‘Dying fast and young’:
Contemporary Aboriginal health

This chapter examines the state of Indigenous health in Australia across the life-span, with particular emphases on infant health and adult morbidities that contribute to excess mortality, especially the impact of Type 2 diabetes. Diabetes is directly responsible for a significant number of deaths and contributes substantially to deaths from CVD and renal disease. Complications of diabetes are in large measure responsible for the high prevalence of disability within the Indigenous population.

The health of Australia’s Indigenous population falls well below that of its non-Indigenous population. In early life this is manifested in much higher rates of low birthweight and patterns of disrupted child growth. Infant mortality rate (IMR) has improved over recent decades but remains much higher than in the general population. As adults, Indigenous people are affected by higher rates of degenerative disease than Australia’s non-Indigenous population. Although rates of infectious diseases have declined, there are many preventable deaths due to non-infectious causes occurring in younger and middle-aged adults. Despite improvements in Aboriginal health, the health gap between Indigenous and non-Indigenous people has widened, making Australia ‘an anomaly in the developed world’ (Zinn 1995, p. 1157).

Life expectancy at birth for Indigenous Australians falls well below the levels for Australia as a whole. Sixty-five percent of Indigenous female deaths and 76% of Indigenous male deaths occur before age 65, while the majority of non-Indigenous deaths (80% female and 70% male) occur after 65 years; before 45 years 34% and 45% of Indigenous females and males have died, compared with only 6% and 10% of non-Indigenous females and males (ABS and AIHW 2001). In 1997-1999, the life expectancy of Indigenous females and males was 63 years and 56 years respectively, while for the non-Indigenous population it was 82 years and 76 years (ABS and AIHW 2001). Death rates were higher for almost all causes and in every age group, the largest difference being in the 35-54 years age group where mortality was five to six times the overall rate for Australia (AIHW 2002a). In this chapter I examine in more detail some of the causal factors underlying these differences, and present additional evidence from studies outside Australia in discussion of fundamental background causes.
3.1. Health in socioeconomic context

Aboriginal people, regardless of location, experience greater health problems than their non-Aboriginal counterparts, although health of urban Aboriginal people tends to be better than rural Aboriginal populations (Najman et al. 1996). Aboriginal demography and health resemble those of less industrialised countries more closely than those of Australia as a whole. In this population ‘in transition’ in terms of health, infectious disease remains at a higher level than among non-Indigenous people, while chronic degenerative disease has overtaken rates in other groups. A recurring theme for many of the post-colonial populations most affected by diabetes is that they are socially marginalised, have lower SES and are not culturally dominant. The explanation of diabetes predisposition among such populations may therefore have more to do with the socioeconomic environment than with ethnically specific genes.

Poverty and social disruption means fewer life choices are readily available, and those that are available tend to be less healthy (Anderson 1996). For example, Indigenous people may not have the same opportunities as non-Indigenous people to obtain affordable healthy food. In rural and remote areas, stores are less likely to stock items such as reduced-fat milk and wholemeal bread (ABS and AIHW 2001). When healthy food is available, there may be competing priorities for income, and a lack of knowledge about the nutritional values of certain foods (ABS and AIHW 2001). Knowledge about healthy foods does not necessarily influence dietary behaviour (Harrison 1991). In some communities there are problems with food supply and fresh food in particular may be very expensive in rural and remote areas (Hoy et al. 1997), and the relative costs of take-away food and tobacco have risen less in recent years (ABS and AIHW 2001) (see Section 4.4.6 for fresh food availability and cost in the study community).

Current socioeconomic disadvantage arises from a history of dispossession (Chapter Four); colonisation deprived Indigenous people of their established means of shelter, good nutrition, and a healthy environment through the destruction of the Aboriginal economy (Saggers and Gray 1991). The subsequent socioeconomic inequalities persist to the present day, and have a profound impact on the health status of the Indigenous population. Socioeconomically, Australia’s Indigenous people are markedly disadvantaged in relation the rest of the population, in terms of education, employment, income, housing and a range of other socioeconomic factors (ABS and AIHW 2001).

Health and SES have long been recognised to be linked so that in a highly industrialised country such as Australia, high SES groups have better health than low SES groups (for example,
SES influences access to resources (Wohlfarth 1998) and the social structure of a society distributes exposure to environmental hazards by conditioning or promoting behaviours which can either damage or promote health (Blane 2001). Although some aspects of health inequality can be attributed to differences in exposure to specific risks, influence of SES remains even after controlling for known risk factors such as smoking (Chaturvedi et al. 1998; Birch 2001). According to Najman and Davey Smith (2000), social class becomes embodied by two related means: firstly, through a lifelong accumulation of experience and its impact on human biology and behaviour, and secondly through developmental influences during a critical period, as in the programming hypothesis.

Inequalities in SES are associated with inequalities in mortality for almost all causes of death in all age groups (Najman and Davey Smith 2000). This pattern is repeated in all the more industrialised countries, so that even if overall health is improving, inequalities remain (Blane 2001). In addition, the relationship between low SES and poorer health does not necessarily hold if a group as a whole is already at risk, as Gaillard et al. (1997) found among African-Americans. Thus even relatively economically better off Indigenous people are likely to suffer from socioeconomic disadvantages in health compared with non-Indigenous people. As long as inequalities remain, disadvantage in health persists, or even increases, even when over time there is an overall decline in mortality rates (Turrell and Mathers 2001).

Women with low SES are more likely to bear low birthweight babies, and the wider socioeconomic and physical environment is a major contributing factor to infant mortality (Turrell and Mengersen 2000). Low birthweight correlates with lower Apgar score (Streatfield et al. 1990) and is a major predictor of infant mortality, especially neonatal mortality (Streatfield et al. 1990; Fejo and Rae 1996; Turrell and Mengersen 2000).

Lower SES groups have higher rates of infant and child morbidity, associated with higher rates of low birthweight through both pre-term births and IUGR, higher placental to weight ratio at birth, developmental delays, more episodes of diarrhoea and vomiting, prolonged acute illnesses and more chronic health problems (Turrell et al. 1999). Infant mortality is inversely associated with SES in all countries with available data (Turrell and Mengersen 2000). In an Australian
study of statistical local areas\textsuperscript{14} in mainland capitals, standardised infant mortality ratio was found to be highest in areas with a greater concentration of low income families, independent of low birthweight, Aboriginality, and ethnicity (Turrell and Mengersen 2000).

Within highly industrialised Western countries, those in lower SES groups are not only more likely to develop diabetes, but are also more likely to die from it than those in higher SES groups, as Chaturvedi \textit{et al}, (1998) found in the UK. A similar pattern is found in Australia: excess mortality in low SES groups due to diabetes rose from the 1980s to the 1990s, from 66% to 70% in women and 24% to 32% in men, which Turrell and Mathers (2001) concluded indicated improved control of the disease among those in higher SES groups.

While the association between SES and health status is undisputed, not all studies of SES and health have been consistent in their findings for both women and men. Generally, women in lower SES groups tend to suffer most from the effects of poverty, as their social disadvantage is compounded by their gender (Verbrugge 1989). Turrell and Mathers (2001) found in an Australian study that excess mortality due to socioeconomic circumstances was more pronounced among women than men. This suggests that other culturally specific factors related to gender come into play; where women have lower social and economic status in the general population, then they suffer more from chronic disease than men of comparable status.

