diabetes in Aboriginal people (White et al. 1997).

The program began with a screening of the adult population for obesity, diabetes and cardiovascular risk factors. After the screening, a group of subjects who had Type 2 diabetes or impaired glucose tolerance, as well as some with normal glucose tolerance who were overweight or obese, began a program of informal education, physical activity and simple dietary modification. Among this high-risk cohort, those who commenced dietary and/or physical activity modification had significantly better outcomes in glucose and fasting triglyceride concentrations than those who did not undertake such strategies. However, sustained weight loss was not achieved over a two-year follow-up period.

Community-based initiatives to promote good nutrition have included hunting trips, health promotion through sports carnivals, educational tours of the community foodstore, food tastings and reducing the number of days the take-away food outlet operated. The intervention was supported by the Community Council, including assistance through the appointment of community members as Sports and Recreation Officer and Store Manager and the banning of smoking in public buildings. Since 1993, there has been an improvement in dietary quality of food sold through the store, including an increase in sales of fresh vegetables and fruit (Skinner et al. 1998). At the community level, this was associated with significantly decreased average plasma concentrations of cholesterol and homocysteine and improved antioxidant levels.

Although there were no significant changes in the prevalence of obesity or diabetes over four years of follow-up, apparent insulin sensitivity improved. Since weight gain remained a problem among younger people, the focus of the program shifted to include the community school. The school now has a healthy food policy for its canteen and a breakfast and lunch program. An evaluation of the changes to the foods supplied by the canteen has shown extremely positive results (Cincotta et al. 1998). Community control and ownership enabled embedding and sustainability of
this program in the community, in association with social environmental policy changes and long-term improvements in important risk factors for chronic disease.

Yambacoona Diabetes Program

An example of an urban program is the Yambacoona Diabetes Program which operates at Blacktown in the mid-west of the Sydney metropolitan area. The Blacktown Diabetes program was initiated in May 1993 by the Aboriginal Liaison Officer at Blacktown Hospital to raise awareness in the Aboriginal community about diabetes. Originally the program was called ‘The Sugar Babies’ program but as it took on a wider focus it was changed to ‘Yambacoona’ which means ‘plenty of good stuff’.

Regular meetings were held in Nurragingy Reserve where there were good walking tracks and BBQ sites. At the meetings people talked about health problems caused by diabetes, practical ways to make changes to diets and levels of physical activity, and how to check for diabetic complications. Initially, funding was obtained for food. When this ran out everyone took it in turns in making salads and dampers. The program was supported by the local Aboriginal Medical Service which also provided transport (Bear-Wingfield 1996).

Other examples of diabetes projects within Aboriginal communities include:

- the Melville Island Project, which uses an Aboriginal story to inform Aboriginal people about the link between diet, lifestyle and diabetes (Scimgeour 1993); and
- the Alice Springs Hospital Project, which includes an evaluation of the effectiveness of a hospital-based education program for Aboriginal people with diabetes. Nutrition awareness increased by the end of the sessions, although there were no behavioural changes (Kline et al 1989).
**Type 2 diabetes**

**What is Type 2 diabetes?**

- The body needs insulin to transport glucose (or blood sugar) across cell membranes. Once inside the cell, glucose can be used for energy. Insulin also increases the transport of amino acids into the cells and improves their use in the manufacture of proteins. It also stimulates fat deposition by inhibiting fat breakdown and stimulating fat synthesis and uptake into tissues.

- All people with Type 2 diabetes have a defect in insulin secretion, and the vast majority are also insulin resistant. Insulin resistance usually precedes defects in insulin secretion. Insulin resistance is secondary to obesity (especially in the abdominal region), excessive energy intake, and physical inactivity. This places an increased load on the pancreatic β-cells. Diabetes develops when the β-cells can no longer continue to secrete sufficient insulin to control blood glucose levels.

- People with Type 2 diabetes are more likely to develop coronary heart disease, stroke and disease of blood vessels in the legs and feet. There is also a greater risk of kidney disease, diabetic eye disease (retinopathy and cataracts), and damage to the nerves (mainly in the feet and legs).

**Prevention and early detection of diabetes**

- The main risk factors for Type 2 diabetes are obesity and lack of physical activity. This means that the chance of developing Type 2 diabetes can be lessened through sensible eating and regular physical activity.

- Diet should be rich in bulky foods (such as vegetables and fruit, lean meat, fish, whole cereals and legumes) and low in energy-dense foods (such as refined carbohydrates and fatty foods).

- Children should also be taught about the benefits of controlling their weight and being physically active.

**Management of diabetes**

- Type 2 diabetes can usually be controlled by diet (see above), physical activity and weight loss.

- Tablets (oral hypoglycaemic agents) are also prescribed to reduce severe hyperglycaemia. However, this approach should always be seen as an adjunct to, not a substitute for, diet, physical activity and weight control.

- Treatment with injectable insulin may be necessary during illness or other stress (including pregnancy).

- It is important that people with Type 2 diabetes are frequently checked for any of the complications discussed above.
Cardiovascular disease comprises all the diseases of the heart and blood vessels including coronary heart disease (ischaemic heart disease or heart attack), rheumatic heart disease, stroke (cerebrovascular disease), high blood pressure (which is itself a risk factor for coronary heart disease or stroke) heart failure and peripheral vascular disease (caused by damaged blood supply to the heart, brain and legs). The main underlying causes of cardiovascular disease are atherosclerosis (fatty deposits in artery walls) and thrombosis (blood clotting), which individually or together can obstruct blood vessels. It is most serious when it affects the blood supply to the heart, causing angina or heart attack (myocardial infarction), or to the brain, which can lead to stroke. Most cardiovascular deaths are caused by coronary heart disease or stroke (Adams et al 1994).

Cardiovascular disease was rare or unknown in Australian Aboriginals in pre-contact times when they were lean, active hunter-gatherers eating high quality diets. Blood pressures were low and did not rise with age (MacFarlane 1978; White 1985) (see page 24). Aboriginals living traditionally orientated lifestyles in north-east Arnhem Land in quite recent times had very low fasting glucose and cholesterol levels and low blood pressure, consistent with low risk of cardiovascular disease (O’Dea et al 1988). This has changed dramatically so that today these diseases are the number one cause of deaths among Indigenous people who are generally less active and whose diets are predominantly westernised (Reid & Trompf 1991).

13.1 Extent of the problem

Cardiovascular disease is the leading cause of death for both Indigenous and non-Indigenous Australians and it is estimated that coronary heart disease, high blood pressure and stroke cost the Australian community $474 million, $364 million, $270 million, respectively. The only other diet-related condition that costs the community more is dental caries (estimated at $478 million). The figures presented are the middle estimates of the total costs of health care, the net present value of lost earnings (due to premature mortality) and the direct and indirect costs of sick leave (Crowley et al 1992).

For 1995–1997, cardiovascular disease was the leading cause of death among Indigenous people (in Western Australia, South Australia and the Northern Territory), accounting for almost 28 per cent of deaths of Indigenous males and 23 per cent of deaths in Indigenous females (ABS & AIHW 1999). Indigenous death rates for cardiovascular disease were three to four times higher than for non-Indigenous persons, with coronary heart disease and cerebrovascular disease being among the largest contributors to mortality (ABS & AIHW 1999). In young and middle-aged Indigenous adults, recently reported death rates for cardiovascular disease have been 10 to 20 times higher than in the rest of the Australian population (Veroni et al 1994; Anderson et al 1996; National Heart Foundation 1996).
Coronary heart disease

Coronary heart disease is the major cardiovascular cause of death for Indigenous people, representing 55 per cent of deaths among males and 41 per cent among females in 1994-1996. Figure 13.1 shows death rates for coronary heart disease in the Aboriginal and Torres Strait Islander population and in the total population. Data for Indigenous death rates are only available for 1985 to 1996 and there are quality concerns regarding data before 1991. What the data do not show is the very high rates of premature death from heart disease among Indigenous people. Indigenous men and women are dying in their 20s, 30s and 40s, while in the non-Indigenous population most heart disease deaths are in those over 60 years.

Figure 13.1 Coronary heart disease death rates, 1991-1996

![Graph showing coronary heart disease death rates, 1991-1996.](image-url)
Stroke

Mortality from stroke in the Indigenous population is about double that in the non-Indigenous population, with males at greater risk than females.

13.2 Risks factors for cardiovascular disease

In addition to the conventional risk factors (dyslipidaemia, high blood pressure, cigarette smoking), many other factors could also be operating to increase the risk of cardiovascular disease among Indigenous Australians. These include central obesity (see Chapter 11), insulin resistance, diabetes (see Chapter 12), haemostatic factors such as fibrinogen, factor VII, plasminogen activator inhibitors, and new factors such as apolipoprotein E4 and homocysteine, which are known to increase the risk of developing clinical coronary heart disease. High homocysteine levels are one of the consequences of diets very low in folate, which in turn are the consequence of low intakes of fresh fruit and vegetables.

The status of cardiovascular risk factors in adult Aboriginals and Europeans of rural south-eastern Australia has been reviewed (Guest et al 1994) and compared with outcomes among urban populations of this area (Guest et al 1992a; Guest et al 1993a; Guest et al 1993b). The prevalence of at least one major risk factor for cardiovascular disease in people aged 25 to 64 years was higher in Aboriginals (males 94 per cent, females 89 per cent) than in non-Indigenous people (males 70 per cent, females 59 per cent) in rural and urban areas (Guest et al 1994).

Glucose intolerance is another risk factor that is more prevalent in Aboriginals than in non-Aboriginal people in south-eastern Australia (Guest et al 1992b).

As discussed in Subsection 1.2, the Barker hypothesis suggests that under-nutrition during fetal life and consequent low birthweight may also be a risk factor for cardiovascular disease in adults.

Dietary risk factors

Dietary risk factors include:

- saturated fat from meat and processed foods;
- lack of fresh fruit and vegetables;
- high salt intake (directly and from processed foods);
- excess energy intake (through a high proportion of energy-dense foods); and
- alcohol consumption.
Lipids

Two simple tests for determining plasma lipid levels can be used to identify those individuals with an atherogenic (cardiovascular disease-causing) lipid profile who are, therefore, at increased risk of cardiovascular disease:

- ratio of total cholesterol to high-density cholesterol (this ratio has been found to be a better predictor of coronary heart disease than low-density lipoprotein alone) (Castelli et al 1986; Stampfer et al 1991); and

- measurement of plasma triglyceride concentrations (Hokanson & Austin 1996; Stampfer et al 1996), which will allow differentiation as to whether the low density lipoproteins, high density lipoprotein cholesterol or triglyceride-rich particles are the major cause for concern (Castelli 1996).

Triglycerides

High levels of triglycerides may be an independent risk factor for coronary heart disease. Triglycerides are increased by:

- insulin resistance:
  - diabetes and impaired glucose tolerance;
  - central obesity;
  - excessive sucrose consumption (particularly if the background diet is high in saturated fat);
  - lack of physical activity;
- excessive alcohol consumption; and
- pregnancy — mild elevations are not a concern, and reverse after the birth of the infant.

Elevated triglycerides are reduced by:

- weight loss in the overweight;
- cessation of excessive alcohol consumption; and
- regular physical activity.

Vegetables and fruit

Increasing intakes of fresh vegetables and fruit has an impact on a number of pathways in cardiovascular disease (Zatonski et al 1998) (see page 176). The high levels of folate they provide help lower plasma homocysteine, and the antioxidants reduce atherogenesis. As most vegetables and fruit are high in potassium and low in sodium, they also have a positive effect on high blood pressure. Vegetables and fruit are also rich sources of dietary fibre and their high bulk and low energy density helps displace energy-dense foods and facilitates weight reduction.
Lifestyle risk factors

Cigarette smoking

Smoking is probably the most important modifiable risk factor for heart disease (US Surgeon General 1983). There is a clear relationship between smoking and coronary heart disease (Prescott et al 1998), stroke (Robbins et al 1994) and peripheral vascular disease (Krupski 1991). Passive smoking has also been associated with increased risk of heart disease (Kawachi et al 1997; Law & Hackshaw 1996; Lam & He 1997).

