

**AUSTRALIAN ABORIGINAL MORTALITY AND CORONARY
HEART DISEASE: A DEMOGRAPHIC INQUIRY**

By

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Declaration

**Except where otherwise indicated this
thesis is my own original work.**

Robert Stephen Hogg

March 1991

For Heather, Erin, and Gloria

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ABSTRACT

This thesis has attempted to evaluate whether Australian Aboriginal mortality patterns are different from those exhibited by indigenous groups in other countries or by the general Australian population; and whether the high rate of coronary heart disease among Australian Aborigines is attributable to the underlying prevalence of specific lifestyle (risk) factors. At a macro-level, I have compared contemporary mortality patterns of and risk factor prevalence levels among Australian Aborigines and other indigenous groups. At a micro-level, I have made the same comparisons between Aborigines living in western New South Wales (especially those in Bourke) and other Australians.

This study has made four important points regarding these issues. The first point is that although Australian Aborigines and other indigenous groups have similar patterns of high adult mortality, the relative importance of some more notable causes of death is strikingly different. The second point is that Aborigines have much higher total and age-specific death rates than other Australians, especially during adulthood and most notably from circulatory system diseases. The third point is that, compared with the general Australian population, Bourke Aborigines have much higher risk factor prevalence levels for a number of coronary heart disease risk factors. The fourth and final point is that Bourke Aboriginal risk factor prevalence levels tend to vary quite considerably by several demographic, environmental, and social-cultural conditions (risk markers) — more specifically, by age, location, employment status, and pay period.

Overall, these findings suggest that the mortality pattern of Australian Aborigines is experienced by no other population; and that lifestyle factors, which are influenced by often uncontrollable conditions, can at least partially account for the observed high rate of coronary heart disease.

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CHAPTER ONE

INTRODUCTION AND OVERVIEW

Imagine a community of a few hundred thousand people — with such high infant mortality rates, high youth mortality rates, low life expectancy, high morbidity. . . . Somebody is always dying. So many seem to be in mourning — and mourning for premature deaths of infants, of young people; there is always somebody to visit in hospital (if you're lucky) and a funeral to go to (if you're not). . .

(Sykes, 1986)

1.1 Introduction

In comparison with any other group in Australia, Aborigines have alarmingly high adult death rates. The greatest proportion of the excess risk of death during adulthood, especially between the ages of 25 and 64 years, is due to circulatory system diseases — largely coronary heart and cerebrovascular disease. Other important causes of death in adults are accidental and violent events and respiratory system diseases. This pattern of high adult mortality is by no means unique to Australian Aborigines, but is also quite common among other indigenous groups worldwide — including American and Canadian Indians, New Zealand Maoris, and some Pacific Islanders.

In an attempt to account for the observed high rates of premature deaths from degenerative diseases among indigenous groups, researchers have developed three different hypotheses. First, lifestyle factors, such as alcohol consumption, cigarette smoking, dietary habits, and exercise patterns, are seen as playing a major role; and the prevalence of these lifestyle factors is assumed to be influenced by underlying demographic, environmental, and social-cultural conditions.

Second, indigenous people are seen as having a genetic predisposition to degenerative diseases which are brought on by the discordance between their genetic make-up and their current lifestyles. Neel (1962, 1982) has suggested that the high prevalence of

non-insulin dependent diabetes mellitus (type 2 diabetes) in some contemporary human populations is due to the imposition of an over-sufficient and steady food supply on a thrifty genotype which was initially designed to take advantage of the sporadic food supply characteristically available to subsistence hunters and gatherers. Weiss and colleagues (1984) have used a similar argument to explain why North American Indians and Mexican Americans of mixed descent have a higher prevalence of obesity, type 2 diabetes, and gall-bladder disorders. This constellation of diseases, designated as the New World Syndrome (Weiss *et al.*, 1984), is thought to have arisen in American Indians by positive natural selection or genetic drift somewhat before or early in the settlement of North America. The current high rates of these diseases are thought to be attributable to the exposure of the genotypes associated with this syndrome to contemporary environmental conditions, especially those associated with recent dietary change.

Third, underlying psycho-social factors are also seen as being significant. While there are undoubtedly many sources of stress among Australian Aborigines, Sibthorpe (1988: 324-345) has identified several psycho-social factors which may affect health and have been noticeable in both the past and the present. These include: dispossession and institutionalization; separation and loss; unemployment; alienation and inequity; and stigmatization and assimilation. Although Sibthorpe's schema was devised with Australian Aborigines in mind, it is also a way of viewing the impact of stress on morbidity and mortality among other indigenous groups.

Dispossession and institutionalization refer to the processes of social and economic disruption, caused by early European contact, and to the varying degrees of government control and restriction that were and still are put on the movement and behaviour of Aborigines.

Separation and loss refer to the loss or removal of a loved one, which in Aboriginal society was formerly brought about by the long-term policies of the Aborigines

Protection and Aborigines Welfare Boards to remove Aboriginal children from their parents; and is currently brought about by the pattern of high adult mortality. Read (n.d.) estimates that during this century one in six or seven Aboriginal children living in New South Wales was removed from his or her parents and that all of these estimated 5,000 children had and have their own private and bitter memories of separation and later problems of adjustment (see Edwards and Read, 1989). Adult premature death has also had a profound effect on Aboriginal society. As Gray (1984a) suggests, the high level of adult mortality reduces the integrity of Aboriginal society and the security of Aboriginal marriage practice and family structure, and underlies the high incidence of single-parent households.

Although unemployment is a usual state for the majority of working-age Aborigines throughout their adult lives, its psycho-social impact on Aboriginal society is not clear. As Sibthorpe (1988: 336-337) states:

Loss of social integration, identified as a critical outcome of unemployment, may be less relevant in Aboriginal communities. Thus while unemployment perpetuates Aboriginal exclusion from the social mainstream and constitutes an element in discrimination and alienation, it may be positively integrative in the context of the minority group. . . . Nevertheless, it would appear that satisfying work conditions, appropriate to Aboriginal values and expectations, are necessary for sustained social reform. . .