The following two sections draw heavily on two biennial AIHW reports, \textit{The Health and Welfare of Australia’s Aboriginal and Torres Strait Islander Peoples} (ABS and AIHW 2001), and \textit{Australia’s Health 2002} (AIHW 2002a), which provide the most up-to-date information available on contemporary health status of Indigenous people. These reports place a particular focus on comparison of health indices with those of the non-Indigenous population, and contain much more detailed and thorough accounts of Indigenous health status than the summary presented here.

### 3.2. Infant and child health

Infant and child mortality are key indicators of population health, and the poorest groups in less industrialised countries suffer poorer infant health (Wagstaff 2000). In Australia, the picture of

\textsuperscript{14} SLAs – the smallest divisions identified in the Census of Population and Housing conducted five-yearly by the Australian Bureau of Statistics.
Aboriginal child health is very similar to that of less industrialised nations. Even in urban settings, greater health problems are experienced by Aboriginal children than non-Aboriginal children, although health tends to be better than in rural populations (Najman et al. 1996). In a recent study within an urban population Indigenous mothers were less likely than non-Indigenous mothers to describe their child’s health as ‘excellent’, their children were more likely to have been treated by a doctor in the six months prior to the study and more likely to report child health problems as limiting their activity (Najman et al. 1996).

Between 1988 and 2000, IMR for Indigenous infants was 14.9 per 1000, nearly three times that of IMR for Australia as a whole (AIHW 2002a). The primary causes of infant death were conditions originating in the perinatal period, including maternal complications of pregnancy, slow fetal growth and fetal malnutrition, disorders relating to short gestation and unspecified birthweight (AIHW 1998). High rates of infant mortality in Indigenous babies are associated with poverty and economic deprivation, less prenatal care, poor nutrition and other lifestyle factors (Turrell and Mengersen 2000). The same study found that there was no correlation between IMR and indigeneity in capital cities, once the effects of income were accounted for. Similarly, a study by Powell and Dugdale (1999) in a rural area which included Cherbourg concluded that Aboriginality had little effect on birth outcomes once specific influences on birthweight, such as maternal anaemia and alcohol use, were considered.

The mortality rates of older children are three times higher than their non-Indigenous counterparts, and although Indigenous male deaths outnumber female deaths, the differences between Indigenous and non-Indigenous were similar for both sexes (AIHW 2002a). Hospitalisation rates are higher for children identified as Indigenous than those not so identified. The rates are also likely to be greatly underestimated due to under-identification of Indigenous children as such (AIHW 2002a).

3.2.1. Birth outcomes
Recent perinatal statistics for Indigenous births compared with non-Indigenous births are summarised in Table 3.1 below. Queensland data, where available, are included in addition to summary figures for all of Australia, as Cherbourg is located in Queensland. Queensland had the greatest number of babies born to Indigenous mothers of any state or territory (Day et al. 1999).
Table 3.1. Summary of perinatal statistics, Indigenous and non-Indigenous, Queensland and all Australia.

<table>
<thead>
<tr>
<th></th>
<th>Queensland Indigenous</th>
<th>Queensland Non-Indigenous</th>
<th>All Australia Indigenous</th>
<th>All Australia Non-Indigenous</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low birthweight babies (%)³</td>
<td>12</td>
<td>7</td>
<td>12</td>
<td>6</td>
</tr>
<tr>
<td>Mean birthweight (g)⁴</td>
<td>3177</td>
<td>-</td>
<td>3149</td>
<td>3365</td>
</tr>
<tr>
<td>Neonatal mortality rate (per 1000 live births)³⁵</td>
<td>10.0</td>
<td>3.9</td>
<td>7.6</td>
<td>3.0</td>
</tr>
<tr>
<td>Perinatal mortality rate (per 1000 total births)³⁶</td>
<td>22.6</td>
<td>10.9</td>
<td>20.7</td>
<td>9.8</td>
</tr>
<tr>
<td>Births to mothers aged less than 20 years (%)³</td>
<td>19</td>
<td>6</td>
<td>22</td>
<td>5</td>
</tr>
<tr>
<td>Mean maternal age (years)⁶</td>
<td>-</td>
<td>-</td>
<td>24.4</td>
<td>28.9</td>
</tr>
</tbody>
</table>

⁴ Data collected 1994-1996 (Day et al. 1999).
⁵ Death within 28 days of live birth.
⁶ Includes fetal deaths (stillbirths) and neonatal deaths.

Babies born to Indigenous women are twice as likely as non-Indigenous to weigh less than 2500g and hence be classified as low birthweight. Indigenous female babies are more likely to be low birthweight than males (13.6% vs. 11.2%) with mean birthweights of 3087g and 3209g respectively (Coory 1997; Day et al. 1999). Female infants tend to weigh slightly less at birth than males (Section 7.1.2).

Indigenous babies are also more likely to be of very low (<1500g) or extremely low (<1000g) birthweight. From 1994-1996, 2.7% and 1.3% of Indigenous and non-Indigenous babies respectively were very low birthweight, while 1.6% and 0.7% were extremely low birthweight (Day et al. 1999). The excess of low birthweight in Aboriginal infants can largely be explained by a shift to the left of the entire birthweight distribution (Dugdale et al. 1990b). There has been little change in the proportion of low birthweight Indigenous babies over the last decade (ABS and AIHW 2001), although mean birthweight has increased marginally (Day et al. 1999).

Birthweight has a U-shaped association with infant survival, with both the low- and the high-weight babies at increased risk (Mackerras 2001). Heavy babies (>4500g) also have increased risk of neonatal mortality (Coory 2000b), often due to complications from maternal...
diabetes. In the period 1994-1996, 1.2% of Indigenous babies and 1.7% of non-Indigenous babies were of high birthweight (Day et al. 1999). In some Aboriginal and Torres Strait Islander communities, recent increases in mean birthweight have occurred so that mean birthweight approaches the non-Indigenous mean. This increase may reflect obesity among the mothers and a higher prevalence of diabetes, which implies that higher birthweights are not necessarily associated with a decrease in mortality (Streatfield et al. 1990; Coory 2000b). In the Torres Strait, for example, the distribution of birthweight is more like the non-Indigenous population, but infant mortality rates are similar to the Aboriginal population, approximately 2.5 times non-Indigenous rates, leading Coory (2000a) to conclude that birthweight may not be a useful measure of infant health in populations with a high rate of diabetes and obesity, as babies can be heavier without being any healthier. Higher birthweights in the Torres Strait relative to those of mainland Aboriginal babies may also simply reflect associations with larger adult body size.

Table 3.1 also illustrates that the perinatal statistics for Queensland for Indigenous babies are slightly worse than the average for Australia, with higher neonatal and perinatal mortality. Interestingly, the proportion of Indigenous low birthweight babies is slightly lower in Queensland than for all of Australia. This may be at least partly accounted for by other factors, such as the smaller proportion of mothers aged under 20 years (see Section 3.2.2). Alternatively, higher risk pregnancies may for some reason not be surviving to term, resulting in fewer low birthweight babies being born. A further possibility is that there are a significant number of Torres Strait Islander mothers living in Queensland (Torres Strait and mainland Indigenous data are not separated). As non-Indigenous Queensland babies also fare slightly worse on nearly all measures than non-Indigenous babies nationally, it may be that similar factors are contributing to both, amplifying poorer Indigenous outcomes. Queensland is the most decentralised state in Australia, having the lowest proportion of its population living in major cities. Hospital and health care facilities available outside cities, even in major centres, may be less adequate. For example, in central Queensland Mercer et al. (1994) found survival of Aboriginal babies to be very similar to that of all babies, which is suggestive of location being more important than indigeneity.