Smoking is generally much more common among Indigenous than non-Indigenous people. In a number of local or regional studies, adult smoking rates of 50 per cent to almost 90 per cent have been observed. The 1994 NATIS reported that an estimated 56 per cent of Indigenous males and 48 per cent of Indigenous females aged 15 years and over were current smokers (Cunningham 1994). This is in sharp contrast to the estimated prevalence in the Australian population as a whole, in which about 27 per cent of males and 20 per cent of females aged 18 years and over said they smoked in 1995 (ABS 1996a).

Smokers tend to have poorer quality diets compared to non-smokers, possibly due to the influence of smoking on taste. It has been suggested that this may encourage the addition of salt to food and make polyunsaturated fats less palatable (Fulton et al 1988; Oliver 1989; Guest et al 1992a). Smoking may exacerbate the tendency to central obesity in Aboriginals (Guest et al 1992a) which, in turn, is linked with other cardiovascular disease risk factors including diabetes and high blood pressure (Bjorntorp 1988b).

Stopping smoking reduces morbidity and mortality from cardiovascular disease within two years (Kawachi et al 1994). Smoking cessation is therefore one of the most important components of both primary and secondary preventive strategies for cardiovascular disease.
Physical inactivity

People who are physically inactive are almost twice as likely to die from coronary heart disease as those who are active (Berlin & Colditz 1990; Leon & Connett 1991). Physical inactivity also relates to other risk factors for cardiovascular disease, such as obesity, high blood pressure, total blood cholesterol and a poor cholesterol pattern (Blair 1997). There is evidence that an increase in physical activity over time is associated with a fall in levels of those risk factors (Young et al 1993). Evidence that physical activity plays a protective role against stroke is less clear, although such evidence is increasing (Stacco et al 1998; US Department of Health and Human Services 1996).

**Risk factors occurring as a consequence of poor diet and lifestyle**

Risk factors occurring as a consequence of poor diet and lifestyle include:

- high blood pressure;
- high blood cholesterol;
- overweight and obesity (particularly central obesity); and
- diabetes.

**High blood pressure**

High blood pressure is a major risk factor for coronary heart disease (Kannel et al 1969; 1971), stroke (Wolf et al 1986; Davis et al 1987) and renal failure (Frolich et al 1991). The risk of heart attack, stroke and renal dysfunction increases with even mildly elevated blood pressure (Paul 1971). The higher the blood pressure, the higher the risk — especially when other risk factors such as smoking, high serum cholesterol and diabetes are present (Paul 1971; Kannel 1976; NHMRC 1997d). High blood pressure can also lead to morbidity and mortality in its own right.

It is very important for individuals and groups to modify their lifestyles to reduce blood pressure and the consequent risks of ill-health and death. Lifestyle modification will provide benefits additional to the direct effects on blood pressure. Improvements to health can be made through weight control (lower saturated fat intake and higher fruit, vegetable and fish consumption, moderation of heavy alcohol intake, and reduction of cigarette smoking) and through increased physical activity. Prevention of high blood pressure is further discussed in Chapter 14.

**Obesity**

Weight gain is associated with increased risk of cardiovascular disease. In the Nurses Health Study of more than 120,000 women in the United States (Huang et al 1998), risk of high blood pressure increased by an estimated 12 per cent for each unit increase in current body mass index and by 5 per cent for each 1 kg of weight gain in the long term.
High blood pressure, insulin resistance, dyslipidaemia and increased risk of coronary heart disease are associated with central obesity (Kaplan 1994). Central obesity can occur when the body weight or body mass index is normal, particularly in smokers.

**Cholesterol**

In general, cholesterol concentrations among Aboriginals have been found to be low (O’Dea et al 1988; O’Dea et al 1982; O’Dea et al 1990). However, cholesterol levels tend to rise with increasing westernisation (Casley-Smith 1959; Bastian 1979). High serum cholesterol concentrations have been described in established communities that were relatively affluent and intensely exposed to non-Aboriginal values and diet for an extended period (Lee et al 1994a). This suggests that serum cholesterol levels may become an increasing problem in Aboriginal and Torres Strait Island communities.

**Diabetes**

As discussed in Chapter 12, the overall prevalence of diabetes among Indigenous adults is at least two to four times that of the non-Indigenous population (de Courten et al 1998). Because of its early age of onset, in the 20–50 year age group diabetes is more than ten times more prevalent among Indigenous than non-Indigenous Australians (O’Dea et al 1993). It is likely to be an underestimated contributor to the high rates of premature cardiovascular disease in Indigenous populations. In other populations, diabetes increases the risk of cardiovascular disease two- to five-fold (Zimmet & Alberti 1997).

**13.3 Health outcomes**

**Coronary heart disease**

In about 25 per cent of cases, the first clinical manifestation of coronary heart disease is fatal. This highlights the importance of effective prevention and early treatment. In 1996, coronary heart disease was the major cardiovascular cause of death, accounting for 23 per cent of all deaths (Health & AIHW 1999b).

**High blood pressure**

Patients with high blood pressure die prematurely. The most common cause of death is heart disease, with stroke and renal failure also frequent (Williams 1998).

Heart disease is due, ultimately, to decompensation because of the excessive load imposed on the left ventricle, which causes heart failure.

Ischaemic stroke is secondary to the increased atherosclerosis which occurs in people with high blood pressure. Haemorrhagic stroke is due to the combination of elevated arterial pressure and the development of cerebral vascular microaneurysms.

Arteriosclerotic lesions in the kidney in high blood pressure interfere with renal function. Proteinuria and microscopic haematuria occur and about 10 per cent of deaths in high blood pressure are due to renal failure.
Stroke

Ischaemic or haemorrhagic stroke (cerebrovascular disease) is probably an important cause of morbidity and death in Aboriginals. The ischaemic episodes are often associated with modifiable vascular risk factors or atrial fibrillation. Half of a series of haemorrhagic strokes in Aboriginals in Perth occurred in patients who abused alcohol (Crowley & Hankey 1995).

13.4 What can be done about it?

Primary prevention programs for cardiovascular disease need to promote healthy eating patterns and regular physical activity early in life, with a particular focus on children and adolescents. Programs that target overweight and obesity as well as diabetes (preferably before they develop), are instrumental in reducing risk factors (including high blood pressure) for cardiovascular disease.

Prevention and early detection of cardiovascular disease

Cardiovascular disease is often clinically manifested early in Aboriginals, even in their third and fourth decades of life (Gracey 1991). Risk factors for cardiovascular disease, including elevated plasma cholesterol (particularly elevated low-density lipoprotein cholesterol), dyslipidaemia, diabetes and overweight or central obesity can already be well established during adolescence and early adulthood. This has important implications for health among adult Aboriginals (Braun et al 1996). It is, therefore, important to encourage the early detection and prevention of cardiovascular disease (and other interrelated conditions, risk factors and comorbidities) through the use of early screening methods and appropriate interventions, including diet and increased regular physical activity (Gracey 1991).

Modification of risk factors

Physiological cardiovascular risk factors, such as elevated blood pressure, tend to ‘cluster’ with other adverse health behaviours such as physical inactivity, diets high in fat and low in fibre and greater alcohol consumption (Prattala et al 1994). These are all modifiable influences that should be targeted.

People with cardiovascular disease who have multiple risk factors and/or have already had cardiovascular events such as angina, heart attack or stroke, should be targeted for lifestyle changes to block the progression and recurrence of disease. However, if the goal is primary prevention, the target group for this cluster of conditions, which are so strongly lifestyle related, should be children and adolescents.

Cardiovascular health programs in Australia

The homelands movement

The ‘homelands movement’ (ie moving back to traditional homelands and away from centralised communities) has been proposed as a beneficial approach to Aboriginal health. Work done in Central Australia suggests that this is so in relation to cardiovascular risk factors including high blood pressure, diabetes and overweight/obesity. Homelands residents have also been shown to have overall reduced risks of
hospitalisation and death (McDermott et al 1998). Other dietary and ‘lifestyle’
guidelines should also help develop approaches for individuals and groups with or at
risk of developing cardiovascular disease.

Minjilang

The Minjilang project in the Northern Territory (see Chapter 6) aimed to improve
dietary intake and encourage physical activity. The project showed significant
improvements in biochemical indices, including a 12 per cent reduction in mean
serum cholesterol, a marked decrease in homocysteine, and increases in serum and
red blood cell folate, serum vitamin B₆ and plasma ascorbic acid concentration.
Decreases in mean systolic and diastolic blood pressures, normalisation of body mass
index and normalisation of haematological indices were also found (Lee et al 1995b).
Prevention of cardiovascular disease

Priorities for prevention of cardiovascular disease in the Indigenous population include nutrition programs that include improved access to good quality affordable fresh vegetables and fruit in remote communities (Leonard et al 1995; Lee et al 1994a).

Diets rich in fresh vegetables and fruit have the following attributes that impact on a number of pathways in cardiovascular disease:

- high in folate (lowers plasma homocysteine);
- high in antioxidants (reduces atherogenesis);
- high in other bioactive phytochemicals (ie phytoestrogens and polyphenols);
- high in potassium, low in sodium (reduces blood pressure);
- high bulk, low energy density (displaces energy-dense foods high in fat/refined carbohydrate, helps prevent weight gain and facilitates weight reduction);
- rich source of dietary fibre (diabetes, high lipids).

Other components of a nutrition program that are also important include:

- changes to the diet to include lean or trimmed meat (low in saturated fat) and fish (which is an excellent source of n-3 fats and helps reduce thrombosis and lower triglycerides);
- reducing sugar consumption (ie in cool drinks, tea);
- reducing the prevalence of cigarette smoking which reduces exposure to a major oxidative stress that damages the vascular endothelium);
- increasing physical activity which increases energy expenditure, contributes to weight control (high lipids, blood pressure, diabetes), improves insulin sensitivity (diabetes, blood pressure, cardiovascular disease), and reduces the risk of Type 2 diabetes and of high blood pressure;
- reducing the prevalence of harmful and hazardous consumption of alcohol which reduces the risk of high blood pressure; and
- improving maternal and child health — evidence is accumulating that undernutrition in early life (in utero and in infancy) can amplify the risk of chronic degenerative diseases, such as cardiovascular disease, high blood pressure, and Type 2 diabetes in later life (see Subsection 1.2).

Prevention of high blood pressure is discussed in Chapter 14.
Kidneys can be affected by a variety of factors including infection, toxic substances, high blood pressure and diabetes. The body can compensate for damage to the kidneys up to a certain point as there is plenty of capacity. When damage is extensive enough, however, the kidneys begin to fail. When about 95 per cent of kidney function has been lost, end-stage renal disease, which requires dialysis or transplantation for survival, results (ABS & AIHW 1999).

Renal (kidney) disease has become recognised as an important and increasingly prevalent and severe disease in Aboriginal Australians. Prevalence is now so high that it is difficult and very expensive to meet the needs for renal dialysis (and in some instances transplantation) in Aboriginal patients with end-stage renal disease. This is particularly so in remote areas where logistical and financial problems make the provision of highly specialised medical and laboratory staff and equipment, as well as the running of highly technical dialysis programs, extremely difficult. Despite these difficulties, dialysis is available in many areas. This disease has important nutritional dimensions particularly because of the nutritional complications of the disorder and the importance of excellent nutritional support for affected patients.

### 14.1 Extent of the problem

Renal disease is associated with high rates of morbidity and mortality in Australian Aboriginals, particularly in association with diabetes, cardiovascular disease and infections (Phillips et al 1995; Hoy et al 1997). It has been estimated that renal disease is the direct cause of death in 22 per cent of Indigenous people with diabetes in central Australia (Phillips et al 1995).

In 1996, there were 1,405 new dialysis patients registered in Australia, of whom 100 (7 per cent) were identified as Aboriginal or Torres Strait Islander (Disney et al 1997). The Northern Territory accounted for the largest number of new Indigenous dialysis patients in 1996 (40), followed by Queensland (27) and Western Australia (18). Over the last several years, these three jurisdictions have accounted for 75–85 per cent of all newly registered dialysis patients identified as Indigenous (ABS & AIHW 1999).

In 1994 the crude incidence of end-stage renal disease in Aboriginals, nationally, was about six to ten times that of non-Aboriginal peoples. This rate had doubled in the previous four years (Bennett et al 1995; Hoy et al 1996b).