Even though the psycho-social impact of unemployment is not clear, its possible health impact should not be underestimated. Verbrugge (1983) suggests that social roles, like the ones associated with employment, marriage, and parenthood, are important because the roles an individual plays can influence the exposure to particular risks of illness and injury and people's attitudes towards physical symptoms, and can affect the willingness and ability of individuals to take appropriate curative actions. Ultimately, these differences in risks, attitudes, and health care opportunities lead to differential rates of health service use, disability, morbidity, and mortality.

Alienation and inequity refer to the social distress in Aboriginal society which is brought about by powerlessness and injustice. The House of Representatives

Committee on Aboriginal Affairs (Parliament of the Commonwealth of Australia, 1979) and other researchers (Thomson, 1984; Gray, 1987a) have recognized that poor health among Aborigines is a product of poverty and powerlessness related to dispossession, discrimination, and restricted opportunities for social participation.

Lastly, stigmatization and assimilation refer to the negative effects on Aboriginal images of self and culture which are brought about by white people's devaluation of Aboriginal life, religion, and culture and by the high social costs of assimilation into white Australian society.

My research interests in this thesis are centred primarily on the first hypothesis — that the high rates of premature deaths from degenerative diseases among indigenous people are due to underlying lifestyle factors. The other two hypotheses, which link high adult death rates with a genetic predisposition to degenerative diseases and with psychosocial factors, are seen as underlying antecedents of morbidity and mortality that can either influence the prevalence or impact of lifestyle factors or put individuals at a greater risk of ill-health.

As a health concept, lifestyle is defined here as a combination of habitual behaviours, or ways of life, that each individual, rich or poor, acquires voluntarily or involuntarily (Hetzl and McMichael, 1989). Rather than focusing on the influence of lifestyle factors on all degenerative diseases, my sole interest in this study is to examine the prevalence of the lifestyle factors that can increase the risk of coronary heart disease, which is one of the primary causes of death among adult Aborigines. In this case they are seen as being biological and behavioural risk factors of coronary heart disease which are influenced by demographic, environmental, and social-cultural conditions (risk markers). At a macro-level, I compare the contemporary mortality patterns of and risk factor prevalence levels among Australian Aborigines and other indigenous groups. At a micro-level, I make the same comparisons between Aborigines living in western New South Wales and other Australians; more specifically, I explore the

interrelationships between several behavioural and biological risk factors of coronary heart disease — including alcohol and tobacco consumption, blood pressure, body composition, serum cholesterol, diet, and physical activity — and the influence of various risk markers on these risk factors. At both levels of this inquiry I am asking: (1) how do the mortality patterns of Australian Aborigines compare with those of other indigenous groups or the general Australian population? and (2) is the high rate of coronary heart disease among Australian Aborigines attributable to the underlying prevalence of specific lifestyle factors?

1.2 The circulatory system and heart disease

The human heart is part of a vast network of arteries and veins which make up the circulatory system. Degenerative heart diseases, such as coronary heart disease, are a product of the deterioration of the total system. In this review the aetiology of coronary heart disease and its associated risk factors will be described. The purpose here is to review the evidence for all the appropriate risk factors and their synergistic nature.

1.2.1 Coronary heart disease

Coronary heart disease (synonymous with ischaemic heart disease) is an acute or chronic cardiac disability arising from a reduction or arrest of the blood supply to the heart muscle (World Health Organization, 1959: 12). The major clinical manifestations of coronary heart disease are angina pectoris, myocardial infarction, and sudden death (Davidson *et al.*, 1979). Angina pectoris is a temporary and often severe chest pain associated with a relative shortage of oxygen in the heart muscle and is most frequently due to a narrowing or an obstruction of at least one of the coronary arteries. Myocardial infarction is a blockage of the blood supply to and necrosis (death) of part of the heart muscle. The occlusion of the coronary artery during a myocardial infarction is often due to the clotting of blood (thrombosis) in a damaged or narrowed artery. Sudden deaths usually occur in people who have had angina pectoris or a myocardial infarction. In all major forms of coronary heart disease there is oxygen insufficiency arising from

impaired coronary arterial blood flow. This impairment is often referred to as coronary heart disease and is very often preceded by or associated with the effects of atherosclerosis.

Atherosclerosis, the most important of the degenerative diseases of the arteries, is the progressive growth of smooth muscle lipid deposits and connective tissues into the intimal layer of the arteries and their development into lesions that eventually narrow the arteries (Ross and Glomset, 1976). Three major forms of atherosclerotic disease arise from the involvement of the coronary arteries (coronary heart disease), the cerebral arteries (cerebrovascular disease), and the femoral artery and its branches (intermittent claudication). Other arteries, like the mesenteric or renal, can be affected with possible consequences of disease in the organ supplied (Davidson *et al.*, 1979).

1.2.2 Antecedents of coronary heart disease

Epidemiological studies have revealed a great variability in the resistance of individuals and populations to heart disease. This variability is not a matter of chance; on the contrary, it seems to reflect the importance of some underlying factors in the aetiology of coronary heart disease. These characteristics can be classified into three main groups: biological risk factors, behavioural risk factors, and risk markers (see Péron and Strohmenger, 1985: 93).

1.2.2.1 Biological risk factors

Biological risk factors are biological abnormalities detectable before the appearance of the first symptoms or clinical problems characteristic of the disease. In this case the precursors of ischaemic heart disease are seen as being diabetes mellitus (diabetes), genetic susceptibility, hypertension, obesity, and elevated serum cholesterol.

Diabetes has long been recognized as a major predisposing factor in atherosclerosis of both the coronary arteries and the larger vessels. The condition occurs in two forms: as an insulin deficient diabetes mellitus in young persons; or as an insulin resistant form

(non-insulin dependent diabetes mellitus, or type 2 diabetes) in adults, which is usually associated with obesity and a family history of the disease (Hetzel and McMichael, 1989). The concern in this review is with the latter type of diabetes.