Medical intervention in delivery, such as the use of forceps, vacuum extraction or caesarean section, was less likely to occur for Indigenous births, although when a procedure such as a caesarean was used it was more likely to be for an emergency rather than elective (Day et al. 1999). This may be because on average Indigenous women have less access to specialised
Contemporary Aboriginal health

maternity services, and when they do attend, any problem may be more advanced. Maternal mortality is much higher among Indigenous women, who account for nearly 15% of Australia’s maternal deaths (Bastian 1993) but only 3% of births (Day et al. 1999). Pre-term deliveries in Aboriginal pregnancies have between three and four times the risk of still birth in non-Aboriginal pregnancies (Coory 1998).

Reduced access to, or uptake of, antenatal care may contribute to low birthweight and higher perinatal mortality and morbidity rates for Aboriginal women (de Costa and Child 1996), even in urban areas. This could explain why in Brisbane, for instance, Aboriginal mothers are 1.5 to two times as likely as non-Aboriginal mothers to have an adverse pregnancy outcome (Najman et al. 1996). Perinatal mortality among Aboriginal babies in South Australia is three times the rate for all of South Australia, and is not declining parallel to non-Aboriginal perinatal mortality (Roder et al. 1995). Available antenatal and postnatal care may not be culturally appropriate, for example, ‘intimate care’ may be provided by male practitioners (Bastian 1993).

3.2.2. Influences on Aboriginal birthweight

*Maternal anthropometry*

Indigenous mothers tend to be shorter than non-Indigenous mothers (Gracey 1991; Blair 1996; Cameron 1996; Sayers and Powers 1997a), and this may partially account for differences in observed birthweights. Sayers and Powers (1997a) and Mathews et al. (1999) found that Aboriginal birthweight increased by 16g and 17g respectively for every centimetre increase in maternal height. Unfortunately, data on maternal height and BMI, pre-pregnancy weight and gain during pregnancy are not available for use in the present study.

*Maternal age and parity*

Indigenous women tend to give birth at much younger ages (Streatfield et al. 1990; Gracey 1991). Over 80% of Indigenous women giving birth are younger than 30 years compared with 54% for non-Indigenous mothers (AIHW 2002a).

In a study of births between 1972 and 1988 on seven Queensland Aboriginal communities, Streatfield et al. (1990) found that mothers under 20 years accounted for 30% of births, and that these births were more likely to be SGA. As parity and maternal age are related, early births are more likely to be of lower birthweight than later births. Birth rates to Indigenous women under 20 years have declined in recent years (Day et al. 1999).
Aboriginal mothers are also more likely to be multiparae, which may increase overall obstetric risk (Gracey 1991), but reduce the chance of having a low birthweight infant as birthweight usually increases with both age and parity (Blair 1996; Cameron 1996; Catalano et al. 1998; Day et al. 1999). Very high maternal parity (more than four previous births) is, however, also associated with reduced birthweight (Humphrey and Holzheimer 2000).

**Maternal infection**

Infections such as pelvic inflammatory disease (PID), urinary tract infections (UTI) and reproductive tract infections occur at higher rates among Aboriginal women than among non-Aboriginal women (Blair 1996; de Costa and Child 1996; Mackerras 2000). *Ascaris* infection has been extremely common in some communities (Dugdale 1980), and may contribute to nutrient deficiencies among pregnant women (and their children, see Section 3.2.3).

**Gestational age**

Low birthweight may be due to either prematurity or IUGR, and often it is not possible to distinguish between those who are SGA but born at full term and those who were born prematurely, especially when using archival data when length of gestation may not have been known or recorded accurately. Whether pre-term or SGA babies comprise the majority of Aboriginal low birthweight is debated. Predictors for Aboriginal SGA babies are low maternal pre-pregnancy weight and/or low gestational weight gain (indicating poor maternal nutrition), late presentation to antenatal care, low maternal age, maternal smoking and both low and high parity (Algert et al. 1993; Mackerras 2001). Predictors for Aboriginal pre-term birth are maternal anaemia, infections, smoking, diabetes and pregnancy-induced hypertension (Coory 1997; Mackerras 2001). Risk of pre-term labour also increases with distance from an urban centre (Fitzpatrick 1996). These clusters overlap, and the elements of each are more common among Aboriginal women (Mackerras 2001).

Determining gestational age can be very difficult, especially where date of last menstrual period (LMP) is unknown or there is little antenatal care (Coory 1997). Such information for Aboriginal pregnancies is not often available (Blair 1996).

Humphrey (1996) concluded from a Cairns study that there was no significant difference in birthweights between full-term Aboriginal and non-Aboriginal infants, but that there was a significant excess in pre-term births. Coory (1997) agrees that pre-term births among Aboriginal infants contribute significantly to low birthweight, accounting for about two-thirds of low birthweight infants. Coory (1997) also states that methods for calculating gestational age
are inconsistent and may lead to misclassification. If pre-term births did not contribute significantly to low birthweight, Coory concludes that an ‘implausibly large’ number of full-term babies must have been misclassified as pre-term (Coory 1997, p. 85). As the maximum rate of fetal growth occurs between 32 and 38 weeks, however, even a slightly pre-term infant may have a much reduced birthweight (Streatfield et al. 1990).

Sayers and Powers (1997b) found that Aboriginal gestational ages are frequently underestimated by approximately one week, so the assumption is reasonable that many Aboriginal babies are born small rather than early. They also point out that the pregnancies for which an accurate estimate of gestational age is available, such as LMP or ultrasonography, are likely to belong to a group with lower risk factors than for those without certain dates (Sayers and Powers 1997b). In a study in the Pilbara and Kimberley regions, Smith et al. (2000) found the birthweights of full-term Aboriginal infants to be only moderately depressed, and within two weeks they had recovered the deficit. Mackerras and Sayers (2000) argue that misclassification of many infants as pre-term is extremely likely if they are SGA, especially when different methods are used by different people to estimate gestational age, if estimates are available at all. Estimates may still be problematic, especially without the use of ultrasound measurements (Mackerras 2000).

Some authors have argued that because low birthweight Aboriginal babies may have higher survival rates than non-Aboriginal babies of the same size they are more likely to be SGA than premature (Seward and Stanley 1981; Gogna et al. 1986; Mackerras 2000). Aboriginal birthweights are possibly higher at lower gestational ages (<35 weeks), and lower at higher gestational (>35 weeks) ages than non-Aboriginal weights (Kliewer and Stanley 1993; Blair 1996; Mackerras 2000), implying that growth follows different trajectories at different stages of pregnancy. The mean weights for Aboriginal and non-Aboriginal infants at 35 weeks are the same, and it is after this that divergence occurs (Mackerras 2000).

Sayers and Powers (1997a) found in a Darwin study that of the 13.9% of Aboriginal infants who were low birthweight, 70% were SGA, 47% were pre-term (17% were both). In addition Humphrey and Holzheimer (2000), studying low birthweight in Aboriginal infants in Far North Queensland, concluded that although gestational age was the strongest predictor of birthweight, SGA was not the most significant factor in low birthweight among Aboriginal infants. The mean gestational age difference between Aboriginal and non-Aboriginal infants was only four
days, and was therefore not considered relevant to the differences in weight between the two groups.

Humphrey and Holzheimer (2000) suggested that improved social support and improved pregnancy care of individual women are more important than medical interventions aimed at reducing pre-term labour. Risks for having an IUGR baby are mainly sociological and biological, while those for pre-term birth were mainly to do with reproductive history (Sayers and Powers 1997a).