Kidney failure patients identified as Aboriginal tend to be younger than other patients. In 1997, about 70 per cent of Aboriginal patients were less than 55 years of age, while only 52 per cent of other patients were in this age group (Disney et al 1997).
Even very remote Aboriginal people can have high rates of markers of renal disease. In a study of a small group of Aboriginals living in the Great Sandy Desert near the Western Australian-Northern Territory border, 39 per cent of subjects had proteinuria, 49 per cent had haematuria and 30 per cent had definite or possible urinary tract infections (Gracey et al 1996).

A cross-sectional survey of adults in an isolated Northern Territory community with very high death rates showed high rates of cigarette smoking and excessive drinking, of preventable infections and their complications, and of high blood pressure, insulin resistance, diabetes and renal disease (Hoy et al 1997). Many of the infections were more pronounced and the lifestyle diseases were almost entirely new when compared with a health screen done 20 years earlier.

Most morbidities were strongly associated with identifiable risk factors such as being overweight, cigarette smoking, excessive drinking, and skin sores and scabies. All of these are largely amenable to modification. Problems with food supplies and pricing, unhealthy food choices and diversion of money into cigarettes, alcohol and gambling were mentioned as important contributing factors (Hoy et al 1997).

14.2 Risks and associated factors

Risk factors for renal disease in Aboriginals include obesity, insulin resistance, diabetes, high blood pressure, skin infections and perhaps familial tendency (Hoy et al 1998b). Diabetes and high blood pressure can in turn be made worse by renal disease (ABS & AIHW 1999). As discussed in Subsection 1.2, it has been hypothesised that sub-optimal fetal and childhood growth may be linked to adult chronic disease, including renal disease, through poorly understood ‘programming’ mechanisms commencing in early life (Hoy et al 1998b).

Diabetes

Renal disease is a major complication of diabetes. It is first diagnosed by the detection of protein in the urine (albuminuria). On average, more than 40 per cent of people with diabetes have elevated levels of urinary albumin and the prevalence is higher in those with diabetes of longer duration (Health & AIHW 1999a; Nelson et al 1995). A South Australian survey of Australians with Type 2 diabetes found albuminuria in more than 34 per cent of the people and renal dysfunction among 23 per cent of males and 41 per cent of females (Phillips et al 1998). In the 1995 NHS, 7 per cent of people with diabetes reported some form of kidney disease. This proportion was more than four times that noted among those without diabetes (ABS 1997b).

Diabetes is the second most common reason for entering end-stage renal disease programs (Disney 1996).

High blood pressure

As discussed in Chapter 13, high blood pressure is a risk factor for renal failure and even mildly elevated blood pressure can increase the risk of renal dysfunction. Management of elevated blood pressure is therefore helpful in the prevention of renal...
disease. Lifestyle measures to control weight, improve diet and reduce alcohol consumption and cigarette smoking are important in the management of both high blood pressure and diabetes.

14.3 Health consequences
The high rates of diabetes and high blood pressure (discussed in Chapters 12 and 13) predispose Aboriginal people to renal disease. As well, they have increased rates of genitourinary infections and may suffer from epidemics of post-streptococcal glomerulonephritis (Gogna et al 1983; Van Buynder et al 1992). Proteinuria occurred in about 30 per cent of subjects aged 10 years or more in community-based studies in the Northern Territory. This was associated with obesity, high blood pressure and diabetes. Rates were substantially higher in females than in males and increased with age (Van Buynder et al 1993). The relative risks of having elevated albumin:creatinine ratios in Aboriginal men and women compared to non-Aboriginal men and women were approximately 5:1 and more than 2:1, respectively (Guest et al 1993b). Significant proteinuria (protein: creatinine ratio > 50 mg/mmol) gave evidence of renal disease in 30 per cent of relatives of patients and in control subjects (Van Buynder et al 1993). Those authors commented that the very high prevalence of renal disease in Aboriginal Australians resembles that in north American native populations undergoing rapid cultural change. Microalbuminuria, overt albuminuria, haematuria and renal insufficiency were found in significant proportions of Navajo Indians in New Mexico (Hoy et al 1996b).

The very high rates of albuminuria among diabetics in that population were considered to indicate a large reservoir of renal disease and to foreshadow even heavier burdens of end-stage renal disease and cardiovascular disease in the near future. Better detection and control of high blood pressure were identified as being important in slowing the progression of renal disease.

14.4 What can be done about it?
It appears that end-stage renal disease can be delayed through early identification of renal disease, proper nutrition, control of blood pressure, diabetes and infections and through treatment with drugs.

Control of blood pressure can be achieved through weight control and dietary changes. Additive effects on blood pressure occur when weight loss is combined with restriction of alcohol intake (Puddey et al 1992), increased fish consumption (Bao et al 1998) or moderate sodium restriction, and with increased physical activity (Cox et al 1996).

Moderate salt restriction may help in the management of high blood pressure and in assisting the effects of drug therapy including diuretics, beta-blockers and angiotensin-converting enzyme (ACE) inhibitors.
Potassium intake, which is to a large extent related to fruit and vegetable intake, tends to be inversely related with sodium intake. Controlled trials of potassium supplements demonstrate useful blood pressure reduction in people with high blood pressure (Geleinjse et al 1994) but it is generally recommended that potassium intake be increased by increasing fruit and vegetable consumption.

Regular fish or fish oil consumption, combined with weight reduction is helpful in high blood pressure (Bao et al 1998) and substituting fish for meat several times a week can help to reduce total and saturated fat intake with benefits to cardiovascular health. Dietary n-3 fatty acids, which occur mainly in marine animals, are potentially protective against atherosclerosis, thrombosis and high blood pressure. These effects include inhibition of platelet aggregation, reduction in very low density lipoprotein cholesterol synthesis and triglyceride levels and effects on vascular function, cardiac rhythm and anti-inflammatory activity.

Diets rich in vegetables and fruit and low in animal products with saturated fat largely replaced by monounsaturated fat (such as olive oil) have benefits in relation to blood pressure. Such diets are rich sources of fibre, complex carbohydrate, potassium, magnesium, folic acid and antioxidants.

As diabetic nephropathy is the main cause of end-stage renal disease (Brady & Brenner 1998), prevention and management of Type 2 diabetes are also important in the prevention of renal disease. As discussed in Chapter 12, lifestyle measures to prevent diabetes include:

• diet rich in bulky foods and low in energy-dense foods;
• weight control; and
• increased physical activity.

Treatment options for patients with end-stage renal disease are haemodialysis, peritoneal dialysis and in some instances kidney transplantation. Associated medical therapies (eg for diabetes or high blood pressure or immunosuppressive drugs) constitute a key part of management. Choices for treatment options depend on clinical indications, availability of skilled staff and facilities, local and personal circumstances, geography and distance, and the wishes of the patient and his or her family (Hoy et al 1995). Sensitive cultural factors are very important in making decisions about which management strategies to use (Bennett et al 1995).

Risk factors for renal disease which have already been mentioned, including lifestyle changes, socio-economic disadvantage and high rates of infections, need much more concentrated attention in order to reduce the heavy burden of end-stage renal disease and the deaths that this causes. Better living conditions and education, more effective and integrated primary health-care programs, and comprehensive and regular screening for pre-clinical, early renal disease are needed as a matter of urgency. Meanwhile, those already affected by renal disease should benefit from the highest available standards of medical, nursing and supportive care (Spencer et al 1998).
Prevention of high blood pressure

Lifestyle changes are important for primary prevention of high blood pressure, as first-line treatment for mild blood pressure elevation and as a means of minimising the need for medical treatment of people with more severe or resistant high blood pressure (NHMRC 1997d). Appropriate lifestyle changes include:

• weight control (see Chapter 11);
• reducing saturated fats in the diet and increasing intake of polyunsaturated fats (Kaplan 1991; Iacono et al 1982);
• consuming fish or fish oils regularly;
• reducing dietary salt intake (Law et al 1991);
• increasing intake of vegetables and fruit;
• increasing physical activity (Blair 1997; Young et al 1993);
• restricting alcohol to no more than two standard drinks per day (Kaplan 1991); and
• reducing cigarette smoking.

Scabies

• Infections with group A streptococcus can lead to both renal failure and rheumatic heart disease. Aboriginals in northern Australia have among the highest rates in the world of both these conditions (Hoy et al 1998b; Carapetis & Currie 1998).
• In Aboriginals in northern Australia, these infections tend to occur in skin lesions following infestation with scabies. In many other parts of Australia, streptococcal infections tend to occur in the throat.
• Due to the association between scabies and streptococcal infections, the high prevalence of scabies is an important cause of both renal failure and rheumatic heart disease. Current point prevalence of scabies in many remote communities can be up to 50 per cent. The point prevalence of skin sores ranges from 10–70 per cent with over 80 per cent of sores attributable to group A streptococcus (Carapetis et al 1998).
• Treatment programs for scabies have been well documented but implementation has been problematic (Carapetis et al 1998). The relative importance of skin versus throat infections for renal failure in Aboriginals living in the southern parts of Australia is unknown.
Dental health may sometimes be overlooked among the many serious health problems experienced by Aboriginal people. Yet the consequences of poor oral health include pain, local or systemic infection, impaired speech and interference with eating, which may influence dietary patterns. The financial burden of dental treatment is also an important consideration (ABS & AIHW 1997). In 1989–90, dental caries as a diet-related disease cost the Australian community around $478 million, in direct health costs and indirect costs including such factors as worker absenteeism and lost earnings (Crowley et al 1992).

It is believed that Aboriginal people had substantially less dental caries than non-Indigenous people before European contact. A study in 1915 (Mattingly 1915) found a striking feature among Aboriginal children and adults was the absence of dental caries. Surveys of dental health conducted in the early 1950s in a remote Aboriginal settlement in the Northern Territory found that the Aboriginal population had substantially better dental health than did non-Aboriginal peoples. The number of decayed, missing and filled permanent teeth for juveniles, adolescents and adults were among the lowest in the world (Barret 1953).

The frequency of occurrence of dental diseases can be correlated with the extent to which Aboriginals have left their traditional way of life and food consumption and had these supplemented by Government rations of tea, flour and sugar (Campbell 1939) and, more lately, by westernised food and drink consumption patterns.

### 15.1 Extent of the problem

**Children**

Few recent studies have specifically compared the prevalence of dental caries among today's Indigenous and non-Indigenous children (Davies et al 1997). Recently, there appears to have been an improvement of oral health of non-Aboriginal children and deterioration in that of Aboriginal children. This tendency has important implications for dental health services.

The incidence of decayed, missing and filled teeth in Indigenous children is almost double that of non-Aboriginal children (eg at five years, Indigenous children are 33 per cent caries-free while non-Indigenous children are 64 per cent caries-free) (Peninska & Barnard 1997). Results of the 1994 Child Dental Health Survey in Australia showed data for Aboriginal people only from the Northern Territory. Aboriginal children were more likely to have decayed teeth while non-Aboriginal children were more likely than Aboriginal children to have their previously decayed teeth filled (Davies & Spencer 1997).
The caries experience among a weighted sample of Community Dental Service patients aged four to thirteen years in the period January to December 1992 was studied among 4,138 Aboriginal children, 9,674 non-Indigenous Australian-born children, and 957 overseas-born children resident in Darwin. The outcomes considered included the aggregate numbers of decayed, missing and filled deciduous and permanent teeth. Oral disease experience and prevalence of untreated oral disease were higher among Aboriginal and overseas-born children. There were significantly more decayed teeth and higher aggregate caries experience in the deciduous and permanent dentition of Aboriginal and overseas-born children than among non-Aboriginal Australian-born children, while overseas-born children also had more fillings and fissure sealants than the non-Aboriginal Australian-born children (Davies et al 1997). Aboriginal children appear not to have benefited to the same extent as non-Aboriginal children from health improvements in the Northern Territory or in the delivery of dental services.

An examination of the oral health of Aboriginal and non-Aboriginal school children in western New South Wales found that Aboriginal children (members of a transitional community with low socio-economic status) had a higher prevalence of caries and poorer oral hygiene than the non-Aboriginal children (Schamschula et al 1980a). The proportion of caries-free Aboriginal children was 29 per cent compared to 58 per cent of non-Aboriginal children (in the six to eight years age group).

**Adults**

There is limited information about the dental health of Indigenous adults. Edentulism (the loss of all one’s natural teeth) was found to be more common among people using the Commonwealth Dental Health Program (1994–1996) than for the general population. The program was aimed at providing dental care for low-income earners. Edentulism tends to be a result of appropriate treatment failure and the higher proportion of edentulism in this group indicates a high level of unmet dental care needs among low-income groups. Many Indigenous people were included in this group (AIHW Dental Statistics Research Unit 1995).