Despite the use of a wide range of methodologies and criteria, epidemiological studies have shown that non-insulin dependent diabetes mellitus has a global distribution. Investigations into the aetiology of this disease have shown that there appears to be a spectrum of genetic and environmental factors which make certain groups more susceptible than others (Zimmet, 1982). Important environmental factors include age, obesity, physical inactivity, diet, socio-economic status, and parity (among women).

Studies of diabetes mortality in developed countries are unanimous in finding that circulatory system diseases, in particular ischaemic heart and cerebrovascular disease, are the major causes of death for individuals with diabetes (Fuller, 1985). In a multiple cause of death analysis for England and Wales, circulatory system diseases were the underlying cause of death in just over 50 per cent of the death certificates mentioning diabetes. Several studies have also shown that diabetics tend to have a coronary heart disease mortality rate two to three times that of the general population (Pell and d'Alonzo, 1970; Kessler, 1971; Garcia *et al.*, 1974).

The risk factors of coronary heart and cerebrovascular disease appear to be the same for non-insulin dependent diabetics and non-diabetics. Jarrett (1985) argues that the major reason for the higher prevalence of coronary heart disease among diabetics is that the two risks — coronary heart disease and non-insulin dependent diabetes mellitus — often coincide in the same individual.

Genetic factors, like male and female sex hormones, may contribute to the notable differences which exist in the rates of ischaemic heart disease among men and women. Current research suggests that endogenous female sex hormones produced by the ovaries may decrease the risk of coronary heart disease. In contrast, present epidemiological evidence about the male sex hormone testosterone suggests that it does

not have any significant effect on the prevalence of coronary heart disease (Waldron, 1983).

Hypertension is defined by the World Health Organization and the International Society of Hypertension (1986) as a phase-five diastolic blood pressure of 90 mm Hg or more on at least three consecutive and separate occasions. Patients with high blood pressure are usually assigned to one of two categories: either essential or primary hypertension, which are elevations in blood pressure due to unknown causes; or secondary hypertension, which are elevations associated with other diseases. In developed countries, like the United States, about 90 per cent of all blood pressure elevations are classified as essential hypertension and only 10 per cent are classified as secondary hypertension (McGarvey, 1980; Eaton *et al.*, 1988).

Several researchers have argued that the distinction between normotensive and hypertensive is arbitrary and that the basic problem is that blood pressures tend to rise with age. Morbidity data from the Framingham study (Kannel and Gordon, 1970) have confirmed that there is indeed no dividing line. The risks of cerebrovascular and ischaemic heart disease, the two main complications of hypertension, rise steadily with every increment of diastolic blood pressure above 70 mm Hg. When these Framingham study morbidity rates are applied to the distribution of blood pressures among Australian men aged 45 to 64 years, almost half of the myocardial infarctions that are attributable to diastolic blood pressure occur at levels of less than 90 mm Hg — in subjects who are not considered hypertensive by the current World Health Organization definition. Moreover, well over half of these myocardial infarctions can be expected at diastolic blood pressures of less than 100 mm Hg where there is no agreed need for medication (Beard and Heller, 1987).

Although this rise in blood pressure with age invariably occurs in salt-consuming populations, this trend is by no means universal. Among the Yanomamo, where salt is not consumed, blood pressures increase from the first to the second decade of life but,

in contrast to salt-consuming societies, do not systematically increase during subsequent years of life; and diastolic blood pressures for either sex remain on average below 70 mm Hg (Oliver *et al.*, 1975). This and other normotensive cultures, which exist in varied climatic circumstances, share a number of common characteristics, like diets low in sodium and high in potassium (Eaton *et al.*, 1988). However, even in salt-consuming populations, longitudinal studies, like the thousand aviator cohort study, have shown that blood pressures can remain relatively unchanged in a large proportion of men and women over a long period (Oberman *et al.*, 1967).

The occurrence of hypertension in most cases probably reflects the interaction between individual genetic predisposition and pertinent modifiable lifestyle characteristics, such as salt intake (Eaton *et al.*, 1988). Other important factors most probably related to hypertension are obesity and stress (McGarvey, 1980).

Obesity arises when the intake of energy exceeds the energy expended to the extent that there is a marked increase in the amount of fat present in the body. In theory this energy imbalance leading to obesity might occur over a relatively short period of time because of gross excesses of intake, or it might arise very gradually (Durnin, 1988). However, there is no one cause of obesity; rather, its underlying causes — genetic, neurochemical, and psychological — interact in a complex fashion to influence body energy regulation (Eaton *et al.*, 1988).

The linking of obesity to imbalance in diet or to the overconsumption of a particular food remains tenuous. With regard to actual sugar intake, Durnin (1988) suggests that there is little direct evidence that the diet of obese individuals is rich in sweetened foods. On the contrary, available evidence on nutrient intake and food preference studies from different populations has repeatedly shown that sugar appears to play no important role in obesity.

The association between physical inactivity and obesity seems stronger, suggesting that energy output, not energy intake, is what is in imbalance. Total food energy intake

actually has an inverse correlation with fat production, but obese persons have proportionately even lower levels of energy expenditure. Increased levels of physical exercise raise expenditure proportionately more than caloric intake and may lower body weight (Eaton *et al.*, 1988).

Obesity appears to be a relatively weak risk factor for heart disease, especially when separated from diabetes, hypertension, and hyperlipidaemia — the presence of abnormally high concentrations of fats in the blood (Davidson *et al.*, 1979). However, there have been several major prospective studies that have observed an association between obesity and coronary heart disease. The Framingham study demonstrated that the incidence of coronary heart disease, the death rate from coronary artery disease, and the likelihood of developing heart failure can be predicted from the initial degree of overweight — a 20 per cent reduction in weight in an obese individual would result in a 40 per cent reduction in his or her chances of developing and dying from coronary heart disease (Kannel and Gordon, 1974). A second prospective study conducted by the American Cancer Society between 1959 and 1972 on one million volunteers showed that as body mass index increased so did deaths from heart disease. In fact, for individuals who were overweight or obese, the relationship between body mass index and relative mortality from heart disease was almost linear (Lew and Garfinkel, 1979).