For low birthweight infants, IUGR is the more frequent cause in less industrialised countries while pre-term delivery is more likely than IUGR in industrialised countries (Kramer 1987). Aboriginal child health, especially in past decades, follows more closely patterns of less industrialised countries rather than industrialised countries and the rest of Australia. Although the balance between SGA and prematurity as the major factor in low birthweight births to Aboriginal women may vary between communities, the evidence is weighted more in favour of SGA as a primary cause of low birthweight in Aboriginal infants.

3.2.3. Postnatal growth

Height-for-age provides an indication of the long-term nutritional status of a child, while weight-for-height (W//H) gives a short term indication (Skoufias 1998). Weight-for-age (W//A) reflects both to some extent (de Onis et al. 1993). As with birthweight, the same figure can be interpreted in various ways; low W//A may signify recent but not chronic malnutrition (thin but appropriate height-for-age) or stunting from past malnutrition (small but well-proportioned) with satisfactory current health status. For example, a study of children in Havana found those who were fatter were also usually taller than their peers (Amador et al. 1996), so they may have high W//A but appropriate W//H.

There has long been debate within the field of child health about the uses of growth reference data as a comparative term of reference in the study of growth, whether at the individual or population level. The first reference applied internationally was the Harvard Standards. These were derived from a longitudinal study that took place between 1930 and 1956, where the entire study population came from the Boston area of the United States (Jelliffe 1966). The NCHS (National Center for Health Statistics) 1977 reference data were calculated from the US-based Fels Longitudinal study (1928 to 1975) and were adopted by the World Health Organization (WHO) in 1983 as an international reference for child growth. The name change to ‘references’
reflected a softer approach to their use, as a ‘frame of reference’ without implying that it is necessarily optimal, normal, or desirable (Neumann 1979). A ‘standard’ is assumed to represent ‘healthy’ growth, while a ‘reference’ makes no claim as to the health of the sample population (Cole 1998c). (That the children included in calculating the reference were the ones who had survived does imply a minimal criterion of health.)

The NCHS 1977 references have also been criticised for being unrepresentative; the data were gathered from mostly white middle-class families, most infants were breast-fed for only a very short time or formula-fed, while patterns of weight gain among breast-fed infants differ markedly from infants who are fed formula (Rousham and Gracey 1997; Smith et al. 2000; Kuczmarski et al. 2002). Breast-fed infants have higher rates of growth for the first two or three months, while from six to twelve months breast-fed infants weigh less than those fed formula (Kuczmarski et al. 2002). Birthweight also affects breast-milk output, as mothers of smaller babies tend to produce less milk (Rogers et al. 1997). Breast-feeding is frequently associated with slower rates of growth than formula-feeding (Rowland 1985), although in a Mexican study Villalpando and López-Alarcón (1999) found breast-fed infants to be taller and heavier at six months than those fed formula. Among well nourished mothers, exclusive breast-feeding promotes satisfactory growth in infants until four to six months but faltering (see below) may then occur in the absence of supplementary feeding due to insufficient micronutrients (Rogers et al. 1997; Ryan 1997). Supplementary feeding and weaning, however, introduce new challenges to infant growth (Section 3.2.4).

Also at issue is the widespread use of a Western-derived reference that assumes Western patterns of growth to be optimal while rates of childhood obesity in these populations is on the rise (Rowland 1985), as is adult obesity. Cole (1998b), too, questions assumptions that growth curves derived from North American children represent ‘healthy’ growth, as they are not globally among the tallest; aside from some genetic input, greater stature is recognised as attaining maximum growth potential through optimal nutritional status. Therefore what matters is how much importance is placed on variation from the reference population, given the circumstances that influence child growth within a given population.

The most recent WHO-recommended and widely applied reference is the CDC (Centers for Disease Control and Prevention) 2001 growth reference, used for national surveys in the United States. These new references are based on the WHO recommendations of exclusive breast-feeding for the first four months and specifically incorporate data from ethnically diverse
groups, and appear reasonably representative of growth patterns for many different populations (Ulijaszek 2001; Kuczmarski et al. 2002). Percentiles of the weight data for each age (in months) are publicly and readily accessible.\textsuperscript{15} The CDC also make available growth references for other national populations. There are no detailed Australian growth reference data, and none for its Indigenous population (but see Smith et al. 2000, who have created a small set of data).

\textit{Aboriginal postnatal growth}

Health of Indigenous children is significantly worse than the health of non-Indigenous children in Australia (AIHW 2002a), and this is evident in disrupted patterns of child growth. Child growth is often used as a proxy measure for the health of a whole community, as good child health is seen to represent good standards of hygiene, services, economics and education (Dugdale et al. 1994).

‘Growth faltering’ is a term used to describe a flattening in the observed gradient of growth relative to a reference population. Jose and Welch (1970) and Cox (1979) were among the first to document significant faltering in growth among Aboriginal children. Faltering in both height and weight growth has since been observed to occur in a number of different Aboriginal populations throughout Australia,\textsuperscript{16} usually between three and 12 months of age (for example, Gracey and Sullivan 1988; Gracey et al. 1989; Gracey 1991; Dugdale et al. 1994; Rousham and Gracey 1997; Smith et al. 2000; Henneberg et al. 2001; Paterson et al. 2001). Faltering may occur for all children, regardless of their birthweight, although it is especially marked among those who were low birthweight babies (Gracey et al. 1989). Paterson et al. (2001) found that faltering was universal in their study population by 18 months of age. The period of slow growth is usually partially compensated for by catch-up growth in the following one to two years. Faltering growth followed by catch-up is also a frequent pattern in developing countries (Allen 1994; Lunn 2000; Shrimpton et al. 2001) and among disadvantaged groups in developed countries (Binns 1998a).

Faltering child growth in relation to international references is therefore a recurring theme in Aboriginal child growth. Environmental factors which either relate to infant and child growth through nutrition or interact with it include SES, emotional stress, and in some regions season

\begin{itemize}
\item \textsuperscript{15} CDC: \url{http://www.cdc.gov/}; comparison dataset used in EpiInfo 2000 (v. 2). Available percentiles: 3\textsuperscript{rd}, 5\textsuperscript{th}, 10\textsuperscript{th}, 25\textsuperscript{th}, 50\textsuperscript{th}, 75\textsuperscript{th}, 90\textsuperscript{th}, 95\textsuperscript{th} and 97\textsuperscript{th}.
\item \textsuperscript{16} The international reference used in these studies to determine ‘faltering’ has changed over time.
\end{itemize}
and climate (Gracey 1991). Of these, SES, sometimes enhanced by remoteness, may be most important (Dugdale et al. 1994; Mueller et al. 2001). As the mean weights of Aboriginal children in some less remote communities approach international references, there is potential for growth patterns to resemble more closely those of reference populations (Dugdale et al. 1994).

Although infants who are born SGA are more likely than others to undergo growth faltering in infancy, early postnatal growth is usually rapid (Harrison 1991). Impaired growth in utero and early childhood may, however, permanently impair growth, so that full growth potential is never achieved. For example, Roberts et al. (1988) and Gracey (1991) found that children who were lighter than average at five years were also more likely to have been low birthweight. Catch-up growth is therefore neither universal nor automatic; Gracey et al. (1989) found little evidence of catch-up growth in the first five years in a Western Australian Aboriginal sample, and boys showed more of a growth deficit than girls.

3.2.4. Factors involved in growth faltering

There are three main reasons why growth faltering occurs: insufficient intake of certain macro- and micronutrients, infection, and non-organic reasons such as psychosocial stress (Newman et al. 1997; Hulanicki et al. 2001).