A survey of adolescent and adult Aboriginals in fringe settlements in western New South Wales found high mean caries prevalence among the younger Aboriginals and declining mean caries in the older age groups, which is typical of a community exposed to increasing caries risks over the last few decades (eg increase in consumption of sweetened foods) without the benefit of prevention measures (Schamschula et al 1980b). Oral hygiene was poor overall and deteriorated with age. Ninety two per cent of the subjects were affected with gingivitis and periodontitis. No evidence of effective oral health care was observed and there was a lack of awareness of oral health needs. In this group, 38 per cent required emergency treatment. Compared to Australian non-Aboriginal people of all ages, the Aboriginal population studied was at a grave disadvantage in terms of restorative treatment. While caries prevalence has been decreasing in New South Wales as a consequence of fluoridation (Barnard 1977), there was no evidence of this in the Aboriginal sample.
In a small study of access to dental care, Indigenous adults were more likely than non-Indigenous adults to report edentulism. Among patients using public-funded dental services, Indigenous patients were more likely than other patients to have tooth extractions (Brennan & Carter 1998).

15.2 Risks and associated factors

Diet is considered one of the main determinants of dental caries and linked with the causation, prevention and treatment of this condition. The role of dietary sugar as a causative agent for dental caries is well known. Bacteria metabolise simple sugars such as glucose and fructose, as well as lactose, sucrose and maltose, to produce acids that demineralise teeth (Gibbons & van Houte 1978). Studies have shown the importance of frequency of sugar consumption and the form in which it is consumed. Sugars in beverages are more likely to have a temporary influence on acid production. Foods that adhere to the teeth are more cariogenic than those that wash off quickly (Gustafsson et al 1954).

Sugar as a dietary factor, however, is not considered as important in the caries process as it was two decades ago. Factors that contribute most significantly to caries development are the frequency of carbohydrate intake, the number of meals and snacks per day, oral hygiene practices, fluoride intake, regularity of check-ups, intake of non-sugar sweeteners, ethnicity and socio-economic group (Harel-Raviv et al 1996; Kandelman 1997).

Extended contact of sugars with teeth can lead to ‘nursing-bottle caries’ in infants. Nursing-bottle caries is a unique pattern of dental caries in which severe dental caries occurs in the upper incisors in young children. This results when children are put to sleep with a bottle of milk, formula or juice, allowing the sugars in the drink to remain in contact with tooth surfaces for long periods of time (Gardner et al 1977; Ripa 1978; Kelly & Brueurd 1987) (Figure 15.1). In more severe cases the molars can also be involved.

**Figure 15.1 Nursing-bottle caries**

![Nursing bottle caries affecting the upper incisor teeth, with abscesses](image1)

![Severe nursing bottle caries affecting all teeth](image2)

Source: Reproduced with permission of Lismore Dental Health Clinic.
15.3 Health consequences

The consequences of poor oral health may include pain, infection, impaired speech, interference with proper eating function which may necessitate a change in diet, embarrassment about one's appearance and financial burden incurred when dental intervention is necessary (ABS & AIHW 1999). Chronic dental disease, edentulism and ill-fitting dentures are very important causes of poor dietary intakes, lack of dietary fibre, fresh fruit, vegetables and meat in older people. This can lead to malnutrition and nutrient deficiencies in older people.

Early childhood caries is a serious dental condition that occurs during the first three years of life and may lead to early malnutrition and delay in the eruption of primary teeth and possibly to an increase in later caries prevalence (Ismail 1998).

Poor dental health throughout childhood can result in tooth loss in adulthood. The loss of teeth is a pathological result of disease or trauma that affects the normal processes of the jaws, teeth, muscles and nerves of the mouth (US Surgeon General 1988). The loss of natural teeth due to dental caries significantly reduces the ability to chew foods (Kapur et al 1974; Rissin et al 1978; Kapur & Garret 1984). Tooth loss leads to unsatisfactory chewing of such foods as raw vegetables, meats and nuts, lowering chewing performance (Manly & Vinton 1951; Kapur & Garret 1984) and resulting in avoidance of hard-to-chew foods (Yurkstas & Emerson 1964). Calorie-adjusted nutrient intakes decrease with progressively impaired dentition status in adult men while intakes of fibre, vitamins and minerals also decrease with increasing masticatory function impairment (Krall et al 1998). Prevention of tooth loss may significantly improve the diets and nutritional well-being of older adults.

Some early studies reported that inadequate dental function as a result of toothlessness or inadequate dentures and prosthetic devices may contribute to nutritional deficiencies (Mann & Mann 1945; Greene et al 1947). However, dietary intake can be adequate if the food is selected carefully and prepared appropriately (Geissler & Bates 1984).

15.4 What can be done about it?

The Aboriginal and Torres Strait population is exposed to a high risk of oral diseases for several reasons: their scattered distribution throughout the Australian continent, fluoride deficiencies in water supplies, scarcity of dental services, financial constraints, high turnover of dental staff, lack of recognition of the need for dental care, previous poor experiences with dental care, diet high in sugar, and poor oral hygiene (AIHW Dental Statistics Research Unit 1995; Peninska & Barnard 1997).

There is a need for development of educational, nutritional and preventive programs targeting mothers and infants to prevent early childhood caries and the development of dental caries in later years (Ismail 1998). A comprehensive oral health program for adolescent and adult Aboriginals is also a high priority and should include free emergency and low-cost routine dental treatment, fluoride administration and health education with emphasis on hygiene, diet and nutrition. Because poor oral health among Aboriginals is largely due to environmental and cultural changes, the
program should be maintained until improved education and socio-economic standards are established for future generations (Schamschula et al 1980b).

Oral health promotion programs, particularly targeted to Indigenous people, are relatively new in Australia. An example which has proved beneficial is the ‘It's Easier than Pulling Teeth – Changing a Dental Service Program’ for young and Aboriginal people in Adelaide (South Australian Health Commission & South Australian Community Health Association 1992).

The ‘Teeth for Keeps’ project (1991–1992) was aimed at promoting oral health and preventing dental disease in the rural Moree Plains Shire, New South Wales, implementing and evaluating oral health promotion strategies in infants and primary school children. Aboriginal and non-Aboriginal health workers were involved in community and school-based programs. School-based program strategies included targeting school canteens to attempt to increase healthy food alternatives. The project has raised the awareness of dental disease as a health issue and not a fluoridation issue. Early data analysis of the project showed that the percentage of children requiring routine treatment dropped from 28 per cent to 20 per cent and children requiring no treatment rose from 59 per cent to 71 per cent. The success of the project resulted in funding for a further three years (from 1992) (Short & Patterson 1994).
Dental care

Dental disease is an important potential cause of infections in Aboriginal and Torres Strait Islander peoples.

General dietary advice

- **Reduce the consumption of sugar-containing foods.** Replace sweet foods with fruit or vegetables.
- **Reduce the amount of snacks, particularly sweet snacks, during the day.** Acid that attacks the tooth enamel is produced every time we eat, particularly after sugar-containing foods (this includes honey and fruit sugars), and it is important to reduce the amount of acid attacks on the teeth.
- **If consuming sweet foods, it is better to eat them all at the one time, or immediately after meals.**
- **Sweet foods that are also sticky (eg chewy lollies) are more damaging as they can stay in the grooves of the teeth and near the gums for longer periods of time.**
- **Brush your teeth carefully or rinse your mouth with water after eating sugar-containing foods (this will help to dilute the acids).**
- **Chewing sugar-free gum immediately after eating will stimulate the flow of saliva, to dilute acids.**
- **Avoid drinking too many fruit juices or sports drinks during the day.** These drinks are naturally high in acid and can actually wear away the enamel surface of teeth (‘erosion’).
- **Have regular check-ups with the dentist.**

Avoiding nursing bottle caries

- **Don’t start the child on sweetened drinks (eg fruit juice, sweetened milk or tea, cola drinks) in a bottle.**
- **Don’t give the child a bottle to take to bed.** If the child really wants a bottle they should be given one containing water.
- **If a child is dependent on sweetened drinks in a bottle, gradually dilute the drinks with water over several weeks until the child is drinking only water.**
- **Start cleaning the child’s teeth regularly as soon as they appear in the mouth — this can be done using a clean handkerchief for babies and introducing a small toothbrush as the child is able to cope with it.**
- **Babies should not be given baby bottles containing sweetened drinks (cordial, soft drinks, sweetened milk or tea) as this can cause tooth decay (dental caries) in young children. Tooth decay can also be caused by sweetening the teat of the bottle (ie with honey or jam). Leaving the bottle propped in the baby’s mouth can also lead to tooth decay.**
- **The use of dummies and the practice of sweetening them with jam or honey should be discouraged.**
Quantitative studies into dietary intake may be associated with practical and methodological problems. Methodological problems encountered in obtaining a valid assessment of food intake of people in a traditional environment include (Lee et al 1995a):

- intruding into traditional and customary cultural practices (this can only be done by workers who are well accepted by the community);
- not being able to constantly observe males and females as they are usually involved in separate foraging activities; and
- not being able to keep track of the foods that are eaten straight from plants or at ‘dinner camps’ after food collection and foraging expeditions.

A review of dietary intake methods compared the practicality, acceptability and face validity of five dietary intake methods — weighed dietary intake, 24-hour recall, diet history, food frequency and ‘store-turnover’ methods — in two remote Australian Aboriginal communities (Lee et al 1995a). The four conventional dietary survey methods were adapted for use in Aboriginal communities. Neither diet history or food frequency methods were found to obtain quantitative data (Lee et al 1995a). Based on potential sources of bias in different methods (ie reporting errors, coding errors, use of food tables, and sampling and response bias) the store-turnover method was found to have the least potential for bias.

Recall methods
The review (Lee et al 1995a) found that participants using recall methods found it difficult to describe ‘usual’ intake and to categorise frequency of consumption of foods. Recall methods had consistent additions of foods high in protein and vitamin C, possibly reflecting a conscious editing of recall designed to constitute the ‘right’ answer according to perceived nutritional desirability. There was poor agreement between 24-hour recall and weighed dietary intake methods. The 24-hour recall method over-estimated protein, fat and most vitamins and minerals and under-estimated complex carbohydrate intake. Poor compliance appeared to be related to concurrent traditional ceremonies and financial problems (Lee et al 1995a). Another dietary assessment study in remote communities in north-east Arnhem Land concluded that 24-hour recalls are unsuitable as a method of collecting dietary intake data because the availability of foods was so variable (Maggiore 1990).

The store-turnover method
The store-turnover method is based on the systematic analysis of community store food invoices over a three-month period, which usually covers several ordering periods. The method is similar to the Apparent Consumption Data collated by the ABS for the whole of Australia and can be compared to population-adjusted recommended dietary intakes (Lee et al 1994b; Lee et al 1995a). Store-turnover data
has been used to show contemporary dietary practices (Coles-Rutishauser 1985) and to assess the effectiveness of nutrition interventions (Lee et al 1995b).

The method is relevant to relatively small geographical areas, and is relatively non-intrusive, rapid, easy and inexpensive to implement. It does not rely on subjective assessment of diet or on memory and avoids possible problems with language, literacy, numeracy and cultural factors which arise in the process of measuring or recalling individual diet. As purchased foods account for more than 90 per cent of the nutrient intakes in discreet, isolated Aboriginal communities (Cutter 1978; Young 1984), store-turnover data has potential as a useful source of dietary information. The method has also been validated against biological indicators of nutritional status (Lee et al 1994b).

Limitations of the store-turnover method include (Lee et al 1995a):

• it does not account for wastage (e.g. flour ruined by rain, or fed to dogs);
• slow turnover of some stock not being recorded in the usually used three-month period;
• bushfoods are not recorded;
• it is difficult to determine the size of the population needed for per capita data, for example because of visitors, absences, community events;
• no adjustments are made for the proportion of children and adults and food distribution patterns;
• the appropriate nutrient composition values of foods available in the store are not recorded (e.g. vitamin content of fresh fruit and vegetables); and
• it does not assess the diet of individuals, the individual variability of day-to-day dietary intake within the community or the variation of dietary intake within the income cycle.