In summary, although obesity as an independent variable is a weak and inconsistent predictor of coronary heart disease, it predisposes an individual to the development of hypertension and diabetes, and to a lesser extent it is associated with hyperlipidaemia. Directly, obesity increases the work load of the heart, interferes with the functioning of organs, and reduces physical fitness (Department of Health and Social Security Medical Research Council, 1976; Davidson *et al.*, 1979; Bray, 1985).

Cholesterol is a fat-like material (sterol) which is present in the blood and most tissues. Cholesterol levels in the blood (serum cholesterol) have been highly positively correlated with heart disease. More specifically, the relationship between cholesterol

and coronary heart disease is very much linked to the lipoproteins that transport cholesterol through the blood. Lipoproteins transport cholesterol either from the liver to the tissues (high-density lipoprotein) or from the tissues to the liver (low-density lipoprotein). High-density lipoproteins carry about 70 per cent of the serum cholesterol, while low-density lipoproteins carry about 15 per cent. Larger fat molecules, like triglycerides, are carried by very low-density lipoproteins and chylomicrons (Hetzel and McMichael, 1989).

Elevated serum cholesterol and low-density lipoprotein levels are positively correlated with the prevalence of coronary heart disease (Barr *et al.*, 1951; Kannel *et al.*, 1971), and a strong inverse relationship exists between coronary heart disease and high-density lipoproteins (Miller and Miller, 1975). Very low-density lipoproteins and triglycerides, once considered an independent risk factor (Böttiger and Carlson, 1980), are no longer thought to be valuable prognostic or discriminating factors (Avogaro, 1985).

The World Health Organization (1982) considers the elevation of total serum cholesterol on its own to be an important risk factor of heart disease, because total serum cholesterol levels are valuable indicators of the levels of low-density lipoproteins. Several notable prospective studies have also observed a similar relationship between total serum cholesterol levels and ischaemic heart disease. The Framingham study provided early evidence that individuals with high levels of serum cholesterol have an increased risk of developing coronary heart disease (Kannel and Gordon, 1970). Other studies, like the Seven Countries prospective study, also show that the risk of developing coronary heart disease rises step-wise with total serum cholesterol levels (Keys, 1970).

1.2.2.2 Behavioural risk factors

Behavioural risk factors are characteristics which, as they appear before the onset of the disease, can be controlled by clinical, epidemiological, and other types of intervention. Heavy alcohol and tobacco consumption, a diet high in fat intake, and physical

inactivity are considered to be important behavioural risk factors of ischaemic heart disease.

A substantial body of epidemiological evidence indicates that moderate alcohol consumption (no more than two drinks¹ per day) is associated with a lowered risk of coronary heart disease mortality, but that higher levels of consumption predispose an individual to hypertension, arrhythmia, cardiomyopathy, and at some point more cardiovascular mortality (Criqui, 1985). Thus, alcohol consumption is associated with the incidence of coronary heart disease in both beneficial and detrimental ways.

Moderate alcohol consumption may have a dual protective effect: it raises the concentration of high-density lipoproteins in the blood, which appear to protect against heart disease; at the same time it lowers the concentration of low-density lipoproteins, the atherogenic fraction (Criqui, 1985). Furthermore, moderate alcohol consumption can reduce blood clotting through diminished platelet aggregations or clumping, which could be an important preventive agent against heart disease (Hetzl and McMichael, 1989).

Heavy alcohol consumption can affect the heart both directly and indirectly. Alcohol cardiomyopathy, a chronic disorder affecting the muscles of the heart, is common among chronic alcoholics (Urbano-Marquez *et al.*, 1989). Alcohol can also stimulate arrhythmia, even in the apparent absence of underlying heart disease, by altering conduction velocity (Gould *et al.*, 1978) and action potential duration (Fisher and Favaler, 1975). The potential for life-threatening arrhythmia and sudden death exists even after one bout of heavy drinking (Criqui, 1985).

Alcohol consumption has also been linked to high blood pressure, which can indirectly affect the heart. Many epidemiological studies show that moderate and heavy drinkers have higher blood pressures than non-drinkers, and that abstinence is followed by a fall

1. One drink is equal to approximately 15 ml of ethanol.

in blood pressure. An increase in blood pressure can be shown in persons having three to five drinks per day (Hetzel and McMichael, 1989). In one study subjects drinking two drinks per day had blood pressures similar to non-drinkers, but beginning at three drinks per day blood pressures were increasingly higher with each increased level of alcohol consumption (Klatsky *et al.*, 1977).

There are at least four factors influencing the impact of smoking on the heart (Epstein and Jennings, 1986). First, smokers are at risk of getting coronary heart disease at younger ages than non-smokers. Second, a high dose of cigarette smoke, measured in terms of the number of cigarettes smoked per day or inhalation patterns, is related to an increased risk of heart disease. Third, the age at which a person started smoking also influences the risk of coronary heart disease. Fourth, male smokers have a higher risk of heart disease than female smokers, and among female smokers, those who use oral contraceptives are much more likely to have a myocardial infarction than those who do not.

Investigators in the United States have estimated that approximately 30 per cent of coronary heart disease is attributable to cigarette smoking (Epstein and Jennings, 1986). Three main clinical manifestations of coronary heart disease have been related to smoking: myocardial infarction (fatal or not); other death from coronary heart disease (sudden or not); and angina pectoris.

Smoking also interacts synergistically with other risk factors to influence the prevalence of coronary heart disease. Smokers with elevated blood pressure or serum cholesterol levels have a threefold increase in the risk of developing coronary heart disease compared to those with normal blood pressure or serum cholesterol levels; and those with both high blood pressure and serum cholesterol levels have an eightfold increase in the risk of developing coronary heart disease (Epstein and Jennings, 1986). Smokers with diabetes may also have an elevated risk of developing heart disease. In California, a recent nine-year follow-up study of adults aged 60 to 79 years observed that an

estimated 65 per cent of deaths from cardiovascular diseases among diabetics could be attributed to the interaction between diabetes and smoking (Suarez and Barrett-Conner, 1984); however, most prospective studies have not reported a greater impact of any cardiovascular disease risk factors in diabetics than in non-diabetics (Jarrett, 1985). Additionally, smoking is not positively related to hypertension, as the blood pressures of smokers tend to be lower than those of non-smokers.