Nutrition

The types and amounts of foods consumed by children influence their growth patterns. Growth failure may be due not only to low intake of overall energy, but also to reduced intake of high quality protein, vitamins, minerals and essential fatty acids (Neumann et al. 2002).

Breast-feeding declined between the 1930s and 1960s throughout Australia, but has since re-emerged in prominence (Harrison 1991). Breast-feeding has increased among Indigenous women over recent years, so that rates are now slightly lower than for all of Australia, with 27% of Indigenous infants breast-fed for 12 months or more (AIHW 2000). Breast-feeding may prevent growth faltering partly through fewer diarrhoeal episodes (see below), as Villalpando and López-Alarcón (1999) found in Mexico.

Exclusive breast-feeding for several months may not be adequate if the mother is undernourished (Gracey 1991) even if milk volume is relatively unaffected (Allen 1994). Continuing with exclusive breast-feeding beyond six months may limit infant growth due to insufficient nutrients even in well-nourished women, as (Ryan 1997) concluded from an
international review. Specific nutrient deficiencies may be more important than overall energy deficiencies, for example, infant calcium intake can be low if there is low maternal consumption of dairy products (Gracey 1991). Similarly, protein, zinc and iron deficiency may be linked to poor growth (Allen 1994). Deficiencies are often simultaneous, such that when overall energy intake is low, it is also likely that other nutrients which affect growth might be missing (Allen 1994).

Postnatal growth faltering usually begins at around three months, suggesting factors associated with the onset of weaning are involved. The weaning process frequently involves both a decline in nutritional quality and an increase in infection potential.

In a global review, Dettwyler and Fishmann (1992) conclude that supplementary feeding before four months can also limit growth, especially if the quality of food is poor. Common micronutrient deficiencies following from weaning are iron, zinc, iodine, calcium, vitamins A and B12, and folate (Neumann et al. 2002). Many of these nutrients are more readily accessible from animal foods than plant foods, partly because it is difficult for a child to ingest sufficient bulk to gain the nutrients solely from plant foods (Binns 1998b), and even when these exist in high concentrations, they might be poorly absorbed (Neumann et al. 2002). Infants and small children also require a high percentage of their diet to be from lipids, as these are energy-dense (Binns 1998a). For example, Leonard et al. (2000) found in a Papua New Guinea study that linear growth was positively associated with intake of energy and protein from animal sources. Zinc supplements among slow-growing children can increase weight gain and increase the proportion of lean tissue to fat (Golden and Golden 1992).

Physical inactivity reduces energy requirements, and reduces overall intake of food, meaning that deficiencies of micronutrients are more likely among less active children (Hallberg 2001). Child growth can also parallel seasonal variation in food availability, as found for example in Madagascar and Pakistan (Hardenbergh 1997; Liu et al. 1998), while in some Aboriginal communities child diet may vary markedly over much shorter periods, with nutritional intake varying with the pay cycle (Gracey 1991).

*Infection*

Aside from common inadequacies in nutritional content, introducing new foods also has the potential to create opportunities for exposure to novel organisms and subsequent infection, occurring concurrently with reduced maternal immune protection. Exposure to new infections
and the transition to endogenous immunity can interact to exacerbate effects of infection on nutrition and growth (Jose and Welch 1970; Gracey 1991; Lunn 2000).

Infection-malnutrition synergy has been widely documented (for example, Golden 1995; Ryan 1997; Lunn 2000; Neumann et al. 2002). Undernutrition suppresses immune function (Ulijaszek 1998c), and infections in turn promote malnourishment by decreasing appetite, increasing metabolic rate and inhibiting nutrient absorption (Dugdale et al. 1990c; Gracey 1991). Subclinical infections or parasites may also contribute to malabsorption of nutrients (Allen 1994; Lunn 2000). The usual onset of infectious disease such as diarrhoea coincides with weaning and increased infant mortality rates (Kuzawa 1998). Growth faltering is usually associated with repeated infections, especially respiratory and gastrointestinal. Poorly nourished children have a higher incidence of these infections (Dugdale et al. 1990c) and are more likely to be admitted to hospital for them (Gracey 1991). Damage to mucosa may persist into later childhood and into adulthood, causing continuing malabsorption of nutrients (Campbell et al. 2002) well beyond the period of infection.

Most infectious episodes do not have a major impact on child growth, as catch-up growth occurs in the weeks following a diarrhoeal episode (Briend 1990). If on the other hand infections are chronic, long-term consequences on growth are much more likely. In Cherbourg there was widespread infection with *Ascaris* (piperazine was administered frequently to children to reduce parasite loads) (Dugdale 1980). *Ascaris* infection may account for some of the growth deficits observed in Cherbourg (see Section 7.1.3), as heavy loads can reduce nutritional status; infection with intestinal parasites is characterised by iron-deficiency anaemia and the malabsorption of other micronutrients (McGarvey 1998; Wilson et al. 1999; Neumann et al. 2002).

Declines in infection are not always paralleled by improvements in child growth (Dugdale 1996; Rousham and Gracey 1997), suggesting that infection in some Aboriginal communities at least is not as important for growth outcomes as nutrition or other factors.

**Psychosocial stress**

Material deprivation and psychosocial stress tend to co-occur so that children born into social disadvantage are also more likely to suffer from reduced postnatal growth due to stress (Skuse et al. 1996; Skuse 1998). Incarceration and institutional living are demonstrated to affect child growth through contributions to stress, as found for example in a Spanish study (Munoz-Hoyoz...
et al. 2001), probably partly through inadequate physical or psychological stimulation and poor living conditions, which promote high rates of infection and undernutrition (Bogin 1998).

Linear growth failure occurring in the absence of an organic cause has been called ‘psychosocial short stature’ (Skuse et al. 1996), where growth hormone insufficiency is associated with hyperphagia and polydipsia, and affected children might have a normal W/H but low W/A.

**Sex**

Boys showed a greater deficit in W/A than girls in a study of Western Australian Aboriginal children (Gracey et al. 1989); males appear more susceptible to prenatal nutritional stress through more rapid early fetal growth, and weight-matched for girls they have poorer survival (Schell 1998). Postnatally there are differences in energy storage, and girls are more likely to exhibit catch-up growth than boys, as McCowan et al. (1999) found in a New Zealand study. Males appear more susceptible to prenatal stress in terms of brain development and the imprinting effects of maternal glucocorticoids on the developing HPA axis (Purifoy 1981). Rebato et al. (1998), however, concluded from a study in the Basque Province, that girls were more sensitive to socioeconomic conditions in their accumulation of central fat. This propensity may be protective during childhood; fat stores are mobilised during nutritional stress and female infants are generally less susceptible to malnutrition than males, as they make a greater investment in energy storage and increased fatness (Kuzawa 1998).

**3.2.5. ‘Catch-up’ and ‘catch-down’ growth**

Following a period of growth faltering, once the stress (nutritional, infection or psychosocial) resolves or is ameliorated, there is usually a period of rapid growth or recovery, where growth of a small infant or child increases in gradient towards the median. For example, babies who are born with IUGR usually exhibit catch-up growth in the first few postnatal months (Ulijaszek 1998b), especially those who were disproportionate at birth (light for their length) (Newman et al. 1997). After the first six months of catch-up growth, growth rate slows for the first two or three years at least among those who were small and tends to be higher among those who were large (Gracey 1998). By seven years, those who were large at birth tend to be large again, while those who were small tend to be small (Schell 1998). Approximately 20% of SGA infants remain small for their age at two years (McCowan et al. 1999). Rowland (1985) concluded from a study of Aboriginal children that if catch-up growth among has not occurred by three years of age then it may never occur at all. Attaining full catch-up growth may depend on negotiating in particular two sensitive periods successfully, the first few postnatal months and
between eight and 18 months, as Karlberg et al. (1996) concluded for children in both Hong Kong and Sweden. Vik et al. (1996) found that catch-up growth in Norwegian and Swedish infants took longer for the offspring of smokers than of non-smokers. While infants who are below the median tend to catch up, growth of large infants slows so that they ‘catch-down’ towards the median (Cole 1998a; Hediger et al. 1998; Schell 1998).