These limitations are relatively minor in effect. A more serious limitation of the method is that it appears to be too tedious and/or time consuming for widespread application in remote, centralised communities. More research may be needed to determine why the method is not applied more widely.

NATSI S

The recent NATSI S used a simplified fat and sugar index questionnaire and a 24-hour recall methodology. An independent test was undertaken which verified the validity of this approach. Sugar was assessed from the intakes of confectionery, soft drink cordial and the sugar added to drinks such as tea and coffee. Fat was estimated from the consumption of certain foods such as pies, chips, buttered bread or damper, and chocolate. As the range of foods on the questionnaire was limited, both the fat and sugar intakes were corrected to estimate total intake of these items using the calibration equations calculated from the pilot survey data (ABS 1995).
APPENDIX 2

SUMMARY OF STATE/TERRITORY NUTRITION POLICIES

In Queensland, Western Australia and the Northern Territory, nutrition policies specific to the Aboriginal and Torres Strait Islander populations have been implemented. In the other States/Territories, elements of the nutrition policy are specific to the Aboriginal and Torres Strait Islander populations.

Northern Territory Food and Nutrition Policy and Strategic Plan 1995-2000

A project team facilitated the development of the Policy (Territory Health Services 1995). The team consisted of six people, four of whom were employed full-time. Other groups that were formed to support the process were:

- Intersectoral Advisory Group;
- Nutritionist Advisory Group; and
- Aboriginal Advisory Groups.

Following the consultation, the project team prepared a draft Policy. Over 40 organisations and individuals commented on the draft Policy, which was endorsed by the Executive of Territory Health Services in 1996 and has recently undergone mid-term review.

Four priority areas were identified. For each priority area/goal, targets are set for the strategies, which need to be implemented to achieve the priorities within one and five years. The actions required to achieve the goals are listed with the lead organisation which will be responsible for implementation and other groups which will be involved.

The priority areas are as follows.

Food supply in remote Aboriginal communities

Goal: to improve the quantity, quality and affordability of the food supply in remote Aboriginal communities.

Suggested implementation action included:

- food production — promoting self-sufficiency and semi-commercial food production; facilitate discussions about power and water quality;
- foodstores — develop business management skills and nutrition training for foodstore managers and Aboriginal community leaders, develop food policies and food supply guidelines, investigate transport issues, suitable standards for store buildings and food handling and safety, investigate ways to make the ‘book-up’ system more equitable; and
• community — develop accredited nutrition training for Nutrition Workers, improve community leaders’ knowledge of the food system by communication with food producers and distributors.

Public and private sector food services
Goal: to encourage public and commercial food services to adopt nutrition policies consistent with Australian Dietary Guidelines and the core food recommendations.

• public sector food services — nutrition policies and food supply guidelines to be included in accreditation, provision of cooled drinking water foundations in all public and places and venues;
• hospitals — policies for cultural food preferences and dietary guidelines;
• take-away food services — accreditation;
• hotels, clubs and restaurants — ‘Real Meal’ catering improvement scheme; and
• sporting and recreational venues — encourage the provision of nutritious foods.

Food and nutrition education and training
Goal: to increase access to nutrition education in the Territory for consumers, educators and health professionals and for training the nutrition workforce.

• primary school teachers — nutrition education programs;
• managers and store workers — training;
• nutrition workers in Aboriginal communities — site training;
• adults and young people — community physical activity programs;
• promote information on nutritious choices for take-away foods;
• promote awareness of consumer rights about refunding food purchases especially in remote communities;
• develop a consumer charter for rural and remote stores;
• provide food and nutrition orientation and in-service education about priority nutrition issues for all health workers;
• incorporate literacy and numeracy development in nutrition education materials for people from non-English speaking backgrounds;
• develop a five-year nutrition research and training plan;
• establish research and training in nutrition through tertiary institutions education program on bushfoods for Aboriginal and non-Aboriginal people; and
• organise consultations with the National Food Authority about food safety, food labelling, nutritional content, fortification of food and health claims likely to have an impact on the population of the Northern Territory.
Food and Nutrition Information System

Goal: to develop a food and nutrition information system to monitor changes in the food supply and nutritional status of Territorians.

- determine methods to coordinate, collect and feed back information on the Northern Territory food system, particularly for remote areas;
- determine methods to coordinate, collect and feed back information on the Northern Territory population;
- adapt and use food ‘store turnover’ methods in remote communities, and in private and public sector food services;
- develop a standardised system to report food costs and availability using a Market Basket Survey;
- design and validate methods for assessing individual food and nutrient intake of Aboriginal and Torres Strait Islander people in national and local surveys;
- advocate for national nutrition surveys to include suitable sampling of Aboriginal and non-Aboriginal population groups; and
- develop research proposals for funding to develop bicultural approaches to assessment of nutritional status of children and adults.

Queensland Aboriginal and Torres Strait Islander Food and Nutrition Strategy

The Queensland Health strategy was compiled by the Nutrition Program and the Indigenous Primary Health Care Unit, from the University of Queensland.

Groups formed to support the development of the strategy included a management team, a nutrition consultative group and a community consultative group. The Management Team comprised representatives from the Nutrition Program and the Indigenous Primary Health Care Unit, from the University of Queensland and from the following Branches of Queensland Health: Health Advancement, Aboriginal and Torres Strait Islanders Health Policy and Epidemiology and Health Information. The Nutrition Consultative Group consisted of 16 members who were predominantly from Aboriginal and Torres Strait Islanders Health Programs, Tropical Public Health Units, and Community Health Services, Aboriginal and Torres Strait Islander Health Policy Branch and Health Advancements branches of Queensland Health. Members of the Community Consultative Group were from Aboriginal and Torres Strait Islander Community Health Services and Regional Health Authorities.

The main consultations were with Aboriginal and Torres Strait Islander health workers and nutritionist/dietitians. These health professionals suggested other Aboriginal and Torres Strait Islander organisations that should be consulted. A range of rural, remote and urban communities were visited, as it was recognised that food and nutrition issues would vary between these settings.
A background paper (Broomehead et al 1995) was prepared to assist in the development of appropriate strategies. A nutrition consultative committee met on several occasions to provide technical advice on drafts of the strategy and working paper.

The draft strategy was circulated for endorsement by representatives from each of the community controlled health services, Regional Health Authority Aboriginal and Torres Strait Islander Health Services, the State Tripartite Forum, Queensland Aboriginal and Islander Health Forum and the Aboriginal and Torres Strait Islander Commission. The draft strategy was also circulated to Queensland Health's Regional Directors and relevant branches within Central Office for comment. Circulation to other government departments with follow before final endorsement by Queensland Health.

The Strategy is introduced with a rationale for developing a nutrition strategy. The strategy supports the principles of the Queensland Aboriginal and Torres Strait Health Policy, the Torres Strait Health Strategy (1993) and National Aboriginal Health Strategy.

The five essential parts to any strategy developed to improve Aboriginal and Torres Strait Islander health are:

• an integrated approach to community development (infrastructure, employment, education);
• Aboriginal and Torres Strait Islander control of decision making;
• implementation of effective prevention and treatment health services for the major health problems;
• provision of resources for health services at least equal to the rest of the population, or additional to address the higher burden of illness; and
• progressive improvement in the skill levels of health workers providing services to Aboriginal and Torres Strait Islander communities.

The strategy encompasses all aspects of the food and nutrition system including food production, the processing and distribution of food, food purchasing, preparation and consumption. In developing the nutrition strategy is should be recognised that:

• there are differences between the food and nutrition systems in remote and urban settings;
• the cultural meaning of food needs to be considered;
• there is a need to design and evaluate programs on a case-by-case basis with clear objectives and population groups in mind; and
• the holistic approach to health care needs to be accepted. Health is not just the physical well-being of the individual, but the social, emotional and cultural well-being of the whole community.

The Strategy identifies key action areas. There is an over-arching goal and brief rationale for the goal, followed by specific objectives and strategies to achieve the objectives.
The key action areas and goals are:

Aboriginal and Torres Strait Islander empowerment

Goal: empower Aboriginal and Torres Strait Islander people to participate equitably in the food and nutrition systems.

The objectives were to increase the number of Aboriginal and Torres Strait Islander employed, provide nutrition training to CDEP, facilitate the participation of Aboriginal and Torres Strait Islander people in developing food and nutrition policies. The main strategies were to facilitate the process by providing support, resources, advice and training.

Infrastructure and a nutrition workforce

Goal: to maximise communication and cooperation among all groups active in nutrition-related areas.

The objectives addressed coordination and cooperation at the State, regional and local levels. Strategies included establishing working parties and providing appropriate support.

Goal: to ensure an adequate and effective professional workforce to support food and nutrition initiatives in Aboriginal and Torres Strait Islander communities.

The objectives addressed the career structure of Aboriginal and Torres Strait Islander nutrition workers and the development of accredited training; ensuring appropriate training on food and nutrition issues to generalist health workers; establishing a network of nutrition health workers and nutritionists; and encouraging the involvement of Aboriginal and Torres Strait Islander people in food and nutrition-related enterprises.

Food supply

Goal: to increase availability and access to a variety of affordable and nutritious foods in Queensland Aboriginal and Torres Strait Islander communities.

Objectives addressed the need to increase people's awareness within the food production, processing and distribution system of food and nutrition issues affecting Aboriginal and Torres Strait Islander people; promoting cooperation and coordination with relevant sectors; ensuring continuity of access to nutritious food by coordinating money and food supply; facilitating the development of nutrition policies for community stores; encouraging local production of foods.

Community nutrition programs

Goal: to further develop programs and resources to increase the consumption of safe, nutritious food by Aboriginal and Torres Strait Islander people.

Objectives covered the development of culturally appropriate nutrition education programs; ensuring access to health promoting resources and training in local resources development; raising the communities awareness of the value of traditional foods and support behaviour change nutrition programs; promoting breast-feeding.
Goal: to provide appropriate health services for those with diet-related disease and associated complications.

The objective was to maintain and strengthen the nutrition component of appropriate community health services.

Nutrition information systems
Goal: to establish effective food and nutrition information systems within and for Aboriginal and Torres Strait Islander communities.

The objective addressed the development of a system for the ongoing collection, analysis, interpretation, dissemination and use of nutrition-related data.

Aboriginal Food and Nutrition Policy for Western Australia
Community consultations were undertaken throughout Western Australia by Health Promotion Services, Health Department of Western Australia, regional dietitian/nutritionists and nutrition workers to determine community views on the major food and nutrition issues for Aboriginal people, who could address these issues and how.

The draft policy detailed the nutrition issues identified from the literature and raised in the consultations. These issues included access to nutritious foods; food services; traditional foods, promotion and education; environmental health; health services; and monitoring and surveillance. The draft was circulated for comment to managers in the Office of Aboriginal Health and health promotion services and regional dietitians and nutritionists.

The purpose of the second consultation was to seek endorsement of the draft Policy and to involve interested groups in the development of the Strategic Plan.

The Draft Policy was distributed to:

- Aboriginal health workers;
- Western Australian Aboriginal Community Controlled Health Organisations (Aboriginal Medical Services);
- Public Health Units of the Health Department of Western Australia;
- dietitians/nutritionists;
- government departments — including Education, Family and Children's Services, Commerce and Trade (Aboriginal Economic Development Organisation), Homeswest, Employment, Education, Training and Youth Affairs, Social Security;
- non-government organisations — such as National Heart Foundation, Diabetes Australia;
- academic institutions — including the Centre for Aboriginal Studies and the School of Public Health at Curtin University, School of Health Science at Edith Cowan University, The University of Western Australia, Pundermurra College (Port Headland), Kalgoorlie College, Kimberley Aboriginal Medical Services
Council School of Health Studies (Broome), Marr Mooditj Aboriginal Health Worker College (Perth); and

- other interested groups — such as Aboriginal Business Development, Fruit and Vegetable Association.

The Office of Aboriginal Health in conjunction with Health Promotion Services has developed a Food and Nutrition Policy (Office of Aboriginal Health 1998) to:

- raise Aboriginal people’s awareness of the importance of nutrition;
- identify specific Aboriginal concerns in this area; and
- provide a focal point towards which a broad range of organisations can work to improve Aboriginal nutritional health in Western Australia.