In regard to dietary risk factors, a very large body of research has been devoted to the role of fat in the development of atherosclerosis and coronary heart disease. The role of other components of the diet such as carbohydrates, dietary fibre, and some micro-nutrients (salt, potassium, and calcium) has also received attention. Overall, coronary heart disease is most likely associated with more than one dietary factor, but so far the best established component is fat (Hetzel and McMichael, 1989).

For the last 30 to 40 years the diet-heart hypothesis has dominated research about the dietary components of coronary heart disease (Kromhout, 1985). First proposed by Keys (1952), the hypothesis states that saturated fats elevate serum cholesterol levels and persons with elevated serum cholesterol levels are at a high risk of coronary heart disease. Proof for this hypothesis mainly comes from the comparison of saturated fat intakes and median cholesterol levels between countries. For example, the Seven Countries prospective study clearly showed that the ten-year incidence of coronary heart disease in 13 communities was strongly correlated with the dietary intake of saturated fat (Keys, 1970). Correlations were also found between the dietary intake of saturated fats and serum cholesterol levels, and between serum cholesterol levels and coronary heart disease.

However, intra-population studies have not shown this consistent a relationship between the dietary intake of saturated fats and serum cholesterol levels (Nichols *et al.*, 1976; Garcia-Palmieri *et al.*, 1977; Kay *et al.*, 1980; Shekelle *et al.*, 1981). Kromhout (1985) suggests that these zero or low order correlations can be explained by large intra-

individual variations in both fatty acid intakes and serum cholesterol levels. It has been suggested that in order to obtain more accurate information about the saturated fat intakes of individuals, at least twenty-two 24-hour dietary intakes randomly scattered throughout the year are needed (Balough *et al.*, 1971).

On the other hand, metabolic studies conducted under controlled conditions have shown a relationship between various types of fat intakes and serum cholesterol levels. In one such study Keys and Parlin (1966) observed that the serum cholesterol-increasing power of saturated fat is twice as strong as the serum cholesterol-decreasing power of polyunsaturated fat. More recently, saturated fat has been shown to increase total cholesterol and low-density lipoprotein concentrations, while polyunsaturated fat has been shown to lower total cholesterol and low- and high-density lipoprotein concentrations (Mattson and Grundy, 1985). In another study, the serum cholesterol-increasing power of dietary cholesterol was observed to be less than that of saturated fat (Keys *et al.*, 1965). This type of evidence suggests that the type of fat consumed is important in affecting serum lipoprotein concentrations and thrombotic tendency (Sanders, 1988).

Carbohydrates may also play a role in the aetiology of coronary heart disease. However, evidence linking sucrose intake to coronary heart disease is inconclusive. The Seven Countries prospective study correlated sucrose intake with coronary heart disease, but sucrose intake was also correlated with saturated fat intake (Keys, 1970). When the effects of saturated fat intake were controlled for, no significant relationship was found between sucrose intake and coronary heart disease. Additionally, there are some countries, like Mauritius and Venezuela, where sucrose intake is high and coronary heart disease mortality is low, as is the intake of saturated fat (Davidson *et al.*, 1979). Overall, most researchers believe that no direct link has been established between sucrose intake and coronary heart disease.

By contrast, the epidemiological data suggest that starchy foods have a definite beneficial effect on the risk of coronary heart disease. Almost without exception recent prospective studies have shown that starch intake was lower among men who died from heart disease than among survivors (Kromhout, 1985). The intake of dietary fibre, which is often found in starchy foods, also seems to be inversely related to coronary heart disease (Morris *et al.*, 1977; Kromhout *et al.*, 1982).

Micro-nutrients may also play a role in the development of coronary heart disease, especially in the regulation of blood pressure. A comparative study of salt consumption in different populations has demonstrated that a linear relationship exists between sodium intake and blood pressure — often referred to as the salt-blood pressure line (National Health and Medical Research Council, 1984). Within populations the intake of sodium is generally unrelated to blood pressure, probably because of large day-to-day intra-individual variation in both sodium intake and blood pressure.

Potassium also seems to play a role in the regulation of blood pressure. Two recent intervention studies have shown that increasing potassium intake significantly lowers blood pressure (Khaw and Thom, 1982) and may protect an individual from cerebrovascular disease (Khaw and Barrett-Conner, 1987). The Evans County study has also noted a similar inverse relationship between potassium intake and blood pressure (Grim *et al.*, 1980).

An inverse relationship also seems to exist between calcium intake and blood pressure. A recent case-control study observed that the calcium intake of hypertensive subjects was significantly lower than in normotensive subjects (McCarron *et al.*, 1982). Although such studies show promising results, most claims of the association between calcium intake and blood pressure remain unsubstantiated (MacGregor, 1988).

Substantial epidemiological evidence suggests that people who exercise or who are physically active are less likely to die from ischaemic heart disease than inactive people. Regular physical activity or exercise seems also to be associated with higher

levels of fitness and with a more favourable risk factor profile for coronary heart disease — it lowers blood pressure and the heart rate, and inhibits the development of atherosclerotic plaques and coronary-artery thrombosis (Ekelund *et al.*, 1988).

These studies have often been unclear as to whether the lower rate of heart disease in active people is a direct benefit of exercise or a secondary effect of genetic factors — the consequence of fitter people weighing less and having lower blood pressure and better cholesterol profiles (Sobolski *et al.*, 1987). This research dilemma exists because most of the studies on which the direct relationship between heart disease rates and exercise are based have had difficulty: quantifying levels of activity usually performed; determining the extent to which sedentary lifestyle is self-selected; and monitoring changes in fitness as well as activity levels and other lifestyle factors (Dimsdale *et al.*, 1986).