Catch-up weight gain is related linearly to energy intake and not to protein (provided protein makes up at least 9% of diet) (Golden 1998). Catch-up growth is more rapid for weight than for length (McCowan et al. 1999). Severely wasted children can gain weight at 20 times the normal rate in a period of catch-up growth, with velocity declining as the child recovers (Golden 1998). Rapid catch-up growth, however, may lead to depleted stores of one or more nutrients, putting a child at greater risk of subsequent malnutrition and secondary growth faltering (Rowland 1985; Golden 1998). Faster growing infants might also be at greater risk of iron deficiency, as the demands of growth on iron are greater (Ryan 1997).

Leitch (1951), in a study involving pigs, was among the first to observe that variable growth velocity produces different results from constant velocity as body proportions and fatness become altered. Skeletal muscle in children who undergo catch-up growth may remain somewhat atrophic, while fat deposition increases as measured by skinfold thickness (Golden 1998). Where this fat is preferentially deposited may have implications for later diabetes and metabolic disorders. For example, Aboriginal children have a high subscapular-triceps skinfold ratio (SSF:TSF), which suggests they have less subcutaneous fat on their limbs relative to truncal fat than non-Aboriginal children (Dugdale et al. 1980). Children with lower birthweights, lower W//A between one and two years, and lower W//H aged five to 10 years had higher SSF:TSF ratio, suggesting that past (or present) nutritional stress encourages more central fat distribution, and those with lower birthweights and low weight gain from one to two years of age had higher SSF:TSF ratio (Dugdale et al. 1980; Dugdale and Lovell 1981; Dugdale 1988). In well-nourished Aboriginal children, skinfold ratio was similar to that in a comparative group of non-Aboriginal children (Dugdale 1988), suggesting that the pattern is nutrition-related. Not only does catch-up growth appear to alter body composition, but continued poor growth throughout childhood may also contribute to later problems, especially if obesity develops in adulthood.

Given the risks associated with a more centralised pattern of fat distribution, there might be long-term metabolic consequences associated with a centralised pattern of rapid weight gain,
although there are short-term benefits of promoting catch-up among small infants. Catch-up growth could expose infants to risks of later childhood obesity and subsequent adult obesity and CVD risk. Even if catch-up growth is directly associated with later risk factors for diabetes and CVD, promoting adequate fetal and infant growth is more important in the short term, particularly in developing countries. This reflects what may be a trade-off between risk of early infections and later metabolic disease (Victora and Barros 2001).

Dugdale et al. (1980, 1984) suggest that the growth of children depends greatly on the attitudes of parents and the resources they have access to, such as efficient family planning, water supply and sanitation, adequate space and affordable healthy foods, education, housing, income, and access to health care. Added to this list could be familial and social obligations and expectations, health knowledge and understanding, experiences of health care, locus of control and priorities when it comes to health behaviours. The social contexts of health in Cherbourg are discussed in Chapter Ten, particularly in relation to decisions and practices of adult lifestyle.

3.3. Adult health

A striking feature about the nutrition of Aborigines is how children who are often stunted and wasted evolve into obese young adults who seem particularly susceptible to diabetes, hypertension and cardiovascular diseases associated with affluence and a sedentary lifestyle (Gracey 1991, pp. 267-268).

3.3.1. Demography

The age profile of the Indigenous population is younger than that of the total Australian population, with a median age of 20 years whereas 34 years is the median for the total Australian population (AIHW 2002a). (See population pyramid for the study community in Section 4.4.1). The younger age structure is the result of both higher fertility and higher mortality at younger adult ages. As age and health are closely related, ‘the relative youth of the Indigenous population may mask health status when compared with the older non-Indigenous population’ (ABS and AIHW 2001, p.11). This is especially the case where degenerative diseases, such as diabetes which affects a greater proportion of older people, are involved in contributing to a poor health profile overall.

There is similar overall self-assessed health rating between Australia’s Indigenous and non-Indigenous people (Cunningham et al. 1997), suggesting that the validity of the assessment is questionable given higher rates of morbidity and mortality. The relative youth of the
Indigenous population, however, attenuates the health assessment differences between populations.

Aboriginal people tend to acquire degenerative disease at a much younger age than non-Aboriginal; Type 2 diabetes typically has its onset among those in their 30s among Aboriginal people rather than 40s to 50s in the rest of the population (de Courten et al. 1998). Aboriginal people are also more frequently hospitalised, primarily for care relating to dialysis (ABS and AIHW 2001). This reflects high prevalence of end-stage renal disease (ESRD), often arising out of complications of diabetes, in addition to chronic nephritis. Recently, however, it has been suggested that Indigenous hospitalisation data might be skewed by the frequent use of hospital services by a minority rather than an increase in use overall. Dugdale and Watlemaro (2001) found that over a five year period, half of all visits to the Cherbourg community hospital were made by 14% of the patients. Some of the excess hospitalisation rates might also be accounted for by different patterns of hospital use. For example, in the community where the present study took place, the absence of a general practice (until recently) meant that the hospital was relied on for all medical purposes.

Indigenous people are over-represented in mortality statistics for every age group, with approximately three times as many deaths as expected for all causes (ABS and AIHW 2001). Other than injuries and respiratory disease, diabetes and related metabolic disorders such as CVD are responsible for most of the excess morbidity and mortality experienced by the Indigenous population (Cunningham and Paradies 2000). CVD is the major cause of mortality and morbidity among Aboriginal Australians and accounts for 30% of all Indigenous deaths (ABS and AIHW 2001).

### 3.3.2. Diabetes

Between 1997 and 1999, diabetes was the underlying cause of death in 10% of Indigenous females and 6% of Indigenous males, with similar rates for diabetes as an associated cause of death. The standardised mortality ratio (SMR) for diabetes was 13.5 for Indigenous females and 10 for Indigenous males (ABS and AIHW 2001). The category of endocrine and metabolic disorders was the fifth highest cause of Indigenous deaths between 1995 and 1997 (Cunningham and Paradies 2000).

National prevalence estimates of diabetes rely on self-reported diagnosis and studies of individual communities. If only Type 2 diabetes is considered, then overall rates may be as low
as 2% among females and 2.5% among males (ABS and AIHW 2001) (although recent national estimates suggest that in total, 6.8% of Australian females and 8% of males aged over 25 years have diabetes (Dunstan et al. 2002)). In contrast, diabetes diagnosis was reported by 7.1% of Indigenous people aged 20 to 44 years, by 24% of those aged 45 to 54 years, and by 17% aged over 55 years (ABS 2002b). The decline in prevalence in the older age group probably reflects higher mortality of those with diabetes, or it could indicate better past or present health of that cohort. In Queensland as a whole, the prevalence of Type 2 diabetes is slightly below the national average, at 1.7% and 2.2% for females and males (ABS 2002b).