The goal of the policy is to improve the nutritional health of Aboriginal people in Western Australia. The following objectives, which address food supply, promotion and education, environmental health, health services and monitoring/surveillance were identified to:

- increase the communities’ access to affordable nutritious foods in remote and urban communities;
- increase the range of nutritious foods in many food outlets including community foodstores, hostels, workplaces, schools, long day care centres and institutional care, Home and Community Care Services, meals-on-wheels;
- increase the use of traditional bushfoods;
- raise the community’s awareness of the importance of food to the health of their community;
- increase nutrition knowledge and skills among individuals, health workers, community leaders, catering staff and store managers, the school community and other community groups;
- improve the environmental health of communities with particular attention to the supply, storage, preparation and distribution of food;
- ensure that culturally appropriate and effective community-based primary health-care services for Aboriginal people are available to reduce the prevalent diet-related diseases;
- ensure that culturally appropriate and effective hospital-based health services are available for Aboriginal people with diet-related diseases; and
- establish an information system for the development and evaluation of nutrition programs.

The policy presents the nutritional issues identified from the consultations and the process by which programs and services to address these issues will be developed. A Steering Committee has been formed to review the findings of the consultation, and direct the development of the Program Plan and seek funding for its implementation.
APPENDIX 3

NHMRC DIETARY GUIDELINES

Dietary guidelines for Australians
- Enjoy a wide variety of foods.
- Eat plenty of breads and cereals (preferably wholegrain), vegetables (including legumes) and fruits.
- Eat a diet low in fat and, in particular, low in saturated fat.
- Maintain a healthy body weight by balancing food intake and regular physical activity.
- If you drink alcohol, limit your intake.
- Eat only a moderate amount of sugars and foods containing added sugars. Choose low salt foods and use salt sparingly.
- Encourage and support breast-feeding.
- Eat foods containing calcium.
- Eat foods containing iron.

Dietary guidelines for children and adolescents
- Encourage and support breastfeeding.
- Children need appropriate food and physical activity for normal growth and development. Growth should be checked regularly.
- Enjoy a wide variety of nutritious foods.
- Eat plenty of breads and cereals, vegetables (including legumes) and fruits.
- Low fat diets are not suitable for young children. For older children, a diet low in fat and in particular, low in saturated fat, is appropriate.
- Encourage water as a drink. Alcohol is not recommended for children.
- Eat only a moderate amount of sugars and foods containing added sugars.
- Choose low-salt foods.
- Eat foods containing calcium.
- Eat foods containing iron.
**Dietary guidelines for older Australians**

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

SOURCES OF ADDITIONAL INFORMATION

Commonwealth publications


NHMRC publications


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Evaluation of health programs


Websites

• Commonwealth Department of Health and Aged Care (http://www.health.gov.au)

• Australian Bureau of Statistics (http://www.abs.gov.au)

• National Health and Medical Research Council (http://www.health.gov.au/hfs/nhmrc)

• National Aboriginal and Torres Strait Islander Health Clearinghouse (http://www.cowan.edu.au/clearinghouse/)

• Australian Institute of Health and Welfare (http://www.aihw.gov.au)
APPENDIX 5

MEMBERSHIP OF WORKING GROUPS AND EXPERT PANEL

Membership and terms of reference of the Working Party that developed the first two drafts — Report on Aboriginal and Torres Strait Islander Nutrition 1996 and Facing the Future: Action to Improve the Nutritional Health of Aboriginal and Torres Strait Islander People 1997:

Health Promotion
Phillip Mills, Torres Strait and Northern Peninsula Area District Health Services, Thursday Island (Chair)

Public Health Aboriginal and Torres Strait Islander Nutrition
Cheryl Rae, EHNSC, Territory Health Services, Darwin
Dympna Leonard, Tropical Public Health Unit, Cairns
Amanda Lee, Menzies School of Health Research/Public Health Services, Queensland Health

Nutritional Science
Colin Binns, EHNSC, Curtin University, Perth (Deputy Chair)

Paediatrics
John Erlich, Alice Springs Hospital, Alice Springs

Aboriginal Health Worker
Don Whaleboat, Tropical Public Health Unit, Townsville (resigned May 1997)

Community development
Trish Jones, Darwin
Barbara Flick, Apunipima Cape York Health Council, Cairns

Epidemiology
Dorothy Mackerras, Menzies School of Health Research, Darwin

Nominee of ATSIC
Darren Miller

Communicating members
Ian Ring, Queensland Health, Brisbane
Richard Heazelwood, Cairns Base Hospital Cairns, Queensland Health
Michael Gracey, Health Department of Western Australia, Perth

Observer
Aletia Twist, Tropical Public Health Unit, Cairns

Secretariat
Susan Jeffreson
Terms of reference

With reference to the *National Aboriginal Health Strategy*, the report and recommendations from the Royal Commission into Aboriginal Deaths in Custody, the report from the National Conference on Aboriginal Nutrition in Remote and Rural Communities (1991) and recommendations from Aboriginal Nutrition Networks meetings (1992, 1993, 1994 and 1995), provide advice on the key nutritional issues facing Aboriginal and Torres Strait Islander peoples and communities on the causes and effects on health and priorities for action through the following:

1. Make recommendations for culturally appropriate research, implementation and evaluation strategies to improve the nutritional status of Aboriginal and Torres Strait Islander peoples in urban, rural and remote settings by:
   - defining and understanding food, nutrition and health issues from Aboriginal and Torres Strait Islander perspectives;
   - reviewing current programs aimed at improving the nutritional health of Aboriginal and Torres Strait Islander peoples;
   - identifying current successful practices, and where appropriate, suggesting new models of primary health care approaches to improve the nutritional status of Aboriginal and Torres Strait Islander peoples in urban, rural and remote settings;
   - defining the optimal size, skills and composition of primary health care teams to improve the nutritional status of Aboriginal and Torres Strait Islander peoples;
   - developing culturally appropriate health indicators and standards of best practice for monitoring the growth and development of Aboriginal and Torres Strait Islander children;
   - making recommendations on methods for use in the nutritional surveillance of Aboriginal and Torres Strait Islander populations to improve program management and feedback to communities as well as enable comparability between studies.

2. Advise the Working Party on Workforce Issues of the Aboriginal and Torres Strait Islander Health Standing Committee on the education and training needs of Indigenous and non-Indigenous health and community workers in relation to nutrition by:
   - reviewing existing training and education activities in food and nutrition;
   - identifying issues which require further research or development;
   - identifying the requirements for model public health nutrition and primary health care training (nutrition) courses;
   - identifying curricula and accreditation requirements for indigenous and non-indigenous nutrition workers;
   - identifying the requirements for nutrition in-service training of indigenous and non-indigenous health workers;
• developing a model for monitoring and evaluating both established and proposed training and education programs which promote consistency and excellence in training standards at the local, State/Territory and national levels.

3. Identify strategies which are culturally appropriate to Aboriginal and Torres Strait Islander peoples for evaluating the effectiveness of the outcomes of the working party's deliberations.

4. Report to the Environmental Health and Nutrition Standing Committee by August 1996 for report to NHAC and submission to NHMRC by November 1996.

The contractor and team from the Office of Aboriginal Health, Health Department of Western Australia, who developed the draft *Nutrition-related Diseases in Aboriginal and Torres Strait Islander People: Background Information and a Guide for Health Workers:*

Michael Gracey  
Anne Marie Lilburne  
Karen Orlemann  
Glenys Paley  
Mundullulu (Joan) Koops

Membership of the Expert Panel who developed the final information paper:

Barbara Flick (Chair)  
Maari Ma Aboriginal Health Corporation, Broken Hill

Lee-anne Daley  
National Aboriginal Community Controlled Organisation (NACCHO), Canberra

Michael Gracey  
Health Department of Western Australia, Perth

Dorothy Mackerras  
Menzies School of Health Research, Darwin

Kerin O’Dea  
Centre for Population Health and Nutrition, Monash University, Melbourne

Joan Vickery  
Koorie Diabetes Services, Victoria

Technical writer, editor and desktopper

Jennifer Zangger  
Ampersand Editorial & Design, Canberra

Secretariat

Krystyna Szokalski

Evelyn Tulega
A decision was made in 1995 to produce a report on Aboriginal and Torres Strait Islander nutrition. This began as an initiative of the former Environmental Health and Nutrition Standing Committee in conjunction with the former Aboriginal and Torres Strait Islander Health Standing Committee of the NHMRC.

The Aboriginal and Torres Strait Islander Nutrition Working Party was established in late 1995 with broad-ranging terms of reference (detailed in Appendix 5). This Working Party met face to face three times, and had several teleconferences to progress the preparation of its report. Two rounds of public consultation were undertaken — the first from December to January 1996 and the second from December to February 1997.

Colin Binns, School of Public Health at Curtin University of Technology, collated input from the Working Party to produce the first draft document Report on Aboriginal and Torres Strait Islander Nutrition. This report was then released for a second stage of consultation. The outcome of this consultation resulted in the second draft Facing the Future: Action to Improve the Nutritional Health of Aboriginal and Torres Strait Islander People rewritten by a team led by Dorothy Mackerras (Menzies School of Health Research, Darwin) to address the majority of concerns raised in the second-stage public consultation.

Up to this stage the project had been managed by the National Health Promotion and Protection Branch of the Public Health Division of the Commonwealth Department of Health and Family Services. In September 1997 the project was returned to the Health Advisory Committee (HAC) of NHMRC to be finalised. At the December 1997 HAC meeting, a working group was appointed to evaluate the report and decide on a way forward on the project. HAC agreed that additional work was required given the vast amount of information that had been incorporated into the document from submissions received in the second stage of consultations.

After several months of deliberations it was agreed in March 1998 to take the work achieved so far and prepare a clear and easily readable summary of current information with strategies for workers in local communities to improve Aboriginal and Torres Strait Islander nutrition. It was further decided that it would be prudent to take into account further information which had become available since the project began.

In June 1998 Michael Gracey (Office of Aboriginal Health, Health Department of Western Australia) was awarded the contract to conduct a systematic review and critical evaluation of scientific literature and write guidelines relating to Aboriginal and Torres Strait Islander nutrition and health. Michael Gracey collaborated with Anne Marie Lilburne who was seconded from Curtin University, and was assisted by researchers Karen Orlemann and Glenys Paley. He was provided with the following
documents: the draft report *Facing the Future: Action to Improve the Nutritional Health of Aboriginal and Torres Strait Islander People;* copies of all submissions made to the Working Party in the previous consultations; and the unpublished Butlin Report, *Food and Nutrition Programs for Aboriginal and Torres Strait Islander Peoples.*

A review of the literature confirmed there was a lack of research aimed at identifying nutrition interventions that are effective in the Indigenous population and therefore insufficient evidence to produce a guidelines document on nutrition for Aboriginal and Torres Strait Islander peoples. It was agreed this document would be produced as an information paper without recommendations.

The revised draft document renamed *Background Information and a Guide on Nutrition-related Diseases in Aboriginal and Torres Strait Islander Peoples* produced by Michael Gracey and his team was forwarded to HAC members in April 1999.

The finalisation of this report occurred simultaneously with another major national initiative in Aboriginal and Torres Strait Islander nutrition. The Primary Prevention Unit, together with the Office of Aboriginal and Torres Strait Islander Health (OATSIH) was also undertaking the development of a National Aboriginal and Torres Strait Islander Nutrition Strategy. This Strategy was being developed as part of the national public health nutrition strategy for the ‘whole of population’ called *Eat Well Australia.*

A national Aboriginal and Torres Strait Islander Nutrition Working Party had been established to advise and direct the consultative process for development of the Strategy. As there was a common target audience for the NHMRC nutrition document and the proposed National Aboriginal and Torres Strait Islander Nutrition Strategy, the Secretariat met in June 1999 with the Working Party and representatives of OATSIH and the Primary Prevention Unit. Agreement was reached at this meeting to take a collaborative approach to finalising the NHMRC nutrition document.

Following the meeting, it was agreed to disseminate the NHMRC draft document, *Background Information and a Guide on Nutrition-related Diseases in Aboriginal and Torres Strait Islander Peoples* together with a report titled *Informing Strategic Directions for Food and Nutrition in Aboriginal and Torres Strait Islander Populations.* The latter paper had been prepared in 1997 by OATSIH to inform the future development of a National Aboriginal and Torres Strait Islander nutrition strategy.