However, a recent study puts the case for a direct benefit on a much firmer standing (Ekelund *et al.*, 1988). The study, which was carried out on 3,106 adult male Americans who showed no clinical evidence of coronary heart disease, demonstrated that a lower level of physical fitness, as measured from a standard treadmill exercise test, was associated with a higher risk of subsequent death from coronary heart disease. The risk of dying from a heart attack was about three times greater for the least-fit fourth than for the fittest fourth even after adjusting for smoking, blood pressure, and cholesterol. The fitness level of these men was closely related to regular physical activity as reported by the participants. These findings suggest that regular physical exercise is important in determining the level of fitness and therefore might be an important factor in reducing the risk of death from coronary heart disease. Although conclusive proof is probably elusive, this study does suggest the importance of physical activity in maintaining cardiovascular health.

1.2.2.3 Risk markers

Risk markers are characteristics of individuals or their environments which are uncontrollable or can only be controlled with difficulty. In this study age, ethnicity, gender, marital status, socio-economic status, and stress are considered to be risk markers of coronary heart disease. These risk markers are sources of potential variation in the prevalence of biological and behavioural risk factors.

Death rates from coronary heart disease rise with age in both males and females. Although most of these deaths occur in middle adulthood or later, the underlying atherosclerotic process may start earlier in life (World Health Organization, 1982). Autopsy studies in various countries worldwide have shown that the atherosclerotic process appears to begin in childhood and adolescence, and severe atherosclerotic lesions may even be present in young adults (Strong, 1972).

There are also significant ethnic-related differences in the prevalence of coronary heart disease. Studies in Australia have observed that almost all ethnic or birthplace groups exhibit lower mortality from ischaemic heart disease than does the Australian-born group (see Young, 1986a, 1986b). Only males born in Oceania (excluding Australia and New Zealand), Poland, and South Asia and females born in Malta, Poland and South Asia had relatively higher levels of coronary heart disease mortality (Young 1986a). Other studies by Stenhouse and McCall (1970), Dasvarma (1980), and Dunt (1982) have found similar patterns. Dasvarma (1980) has also observed that the coronary heart disease mortality rates of immigrants tend to approach Australian rates with increasing periods of residence.

The prevalence of ischaemic heart disease may be influenced by social stability as well as ethnicity. A study of coronary heart disease in two adjacent but ethnically distinct communities in Pennsylvania showed that the more homogeneous and stable of the two communities had a lower rate of deaths from myocardial infarction (Bruhn *et al.*, 1966). Additionally, studies of men of Japanese ancestry have revealed a gradient in the

occurrence of coronary heart disease which is not completely explained by underlying risk factors (Marmot and Syme, 1976; Yano *et al.*, 1984). Heart disease occurrence was lowest in Japan, intermediate in Hawaii, and highest in California. When Japanese men in California were classified according to the degree to which they adhered to traditional Japanese values, the most traditional group of Japanese-Americans had a prevalence of coronary heart disease as low as that observed in Japan (Marmot and Syme, 1976).

Gender is also an important risk marker. Death rates from ischaemic heart disease have been consistently higher for men in virtually all available international and historical sources of mortality data (Waldron, 1983). Epidemiological studies, like the Framingham study, have revealed that coronary heart disease occurs 10 to 20 years later in women than in men (Dawber, 1980). Among older women, ischaemic heart disease seems not only to be related to age but also to be influenced by the cessation of menstruation (Kannel *et al.*, 1976). When recognized risk factors are taken into account — systolic blood pressure, cigarette smoking, serum cholesterol, relative weight, and glucose intolerance — men still have a two to three times greater risk of developing coronary heart disease than women (Truett *et al.*, 1967).

Married people tend to have better health than those who are not married. Generally, they have fewer chronic disabilities, fewer restricted activity and bed days, better self-rated health, and fewer acute conditions; an understandable exception is acute conditions related to pregnancy and reproduction among married women (Verbrugge, 1983). Evidence also suggests that married people have a lower rate of heart disease. One study showed that among American men younger than age 65, the mortality rate from coronary heart disease for those unmarried was about twice that of those who were married (Lynch, 1977). Chandra and associates (1983) have demonstrated that among survivors of hospital admissions for acute myocardial infarction, the subsequent mortality rate over a ten-year follow-up period was significantly greater among those

who were unmarried — single, divorced, separated, and widowed — than those who were married.

In mortality or lifestyle studies, education, occupation, and income are generally recognized as being the three primary components of socio-economic status. Of the three, education has the advantage for research purposes of usually being fixed after young adulthood (Kitagawa and Hauser, 1973). An inverse relationship is often found when researchers examine the association between education and coronary heart disease. A Canadian study based on a series of national cross-sectional surveys of men and women aged 20 to 69 years showed that the prevalence of cardiovascular risk factors tended to be higher among men and women with only an elementary education (Millar and Wingle, 1986). The largest relative differences in risk factor prevalence levels between the education groups — elementary, secondary, and tertiary — were found for smoking, obesity, physical inactivity, and excessive alcohol consumption.

Kitagawa and Hauser (1973), in their analysis of mortality differentials in the United States by years of schooling, observed that among white Americans deaths from atherosclerosis and degenerative heart diseases were generally inversely related to education. For the least educated females aged 25 to 64 years mortality from these causes was 2.4 times that of the best educated; while for the least educated males in the same age group mortality from these causes was 1.2 to 1.3 times that of the best educated. Among men aged 65 years and over, this inverse pattern tended to disappear, whereas for older women the inverse pattern persisted. Another study conducted in the United States by Weinblatt and colleagues (1978), which examined the relationship between education and sudden death after a myocardial infarction, demonstrated that male survivors with eight years of schooling or less had a threefold greater risk of sudden death than males with more education.