Estimates from various Aboriginal communities demonstrate that diabetes and risk factor prevalence are not uniform across the Aboriginal population, but depend on the degree of Westernisation; more traditional living groups have lower estimates (Harrison 1991). Prevalence of diabetes in remote areas increases with proximity to a store, suggesting that some traditional lifestyle factors, namely diet and physical activity, provide a degree of protection against diabetes and related disorders (Gault et al. 1996). Carter and Bartley (1996) estimated that in some communities, up to 40% of adults might be affected, while IGT may occur in up to 50% of adults (O’Dea 1991). The lowest Indigenous estimates are between 10% and 30% (de Courten et al. 1998). The insulin resistance syndrome is becoming more prevalent, even in more remote groups, as transition from hunter-gatherer lifestyle to predominantly store-bought foods is followed by an increase in metabolic disorders 10 to 12 years later (Gracey 1998). A qualitative study by Thompson and Gifford (2000) found that diabetes was perceived the community as the result of living life ‘out of balance’, a view in line with the themes of discord raised in Section 2.5.1.

Much of the higher death rate among Indigenous adults aged over 25 years reflects the earlier onset of diabetes in the Indigenous population (ABS and AIHW 2001), as damage wrought by poor glucose control commences earlier. Generally, diabetes in the Indigenous population is less well-managed by lifestyle change and outpatient care than for non-Indigenous people; for Indigenous people, there were 10 to 15 times more hospital separations relating to diabetes than the rate for people with diabetes overall in Australia (ABS and AIHW 2001). In Australia as whole, it is estimated that only half the number of people with diabetes have actually been diagnosed and are therefore aware they have the disease (Zimmet and Cohen 1995; International Diabetes Institute 2001), and this estimate may hold true for some Indigenous communities even where regular screening occurs (Section 6.3).
Where diabetes was recorded as an underlying cause of death, the main associated causes were IHD (41%), other CHD (26%) and renal disease (38%) (ABS and AIHW 2001). A rise in ESRD has accompanied the rise in diabetes, hypertension and CVD (Hoy et al. 1998); ESRD is attributable to diabetes in 32% of Indigenous ESRD patients in NSW, compared with 13% for non-Indigenous patients (Cass et al. 1999). Rates of kidney failure among Indigenous Australians are the highest in the world (Mathews 1996), and given its frequent association with diabetes, renal disease has sometimes been included as part of the insulin resistance syndrome (Rowley et al. 1997; Rowley et al. 2000b). Among fully ‘Westernised’ populations, including non-Indigenous Australians, it is usual that males outnumber females in rate of ESRD at a ratio of 1.8:1, but among Australia’s Indigenous population the pattern is reversed, with 0.8:1 the ratio overall, and there were more females in all risk factor categories for ESRD except for hypertension (Hoy et al. 1996a). Some of this may be related to higher rates of UTI among Indigenous women.

Death from renal disease was more common among diabetics in a Central Australian study than macrovascular complications, which are more common in Australia’s non-Aboriginal diabetic population (Phillips et al. 1995). It is uncertain what role diabetes plays in the development of renal disease, but it may exacerbate an underlying risk in Aboriginal communities (Phillips et al. 1995). Other complications and co-morbidities of diabetes and the insulin resistance syndrome include neuropathy, retinopathy, peripheral vascular disease resulting in ulceration, gangrene and amputation (Zimmet and Cohen 1995; ABS and AIHW 2001). People with diabetes are more likely to report poorer overall health and more long-term conditions such as hypertension and heart problems (Cunningham et al. 1997, p. 18).

While risk of diabetes increases linearly with BMI among populations of European descent, such a relationship is less apparent among Australian Aboriginal groups, where in some groups at least an increased risk of diabetes becomes apparent at BMIs that are within the so-called healthy range of 20 to 25kg/m² (Daniel et al. 1999).

**Gestational diabetes**

Gestational diabetes typically occurs in Australian women during between 3% and 8% of pregnancies (AIHW 2002a). As Type 2 diabetes tends to occur at a much younger age among Aboriginal than non-Aboriginal people, many women are affected in their child-bearing years. Type 2 diabetes may be initially diagnosed during pregnancy (and hence classified as gestational diabetes until Type 2 diabetes is subsequently confirmed post-partum), or gestational
diabetes that is only temporary. Diagnosis and management of diabetes in pregnancy requires early antenatal visits, an option that is less likely to be available to or taken up by Aboriginal women than non-Aboriginal women. This increases the risk of perinatal complications due to diabetic pregnancy.

3.3.3. Risk factors
The National Health and Medical Research Council (NHMRC) in a recent review concluded that older age, obesity, central obesity, hypertension and family history are independent risk factors for Type 2 diabetes (NHMRC 2000). Evidence that dyslipidaemia was an independent diabetes risk factor was not found to be strong, despite its position as component of the insulin resistance syndrome. The presence of each independent risk factor signifies significantly increased risk of diabetes.

Age
Diabetes prevalence increases with age. Age becomes an independent diabetes risk factor for diabetes for Australia as a whole at 65 years, as this is when prevalence of undiagnosed diabetes is estimated to reach 5% (NHMRC 2000). Among Indigenous people however, diabetes typically occurs at much younger ages, prompting this threshold to be almost halved to 35 years (NHMRC 2000).

Obesity
Overweight and obesity are established risk factors for diabetes. The NHMRC follow the WHO recommendations, that a BMI $\geq 25$ be regarded as overweight and $\geq 30$ is obese, regardless of a person’s sex (NHMRC 2000). Diabetes is more common in those who are obese than others, but for any given BMI, Aboriginal diabetes rates are higher (Daniel et al. 1999). Table 3.2 shows overweight and obesity prevalence ratios for Australia and for Queensland.
Table 3.2. Prevalence estimates of overweight (BMI ≥25<30) and obesity (BMI ≥30) among adults in Australia and Queensland, Indigenous rates (where available) and non-Indigenous rates compared.

<table>
<thead>
<tr>
<th></th>
<th>Queensland</th>
<th>All Australia</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Females</td>
<td>Males</td>
</tr>
<tr>
<td><strong>Overweight %</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Indigenous</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Non-Indigenous</td>
<td>27.7 b</td>
<td>41.2 b</td>
</tr>
<tr>
<td><strong>Obesity %</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Indigenous</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Non-Indigenous</td>
<td>16.7 a</td>
<td>20.4 a</td>
</tr>
</tbody>
</table>

a Data collected 1995 (AIHW 2003).
c Data collected 1994 (Cunningham and Mackerras 1994).

Before European contact, Aboriginal people are thought to have been lean (Gracey 1991), with probably good nutrition in a variety of environments (Franklin and White 1991; Saggers and Gray 1991). Given its association with obesity, it is highly unlikely that Type 2 diabetes was present at any significant level before European colonisation. After colonisation, many Aboriginal people became reliant on introduced foods as rations. Some of these foods were welcomed, such as flour, beef and mutton (Saggers and Gray 1991), at least partly because Aboriginal people no longer had access to their own resources (Franklin and White 1991). When European diet was initially used to supplement traditional hunting and gathering, Indigenous diet may for a short time have been nutritionally better than pre-contact (Saggers and Gray 1991). But any benefits were short-lived as rations took over as the primary or only source of food. The nutritional quality of rations was generally poor, largely composed of refined carbohydrates (white flour, sugar) and fatty meat (Section 4.3.4). Physical activity levels have also decreased markedly in Australia’s Indigenous populations since European colonisation, although there is no quantifiable evidence to demonstrate change through time (de Courten et al. 1998).