A joint database for the dissemination of these documents was established and pro formas developed to facilitate feedback to the NHMRC draft report; and to inform the early stages of development of a National Aboriginal and Torres Strait Islander nutrition strategy. The two documents were released for consultation on 9 August 1999.

An Expert Panel was established to incorporate comments from the consultation process and to finalise the document. The Panel comprised members nominated by HAC as well as members nominated by the Advisory Committee for the National Aboriginal and Torres Strait Islander Nutrition Strategy and Action Plan.
(NATSIN SAP), which will be released later this year. Barbara Flick, HAC representative, agreed to act as Chair. NATSIN SAP representatives were Joan Vickery (Victorian Aboriginal Medical Service) and Lee-anne Daley (National Aboriginal Community Controlled Organisation [NACCHO]). HAC representatives were Kerin O'Dea, Dorothy Mackerras and Michael Gracey. Jennifer Zangger was engaged as Technical Editor to assist the Expert Panel with the preparation of the final report.

Twenty-five submissions were received. These submissions were carefully considered at a face-to-face meeting of the Expert Panel and Technical Editor in Sydney in November 1999. Further deliberations took place during three teleconferences and numerous e-mail and phone consultations. In its deliberations, the Expert Panel agreed that it would be useful to develop a simplified document with illustrations for Health Workers based on the findings of the main report. This hands-on practical resource for service providers was developed by Jennifer Zangger with feedback from the Expert Panel.

The revised information paper and summary Guide for Health Workers were considered by HAC in February 2000 at its final meeting for the 1997–1999 triennium. HAC accepted both documents which were forwarded to NHMRC later that month for endorsement.

Council endorsed the information paper on 29 February 2000 and recommended that the Guide for Health Workers be distributed to Aboriginal Medical Services for comment on whether it is user friendly and culturally appropriate. This consultation is currently underway and it is envisaged that this document will be published within a very short timeframe following consultation.

The Health Advisory Committee acknowledges the hard work that was put into finalising this process by the Expert Panel and many other experts in the field who gave up their time to provide relevant up-to-date information to ensure that this report was of the highest standard. The Expert Panel commended the work done by Jennifer Zangger in preparing the Guide for Health Workers and for ensuring that this Information Paper is accurate, concise and readable while maintaining the integrity of scientific rigour.
ABBREVIATIONS AND ACRONYMS

ABS    Australian Bureau of Statistics
AH & MRC Aboriginal Health and Medical Research Council (New South Wales)
AIHW   Australian Institute of Health and Welfare
ALPA   Arnhem Land Progress Association
ATSIC  Aboriginal and Torres Strait Islander Commission
CDEP   Community Development Employment Program
DALY   disability-adjusted life year
DHHC   Commonwealth Department of Health, Housing and Community Services
DHSH   Commonwealth Department of Health and Human Services
EHNSC  Environmental Health and Nutrition Standing Committee
Health  Commonwealth Department of Health and Aged Care
NACCHO National Aboriginal Community Controlled Organisation
NAIHO  National Aboriginal and Islander Health Organisation
NATSIN SAP National Aboriginal and Torres Strait Islander Nutrition Strategy and Action Plan
NATSIS National Aboriginal and Torres Strait Islander Survey
NCHS   National Center for Health Statistics (United States)
NHMRC  National Health and Medical Research Council
NHS    National Health Survey
NIDDM  non-insulin-dependent diabetes mellitus
NNS    National Nutrition Survey
OATSIH Office of Aboriginal and Torres Strait Islander Health
SIDS   sudden infant death syndrome
SW-SB-SC Strong Women – Strong Babies – Strong Culture program
UNICEF United Nations International Children's Emergency Fund

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GLOSSARY

Acculturation
The loss or weakening of traditional culture.

Adiposity
Obesity.

Aetiology
Cause.

Age-specific rate
The proportion of events in a specific age group. Usually expressed per 100,000.

Example: Number of deaths among residents age 25-34 in an area in a year
\[
\text{Age-specific death rate (age 25-34)} = \frac{\text{Average (or midyear) population} \times 100,000}{\text{age 25-34 in the area in that year}}
\]

Age-specific death rate (mortality)
See age-specific rate. Rates are usually given separately by sex.

Age-standardised rate
Age-specific rates related to a standard population. This eliminates the effect of different age distributions in different populations and so allows comparison of groups with differing age compositions. By using the same reference population, age-standardised death rates of the Aboriginal and Torres Strait Islander population can be compared to the non-Indigenous population.

Age-standardised death rate
See age-standardised rate

Age-standardised hospital separation ratio
Compares the number of hospital separations recorded as Indigenous to the number which would have been expected if all Australian age-, sex- and cause-specific hospital separation rates had applied to the Indigenous population.

Albuminuria
The presence of albumin in the urine detected by standard clinical tests.

Anaemia
Haemoglobin below standard cut-off points which are determined according to age and sex.

Angina
Temporary chest pain indicating impaired blood supply to the heart muscle, most often brought on by exertion and relieved by rest.
Antenatal
Before birth and during pregnancy.

Anthropometry
The technique of measurement of size, weight and proportions of the human body.

Atherogenic
Factors that promote the deposition of atherosclerotic plaques in the walls of arteries leading to atherosclerosis.

Atherosclerosis
Deposition of cholesterol in arterial walls with formation of blood clots and calcification.

Beta cells
Pancreatic cells that secrete insulin, which lowers blood sugar levels.

Bioavailability
The amount of a substance actually available for metabolic processes. For example, only part of the iron in the diet is available for absorption as the rest is bound to other compounds.

Birthweight
The first weight, measured to the nearest five grams, of the newborn. It is usually obtained within the first hour of birth.
• Low birthweight – a birthweight of less than 2,500 grams
• Very low birthweight – a birthweight of less than 1,500 grams
• Extremely low birthweight – a birthweight of less than 1,000 grams

Blood pressure
The pressure of the blood on the walls of the arteries. Diastolic blood pressure occurs when heart is relaxing. Systolic blood pressure occurs when the heart is contracting.

Body mass index
Body mass index, also known as Quetelet's index, is body weight in kilograms divided by the square of the height in metres. Categories used in this document are those recognised by the WHO Expert Committee on Physical Status.

Used to indicate overweight and obesity in individuals, body mass index is also increased by an increase in muscle mass in very fit individuals. A body mass index of 25 to 30 indicates overweight while a body mass index of >30 indicates obesity. Body mass index predicts illness and death from cardiovascular disease, cancers and other diseases. Risk associated with body mass index applies over the range of body mass index and weight gain leading to increase in body mass index even to levels below the standard cut-points, still implies increased risk.

Body mass index — children and adolescents
Because of growth and changing distribution of body fat during childhood and adolescence some people consider body mass index is not an ideal measure of
overweight and obesity at these ages. However, no satisfactory alternative measure has been agreed on. Usually body mass index between the 85th and 95th percentile for age and sex is considered overweight and body mass index greater than the 95th percentile is considered to be obesity.

Cardiovascular disease
Comprises all diseases of the heart and blood vessels including coronary heart disease, stroke, heart failure and peripheral vascular disease, which are caused by damaged blood supply to the heart, brain and legs. Also known as circulatory disease.

Catch-up growth
Rapid, compensatory growth during rehabilitation from prior nutritional deficits or illness.

Central obesity
Central (or abdominal) obesity is measured as the waist-to-hip ratio (WHR):

\[ \text{WHR} = \frac{\text{waist circumference (cm)}}{\text{hip circumference (cm)}} \]

The WHO recommends the following WHR cut-offs to define increased risk for cardiovascular disease:

- WHR > 0.9 in men aged 19 years and over;
- WHR > 0.8 in women aged 19 years and over.

Increased central obesity increases risk at any weight.

Cerebrovascular disease
Abnormal condition of the vascular system and blood supply of the brain.

Centiles (sometimes called percentiles)
Divisions of a population with observations ranked from lowest to highest into equal subgroups. For example, these may be quartiles (four groups) or based on percentages.

Cholesterol
Refers to blood cholesterol. Fatty substance produced by the liver and carried by the blood to supply the rest of the body. Increased total cholesterol is a risk factor for cardiovascular disease. **High density lipoprotein cholesterol** is the form in which cholesterol is removed from the tissues and returned to the liver. An increased level of this form of cholesterol is associated with lower risk for cardiovascular disease. **Low density lipoprotein cholesterol** is the form in which cholesterol is transported from the liver to the tissues. An increased level is a risk factor for cardiovascular disease.

Congenital anomalies
Present at or from birth with a developmental origin.

Coronary heart disease
Diseases such as heart attack and angina, caused by blockages in the coronary arteries that supply blood to the heart muscle.
Dental caries
Localised and progressive destruction of teeth by bacterial action.

Diabetic nephropathy
Disorders of the kidneys in diabetes often seen with long-standing and poorly controlled diabetes.

Diabetic retinopathy
A disorder of retinal blood vessels characterised by capillary haemorrhage and the formation of new vessels and connective tissue. The disorder occurs most frequently in patients with long-standing, poorly controlled diabetes.

Diabetes
A group of metabolic diseases characterised by hyperglycaemia resulting from defects in insulin secretion, insulin action or both.

Diabetes is clinically classified as:
• Insulin-dependent diabetes mellitus (IDDM) or Type 1
• Non-insulin-dependent diabetes mellitus (Type 2 diabetes) or Type 2
• Malnutrition-related diabetes
• Impaired glucose tolerance (IGT)
• Gestational diabetes

Direct costs
Cost of health-care services (hospital, medical, pharmaceutical, allied professional and nursing home).

Dyslipidaemia
Abnormal levels of blood lipids including cholesterol and triglycerides.

Excess deaths
Observed deaths minus expected deaths.

Expected deaths
Deaths expected in a population are based on the population's age structure related to mortality experience of a particular reference population.
The number of deaths observed is then divided by the number of deaths expected. If the 'standardised mortality ratio' (SMR) is greater than one, then there are more deaths than expected. If the ratio is less than one, there are fewer deaths expected. For instance, an SMR of 2.5 indicates that there were 2.5 times as many deaths as would have been expected.

Energy
The chemical energy in foods that is available to the body from metabolism of carbohydrates, protein, fat and alcohol after digestion and absorption.

Eosinophilia
The formation and accumulation of an abnormally large number of eosinophils in the blood.
Gestation
The period of time from the fertilisation of the ovum (egg) until birth.

Gestational age
The age of the fetus or a newborn, usually expressed in weeks dating from the first day of the mother’s last menstrual period.

Gestational diabetes
Any degree of glucose intolerance with onset or first recognition during pregnancy.

Gestational weight gain
Weight gain during pregnancy has four principal components: laying down of fat stores, growth of breast and uterine tissue, increased plasma volume and growth of the fetus, placenta and amniotic fluid.

Gingivitis
Inflammation of the gums.

Glucose intolerance
Blood glucose responses greater than normal values but below the level diagnostic of diabetes.

Growth chart
A standard ‘tool’ to plot the physical growth if infants and children from birth to 18 years of age.

Growth faltering
The negative departure of a child’s growth path. Failure to gain, or actual loss of weight; a weight gain less than a specified value over a given period.

Growth velocity
The rate of growth over a specified period, eg 5 cm/year.

Haemostatic factors
Blood components involved in the formation of blood clots.

Haematuria
Blood in the urine.

Health status
An individual or population’s overall level of health, taking into account various aspects such as life expectancy, amount of disability, rates of hospitalisation, levels of disease risk factors and mortality rates.

Height-for-age (H FA)
Indicates whether a child is short or tall relative to others of the same sex and age. Low H FA may be due to stunted growth or may be genetic.
Hospitalisation data
The rate of admission to hospital and the diseases responsible for those admissions. The rate is expressed in relation to the population of the group concerned. Hospitalisation rates may provide an indicator of health but do not measure disease prevalence.

Hospital separations
A hospital separation occurs when a patient is discharged, is transferred to another facility or dies. Separation is the formal process by which a hospital records the completion of treatment and/or care for a patient.

Hospitalisation statistics
These are recorded as figures, which represent episodes rather than persons. That is, an individual may have been admitted to hospital on more than one occasion during one year. In addition, each hospital admission represents a mixture of need, access and demand. For instance, low hospitalisation rates may represent lower levels of need such as a healthier population or existing needs, which are not being met due to poor access.

Hypercholesterolaemia
High blood cholesterol.

Hyperglycaemia
Elevated blood glucose.