Although available evidence is largely indirect, stress may also have an influence on coronary heart disease. Matthews (1986), in her conclusion to an edited volume on

stress, reactivity, and cardiovascular disease, cited three main sources of evidence: animal, case-control, and behavioural studies. First, data from studies of Rhesus monkeys have shown that monkeys who exhibit elevated heart rates in response to a common laboratory stressor have upon their subsequent death almost twice the coronary artery atherosclerosis of their lower heart rate reactive counterparts. Second, case-control studies have demonstrated a heightened reactivity to laboratory stressors in patients with histories of angina or previous infarction, compared with non-coronary patients or non-patient controls. Third, the majority of published studies on 'Type A' (a personality type characterized by tenseness, drive, and aggression — traits thought to make one more susceptible to heart disease) and 'Type B' (a personality type characterized by serenity, relaxation, and amiability — traits thought to make one less susceptible to heart disease) behaviours show that 'Type A' men exhibit larger increases in blood pressure, heart rate, and plasma catecholamines and/or cortisol than 'Type B' men when they are exposed to appropriately stressful tasks.

1.3 Thesis outline

As I have already stated, this study attempts to evaluate whether Aboriginal mortality patterns are different from those exhibited by indigenous groups in other countries or by the general Australian population; and whether the high rate of coronary heart disease among Australian Aborigines is attributable to the underlying prevalence of specific lifestyle (risk) factors.

Chapter 2, a literature review, describes Aboriginal mortality patterns and compares them with those exhibited by other indigenous groups — American and Canadian Indians, New Zealand Maoris, and Pacific Islanders; and examines the prevalence of biological and behavioural risk factors of coronary heart disease among Australian Aborigines and these other indigenous groups.

Chapter 3 describes the contemporary environmental and social-cultural conditions of Aborigines living in western New South Wales. In particular, the chapter examines

aspects of Aboriginal identity, family and household formation, employment opportunities, labour force participation and occupation, and income levels.

The next four chapters are mainly analytical. Chapter 4 concentrates on evaluating contemporary Aboriginal mortality in western New South Wales. Chapter 5 summarizes the methods used to examine the prevalence of the risk factors of coronary heart disease as well as the indicators of overall and heart health among Bourke Aborigines. The following two chapters look at the prevalence of and association between various risk factors of coronary heart disease among Bourke Aborigines. Chapter 6 evaluates biological and behavioural risk factors, while chapter 7 looks at dietary risk factors.

Finally, chapter 8 discusses how the evidence presented in this thesis answers the questions I have asked. Additionally, I discuss some of the major limitations to my work, its implications for Aboriginal health care policy, and some avenues for future mortality research.

CHAPTER TWO

INDIGENOUS MORTALITY AND CORONARY HEART DISEASE

2.1 Introduction and overview

The numbers and health of indigenous people living in what is now Australia, Canada, New Zealand, the Pacific Islands, and the United States rapidly deteriorated after Anglo-European contact. Historically, initial depopulation and the high levels of adult mortality which persisted long after contact were mainly due to infectious diseases; presently, however, the varying high levels of adult mortality found in these populations are mainly due to degenerative diseases and accidental and violent events. The purpose here is not to examine the historical patterns of mortality in these indigenous populations, which have already been quite extensively reviewed, but to compare the underlying nature of some of these contemporary mortality regimes accompanied by a review of coronary heart disease risk factor prevalence levels.

Few comparative studies of contemporary indigenous mortality regimes have been conducted. Moodie's (1973) comparison of the mortality patterns of Northern Territory Australian Aborigines in 1964 and 1965 with those of New Zealand Maoris in 1964 and American Indians from 1961 to 1963 is one of the first more thorough investigations of this kind. More recently, Kenen (1987) has compared contemporary Australian Aboriginal and American Indian mortality patterns; Kunitz (1990a) has remarked on the similarities and differences between the mortality experiences of Australian Aborigines, American and Canadian Indians, and New Zealand Maoris; and Gray (1990a) has briefly commented on similarities in Australian Aboriginal and Canadian Indian age-specific mortality levels. In the Pacific, Taylor and colleagues (1989) have examined the mortality transition among various indigenous Pacific Island populations.

In this chapter I attempt to complement this existing body of research by comparing the mortality patterns of Australian Aborigines, in various regions, with those exhibited by Canadian Indians and New Zealand Maoris. Additionally, where information has been available Australian Aboriginal mortality rates are compared with those for American Indians and the indigenous Pacific Island population of Nauru. As noted above, the aim here is to outline how the mortality patterns of Australian Aborigines, in particular deaths from heart disease, differ from other indigenous populations; and to highlight differences in the prevalence of various risk factors of coronary heart disease.

2.2 Indigenous mortality data

Vital event data on Australian Aborigines, American and Canadian Indians, New Zealand Maoris, and other Pacific Islanders are in many cases unavailable or incomplete. The data which are available reflect the idiosyncratic manner in which each country classifies deaths and the persons at risk of dying — the way in which deaths are reported is frequently different from the way the population at risk of dying is defined. In New Zealand, for example, the ethnic classification of deaths is based on the biological origin of the parents of the deceased, while estimates of the population at risk of death are based on census counts of people who actually identify as being Maori (see section 2.2.4 for further details). Furthermore, in some countries sources of data only refer to a subgroup, like those American Indians who live in Reservation states or those Canadian Indians who are Registered. For all these reasons mortality data can often be inaccurate which makes estimating indigenous mortality within a country and comparisons of mortality patterns between countries difficult.

2.2.1 Australia

In Australia, there has been a long history of attempts to obtain better Aboriginal health statistics through the identification of Aborigines on the administrative forms which are used to produce statistics about the total population (Gray, 1986: 11). However, progress has been very slow. Information on Aboriginal deaths has been available from

official death registration systems only since 1980, when in New South Wales a question of Aboriginality was first included as a data item on official death notification forms (Smith *et al.*, 1983). During the mid-1980s, after being urged by the Task Force on Aboriginal Health Statistics (1985), most states and both territories have been moving towards identifying Aboriginal deaths in their official collections (Thomson, 1986; Gray, 1990b).

Although these systems are now in place throughout Australia, mortality studies conducted during the last ten years, based wholly or partly on listings of deaths supplied by state or territory registrars, have often noted that a large number of Aboriginal deaths are not identified (Gray, 1990b). For example, in New South Wales notifications of Aboriginal deaths in the registries have been found to severely underreport the total number of deaths (Gray, 1983; Smith *et al.*, 1983; Gray and Hogg, 1989). To compensate for this lack of identification, researchers have often relied on local registry office records and listings of Aboriginal deaths produced by other agencies, such as the Aboriginal Health Services. However, a small number of deaths may also be recorded interstate and are therefore not available for analysis (see Plant, 1988).