The prevalence of overweight and obesity is not uniform across communities, ranging from 0% in traditional-living communities to over 50% in more acculturated communities (Rowley et al. 1997). In a study of Aboriginal and non-Aboriginal people in south-eastern Australia, Guest et
Contemporary Aboriginal health

al. (1993) found a gradient of obesity, from lowest in urban groups, intermediate in country (rural) groups and highest in Aboriginal groups regardless of the degree of genetic admixture with Europeans, and inversely associated with SES. In southeastern Australia, 38% of urban Aboriginal women were obese compared with 18% of non-Aboriginal women, and 25% of Aboriginal men were obese compared with 17% non-Aboriginal men (Guest et al. 1993). Aboriginal women tend to be overweight at much younger ages than non-Aboriginal women (McGrath et al. 1991). Wong et al. (2000) observed BMI in a remote northern Australian community to be much more variable among the women than men at all ages, and found that a large proportion of females and males population to have extreme BMI at both ends of the distribution indicating that both malnutrition and obesity are major health issues. Wong et al. (2000) also found that BMI decreased in older age groups, especially among women. In a Central Australian community O’Dea et al. (1993) noted that BMI increased with age in women but plateaued in men at about 30 years, while WHR continued to increase among men but reached a plateau in women. These age-related patterns, like diabetes and obesity prevalence, are likely to be population-specific as communities vary greatly in their degree of Westernisation. Relative inactivity may contribute to the greater excess weight among women (Wise et al. 1970; McGrath et al. 1991).

Eleven percent of obese Indigenous people report having diabetes, compared with 7% of those who are overweight and 3% of those who are of acceptable weight. Therefore overweight and obesity do contribute greatly to diabetes risk, but variations in BMI may not fully account for variations in diabetes prevalence. For any given BMI, Aboriginal people tend to have ‘considerably more body fat’ than Australians of European descent (Rowley et al. 1997, p.778), a factor which may contribute to this variation.

Central obesity

The NHMRC (2000) evidence-based guidelines conclude that waist circumference, rather than the previously applied waist-hip ratio, is a sufficient measure of central obesity. Circumference thresholds for excess abdominal fat associated with an increase of obesity-associated diseases are ≥80cm for females and ≥94cm for males.

The propensity for central fat distribution may pre-date European colonisation even with overall leanness (Guest et al. 1993), but becomes detrimental to health in the event of sedentary lifestyle and high overall body fat. Aboriginal women have higher percentage of body fat and more centrally located fat than non-Aboriginal Australian women of the same age (O’Dea et al.
Both diet and physical activity can reduce not only overall body fat but also relative abdominal fat (Gutin and Owens 1999). Even at low BMIs, diabetes rates in Aboriginal people may be three to five times the corresponding rates for non-Aboriginal populations (Daniel et al. 1999). This finding suggests that the ‘healthy’ BMI for an Aboriginal person may be lower than for a non-Aboriginal person (O'Dea et al. 1990; Rowley et al. 1997; Couzos et al. 1998; Daniel et al. 1999). In addition, the BMI threshold for ‘underweight’ (BMI <20) may not necessarily reflect poor nutritional status in traditional-living Aboriginal people (Gracey 1991), and could instead be protective against insulin resistance and the insulin resistance syndrome (Rowley et al. 1997). Obesity, in terms of its effects on health, may therefore be underestimated among Aboriginal people when using the same criteria as those used for people of European descent.

Physical activity may lower risk of diabetes by decreasing total body fat and abdominal fat, in addition to improving insulin sensitivity (Gutin and Owens 1999). In an intervention study promoting increased activity and healthy eating, the prevalence of diabetes in a Central Australian community remained constant despite an increase in BMI that occurred during the intervention (Rowley et al. 2000a), which suggests that activity confers health benefits beyond those that are usually associated with reductions in BMI.

**Hypertension**

Hypertension (blood pressure >140/90mmHg) is considered to be an independent risk factor for diabetes (NHMRC 2000). In Aboriginal people it is twice as common among those with IGT than among those with normal glucose tolerance, and four times as common among people with diabetes (Hoy et al. 1996b). Hypertension is most often associated with ageing among Westernised populations (McGrath et al. 1991). In Australia, rates of hypertension are 25.6% among females and 30.7% among males, while in Queensland the rates are slightly lower, at 23% and 29.5% (AIHW 2003). Estimates for the Indigenous population, based on self-reported health, are approximately 45% for females and 50% for males (Cunningham et al. 1997).

**Family history**

A further risk which was deemed by the NHMRC guidelines to be an independent variable is family history, where lifetime risk of diabetes is 40% if one parent has diabetes (NHMRC 2000). Increased risk of diabetes with a positive family history may be due to shared environment rather than shared genes, for example, the development of food preferences and the propensity to be more or less physically active (Section 2.5).
Stress

Although stress is not a recognised independent risk factor for diabetes, its possible role in diabetes aetiology is gaining attention. The Indigenous population is likely to suffer from higher rates of emotional distress and possible mental illness than the wider community (Sibthorpe 1988; ABS and AIHW 2001). Psychosocial stress might therefore be a contributing factor to high rates of Indigenous diabetes.

3.3.4. Risk behaviours

Low SES is associated with increased health risk behaviours (ABS and AIHW 2001). Health risk behaviours, such as heavy drinking and smoking, and factors such as exposure to violence occur more frequently in many Aboriginal groups but do not fully explain the variation apparent in socioeconomic differentials in health (ABS and AIHW 2001). Some contribution is probably also made by social environment, relative work position, quality of social connections (friends, family, community), perceived degree of inclusion or exclusion by society, and perhaps perceived level of control (ABS and AIHW 2001). Higher levels of perceived stress and higher concentrations of cortisol may contribute to poorer health of those in lower SES groups, beyond contributions made by specific risk behaviours.

A smaller proportion of Indigenous people than non-Indigenous people consume alcohol, but of those who do, a greater proportion drink at dangerous levels (ABS and AIHW 2001). Rates of smoking are about twice those of non-Indigenous people, with 43.6% of females and 53.5% of males smoking, compared with 20.5% and 27% respectively (ABS and AIHW 2001). Although both obesity and heavy alcohol consumption may contribute to diabetes, Cunningham and Mackerras (1994) found that people who did not consume alcohol had increased BMIs relative to those who reported consuming alcohol. On the other hand, excessive alcohol consumption may be a contributing factor in the development of obesity through excessive calorific intake (Hoy et al. 1996b). High alcohol intake is also associated with increased triglyceride levels (McGrath et al. 1991), and may add to psychological stress in addition to damaging the liver and pancreas (Couzos et al. 1998). Both alcohol and tobacco exacerbate the complications associated with diabetes. Use of other drugs and substances such as petrol, glue, aerosols, cannabis, heroin and amphetamines also occur at higher rates among the Indigenous population (ABS and AIHW 2001).

The poor status of Aboriginal health begins before birth and continues throughout the life-span. Given the high rates of low birthweight, the patterns of disrupted child growth and the Western
lifestyle of energy excess contributing to adult obesity, metabolic programming in early life may be partially responsible for the high rates of adult diabetes. Programming may contribute to diabetes risk beyond the adult lifestyle factors of energy-dense diet in combination with low physical activity. Catch-up growth is common, and may also contribute to adult diabetes risk.

The present study follows a theme advocated by Weeramanthri (1996), that of a ‘whole-of-life’ perspective, by implementing a life-span approach to examining diabetes, and considering the wider historical, political and social context of the disease. The following chapter introduces the community under study in its historical context and through to the present day, particularly the political and social legacies that have shaped the current health of the community.