Hyperinsulinaemia
Elevated blood insulin.

Hyperlipidaemia
Elevated levels of blood lipids.

Hypertension
High blood pressure.

Hypertensive disease
Occurs when high blood pressure is high over a long period, especially when this leads to damage of the heart, brain or kidneys.

Hypertriglyceridaemia
Elevated blood levels of triglycerides.

Impaired glycaemic control
Unstable control of blood glucose.

Impaired glucose tolerance
See glucose intolerance.
Incidence
a) The number of instances of illness commencing, or of persons falling ill, during a given period.
b) The number of times an event occurs.
c) The number of new cases of a disease or disorder in a particular period of time. Incidence is often expressed as a ratio, in which the number of cases is the numerator and the population at risk is the denominator.

There are two specific types of incidence measures, cumulative incidence (CI) and incidence rate (IR) or incidence density (ID). CI is the proportion of persons who become diseased during a specific period of time and is calculated as:

$$CI = \frac{\text{Number of new cases of a disease during a given period of time}}{\text{Total population at risk}}$$

IR or ID is considered to be a measure of the instantaneous rate of development of disease in a population and is defined as:

$$ID = \frac{\text{Number of new cases of a disease during a given period of time}}{\text{Total person-time at risk}}$$

Indirect costs
Earnings foregone through illness and premature death. Costs to other agencies, institutions or industries. For example, illness can have 'flow on' effects to employers, service industries and tourism.

Infant mortality
Deaths of children under one year. It has conventionally been accepted as an indicator of the general level of health and well-being in addition to the social development of a population. Infant mortality rate is expressed as the number of infant deaths per 1,000 live births per year.

Insulin
A hormone secreted by the pancreas and involved in metabolism of glucose.

Insulin-like growth factors
Hormones thought to have a central role in the regulation of fetal growth.

Insulin resistance
Impaired response to insulin which may lead to raised glucose levels manifest as impaired glucose tolerance or non-insulin-dependent diabetes mellitus.

Insulin resistance syndrome
Termed ‘Syndrome X’. A condition characterised by relative insensitivity to the hypoglycaemic action of insulin, increased circulating fatty acid concentrations, increased hepatic glucose production, dyslipidaemia and hypertension and central obesity.
Intra-uterine growth restriction
Birthweight below a given low percentile cut-off for gestational age. Also referred to as intra-uterine growth retardation.

Life expectancy
Life expectancy at birth represents the average number of years a newborn baby could expect to live if the mortality rates of today were to continue throughout the baby's life. Life expectancy can be calculated in many ways, yet the most common approach uses actual data on deaths and the population age structure.

Macrosomia
Birthweight more than 4,000 grams, often associated with enlargement of body organs.

Mean
The average value, which is equal to the sum of scores divided by the number of scores.

Meta-analysis
The process of using statistical methods to combine the results of different studies. Meta-analysis includes aspects of an overview and of pooling data.

Metabolic disorders/metabolic abnormalities
Disorders in which the body's metabolic processes are abnormal.

Morbidity
Illness and/or disability.

Mortality
Death.

Myocardial infarction
Cell tissue death of a portion of cardiac muscle caused by obstruction in a coronary artery from atherosclerosis. Also called 'heart attack'.

National Aboriginal and Torres Strait Islander Survey (NATSIS)
The NATSIS survey aimed to provide comprehensive national information on a range of topics relevant to Aboriginal and Torres Strait Islander peoples — family and culture; education and training; employment; income; health; law and justice; and housing. The Survey was conducted by the ABS in consultation with ATSIC. Survey estimates were based on information obtained from a sample of approximately 5,000 dwellings with 17,500 people, covering about 6.6 per cent of the Aboriginal and Torres Strait Islander population as reported in the 1991 Census of Population and Housing.

NCHS 50th percentile (or centile) reference values
The median value for a body dimension (eg weight, length/height) at a particular age for males or females.
Neonatal death
Death of an infant within 28 days of birth.

Obese
Body mass index greater than 30 for adults.

Overweight
Body mass index greater than 25 but less than or equal to 30 for adults.

Parity
Refers to the previous number of pregnancies of at least 20 weeks' gestation.

Pathogenesis
The mechanisms by which disease is caused.

Perinatal
Occurring in the period shortly (usually 28 days) before or after birth.

Perinatal death
A stillbirth or neonatal death.

Perinatal conditions
Conditions of pregnancy, fetal growth, labour, delivery and life of the newborn in the first month. Diseases and conditions originating during pregnancy and in the neonatal period (within 28 days after birth) are included in this period.

Perinatal mortality rate
The number of deaths per 1,000 total births (fetal deaths plus live births).

Periodontitis
Inflammation of the periodontium (between the root of the tooth and the bone of its socket).

Population pyramid
A graphic representation of the age and sex composition of the population. It is constructed by computing the percentage distribution of a population, simultaneously cross-classified by sex and age. A population pyramid is intended to provide a quick overall comprehension of age and sex structure in the population.

Prevalence
The number of instances of a given disease or other condition in a given population at a designated time.

The number of all new and old cases of a disease or occurrence of an event during a particular period of time. Prevalence is expressed as a ratio in which the number of events is the numerator and the population at risk is the denominator. Prevalence quantifies the proportion of individuals in a population who have a disease at a specific instant and provides an estimate of the probability (risk) that an individual will be ill at a particular time. The formula for calculating prevalence (P) is:

\[ P = \frac{\text{Number of existing cases of a disease at a given point in time}}{\text{Total population}} \]
Primipara
A woman who has given birth once.

Prophylaxis
Prevention.

Prospective study
A study designed in advance to determine the relationship between a condition and a characteristic shared by some members of a group. The selected population is healthy at the start of the study. The population is followed over a period of time, noting the rate at which a condition occurs.

Proteinuria
The presence in the urine of abnormally large quantities of protein, usually albumin. Persistent proteinuria is usually a sign of kidney disease or renal complications of another disease, such as heart failure or hypertension.

Remote
The Rural, Remote and Metropolitan Areas classification, developed by the Commonwealth Departments of Primary Industries and Energy and Human Services and Health defines ‘remote’ as remote centres (urban centres with a population of greater than or equal to 5,000) and other remote areas (urban centres with a population of less than 5,000).

Respiratory disorders
Disorders/disease of the upper and lower respiratory tracts.

Rheumatic heart disease
Damage to heart muscle and heart valves caused by rheumatic fever.

Risk factor
An attribute or exposure that is associated with an increased probability of a specified outcome, such as the occurrence of a disease. Risk factors are not necessarily causes of disease.

Rural
The Rural, Remote and Metropolitan Areas classification, developed by the Commonwealth Departments of Primary Industries and Energy and Human Services and Health defines ‘rural’ as large rural centres (urban centres with a population of 25,000–99,999), small rural centres (urban centres with a population of 10,000–24,999) and other rural areas (urban centres of less than 10,000 population).

Self-assessed health status
Refers to the overall health as reported by respondents 13 years and over. For persons less than 13 years, information was provided by the child’s parent or a responsible adult member of the household.
Standard deviation
A measure of dispersion or variation around the mean for values in a population. Most widely used measure of dispersion of a frequency distribution.

Standardised rate
Rate adjusted to allow for comparison between two populations in the presence of a confounding factor. Standardised rates are weighted averages of group-specific rates of the confounding variable using the distribution of a ‘standard’ population to provide the weighting factors.

Standardised Mortality Ratio (SMR)
The ratio of the observed number of cases to the expected number:

\[ SMR = \frac{\text{Observed deaths (O)}}{\text{Expected deaths (E)}} \times 100 \text{ per cent} \]

Stroke
Also called cerebrovascular disease or cerebrovascular accident. A condition of the blood vessels of the brain characterised by blockage or haemorrhage resulting in interference with the blood supply to the brain. Paralysis, weakness, sensory change, speech defect or death may occur.

Stunted
Individuals whose height-for-age is low as the result of the past process of linear growth restriction.

Syndrome X
Insulin resistance syndrome. Condition characterised by relative insensitivity to the hypoglycaemic action of insulin, increased circulatory fatty acid concentrations, increased hepatic glucose production, dyslipidaemia and hypertension and central obesity.

t-test
A statistical test used to find the difference between the means of two different sample groups.

Triglycerides
Blood fats distinct from cholesterol.

Under-nutrition
Wasting or stunting.

Waist-to-hip ratio (WHR)
See abdominal obesity

Wasting (WHZ)
Abnormally low body weight-for-height relative to reference values (in this case expressed as a Z-score).
Weight-for-Age (WFA)
Indicates whether a child is light or heavy compared with others of the same sex and age irrespective of height.

Low WFA
Weight less than -2 SD of the sex-specific reference data relative to age.

Weight-for-Height (WFH)
This indicates whether a child is thin/wasted or overweight compared with others of the same sex and height.

Low WFH
Weight less than -2 SD of the sex-specific reference data relative to height.

Z-score (standard deviation score)
The deviation of an individual’s value from the median value of a reference population, divided by the standard deviation of the reference population.


ABS (2000) *Housing and Infrastructure in Aboriginal and Torres Strait Islander Communities, Australia 1999.* ABS Cat. No. 4710.0, Australian Bureau of Statistics, Canberra.


AH & MRC (1997) National Aboriginal and Torres Strait Islander Health Data Protocols for the Routine Collection of Standardised Data on Aboriginal and Torres Strait Islanders. Aboriginal Health and Medical Research Council, Sydney.


Basedow H (1925) *The Australian Aboriginal*. Preece and Sons, Adelaide.


Bibliography 227


Butlin A, Cashel K, Lee A et al (unpublished) (1997) *Food and Nutrition Programs for Aboriginal and Torres Strait Islander Peoples.* Office for Aboriginal and Torres Strait Islander Health Services, Commonwealth Department of Health and Family Services, Canberra.


Cane S & Stanley O (1985) *Land Use and Resources in Desert Homelands*. Northern Australia Research Unit (NARU), Australian National University, Canberra.


Engeler T, McDonald MA, Miller ME et al (1998) Review of Current Interventions and Identification of Best Practice Currently used by Community Based Aboriginal and Torres Strait Islander Health Service Providers in Promoting and Supporting Breast-feeding and Appropriate Infant Nutrition. Office for Aboriginal and Torres Strait Islander health Services, Commonwealth Department of Health and Family Services, Canberra.


Fuary MM (1991) In so many Worlds: An Ethnography of Life and Identity on Yam Island, Torres Strait. James Cook University, Canberra.


Kimberley Aboriginal Medical Services Council (1998) *Recommendations for Clinical Care Guidelines on the Management of Non-insulin Dependent Diabetes in Aboriginal and Torres Strait Islander Populations.* Commonwealth Department of Health and Family Services & Office of Aboriginal and Torres Strait Islander Health, Canberra.


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Nietschmann, B & Fitzpatrick J (1981) Good dugong, bad dugong; Bad turtle, good turtle. Natural History 90(5).


Office of Aboriginal Health (1998) Aboriginal Food and Nutrition Policy for Western Australia. Health Department of Western Australia, Perth.


Olson J (1984) Serum levels of vitamin A and carotenoids as reflectors of nutritional status. Journal of the National Cancer Institute 73: 1439-44.


Select Committee on Territory Food Prices (1999) *Price, Quality and Choice: Striking a Fair Balance. Inquiry into Food Prices in the Northern Territory*. Legislative Assembly of the Northern Territory, Darwin.


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The National Health and Medical Research Council (NHMRC) is a statutory body within the portfolio of the Commonwealth Minister for Health and Aged Care, established by the National Health and Medical Research Council Act 1992. The NHMRC advises the Australian community and Commonwealth; State and Territory Governments on standards of individual and public health, and supports research to improve those standards.

The NHMRC advises the Commonwealth Government on the funding of medical and public health research and training in Australia and supports many of the medical advances made by Australians.

The NHMRC also develops guidelines and standards for the ethical conduct of health and medical research.

The Council comprises nominees of Commonwealth, State and Territory health authorities, professional and scientific colleges and associations, unions, universities, business, consumer groups, welfare organisations, conservation groups and the Aboriginal and Torres Strait Islander Commission.

The Council meets up to four times a year to consider and make decisions on reports prepared by committees and working parties following wide consultation on the issue under consideration.

A regular publishing program ensures that Council’s recommendations are widely available to governments, the community, scientific, industrial and educational groups.

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