These mortality studies have also had difficulties estimating the population at risk of dying. While the Australian population census remains the only comprehensive source of data on the Aboriginal population, the census coverage of Aborigines is often not complete. The 1986 census counts implied a growth rate of 42 per cent for the Aboriginal population since the count was taken in 1981. While both censuses are generally free of any major inconsistencies, this very large intercensal growth rate in the Aboriginal population is probably due to the increased propensity of people to record themselves and their households as being Aboriginal (Australian Bureau of Statistics, 1989a).

2.2.2 United States

In the United States, states register their deaths on a standard form and then report their data to the National Centre for Health Statistics. These data include information about the deceased's age, sex, race or ethnicity, and place and cause of death. Records on individuals whose race or ethnicity is listed as American Indian or Alaskan Native are then sent annually to the Indian Health Service for Reservation Indians. Except for the individuals' names and former addresses these statistical records contain the same basic demographic information as the original records (Kenen and Hammerslough, 1987; U.S. Department of Health and Human Services, Public Health Service, Indian Health Service, 1988).

This information has been collected by the Indian Health Service for Reservation Indians since 1955. Reservation Indians are members of more than 500 recognized Indian tribes who live primarily on Federal Indian reservations and in small rural communities. The states where these reservations and communities are located are considered Reservation states, and in 1987 the majority of them were located in the western half of the United States. Population counts of Reservation Indians are based on U.S. census counts of individuals who identify themselves as being American Indian, Eskimo or Aleut and who reside in geographic areas for which the Indian Health Service has jurisdiction (U.S. Department of Health and Human Services, Public Health Service, Indian Health Service, n.d., 1988).

Census counts of American Indians also suffer from problems of inconsistency. Passel and Berman (1986) estimate that the 1980 census count of American Indians represents more than a 70 per cent increase over the 1970 census count. Increases occurred in most states, but the amount varied considerably, especially in non-Reservation states. These differences are for the most part outside expected natural increases and sample variability, and suggest that like Australian Aborigines there is an increasing propensity for people in the United States to record themselves as being American Indian.

2.2.3 Canada

As in the case of American Indians, mortality statistics are generally only available for a select group of Canadian Aborigines — Registered or Status Indians. Other groups, such as Metis and non-status Indians, are not recognizable in the statistics as distinct groups. Registered Indians form an unnatural, legally defined group which before 1985 gained members by marriage as well as birth, and lost members by marriage, enfranchisement¹, and death (Duff, 1965: 47). In 1985, amendments to the Indian Act repealed those sections which offended the equality provisions of the Canadian Charter of Rights and Freedoms. Because of these amendments many people who were not recognized by the Indian Act (for example, women who had married non-Indians, ex-nuptial children, families of men who had lost their status, foreign residents, and professionals) regained their Registered Indian status (see Woodward, 1989: 19-26). Since the amendments to the Act took effect, the annual growth rate of the Registered Indian population has more than doubled — from 3.0 in 1982-1983 to 7.2 in 1986-1987 (Indian and Northern Affairs Canada, 1988: 14). In 1986, the total Registered Indian population was approximately 387,000; about 75 per cent of this population was directly served by the Medical Service Branch.

The Medical Service Branch's administrative and public health program statistics constitute the main source of the numerator and denominator data for the generation of crude and age-specific death rates. The quality of the data varies markedly from one region to another depending on the context in which the data are collected and reported (Indian and Inuit Health Services, Medical Services Branch, 1985: 122). In terms of statistical reporting the regions may be broadly divided into two categories: regions which use the provincial or territorial health care services to collect data (Alberta, Saskatchewan, Manitoba, Yukon, Northwest Territories and previously, British Columbia); and regions which rely on Medical Service Branch personnel to collect data

1. The Indian Act of 1876 set forth a process of enfranchisement whereby Indians could acquire full Canadian citizenship by severing their ties to the native community (Pointing and Gibbons, 1980: 10).

(Ontario, Quebec, and the Atlantic provinces). Provincial or territorial health care services gather data on the entire Registered Indian population while Medical Services Branch personnel collect data only on the Registered Indians they serve (Health and Welfare Canada, 1988: 38). Both systems suffer from serious drawbacks; therefore, inter-regional comparisons of health status indicators are difficult to make.

2.2.4 New Zealand and the Pacific

In New Zealand, the National Health Statistics Centre is the main source of Maori mortality data. However, the validity of these mortality statistics has in the past been questioned for two main reasons (Graham *et al.*, 1989: 124-125). First, information on the population at risk of death and the number of deaths in a given year come from two different sources. For any given year, estimates of the population at risk are based on census counts, while estimates of the total number of deaths are based on information extracted from death registration forms. Second, the death registration data themselves may be unreliable, because of the process by which ethnicity is documented. The main problem is not that Maoris are being misclassified as other nationalities, but that ethnic information is not obtained for all deaths. Ethnic classification on death registration forms is based on the biological origin of the parents of the deceased as reported by a close relative. A deceased person is classified as Maori if the average of the fractions of 'Maori blood' of his or her parents is greater than or equal to one-half. The degree of Maori blood of the deceased parents is recorded by the funeral director in the 'ethnic origin' section of the death certificate. Unless a funeral director suspects that the deceased was of Maori descent and is willing to pursue the question of ethnic origin with a close relative, the ethnicity section is left blank. All blank responses are coded as 'other' — non-Maori or non-Pacific Islander.

One recent validity check of registry data compiled by the National Health Statistics Centre on Maori deaths from coronary heart disease noted serious underreporting (Graham *et al.*, 1989). The study discovered that during the 1983-1984 period, Maori

deaths in Auckland from coronary heart disease were underreported by over 80 per cent. There is no reason to believe that death registration form completion practices would be any different for other causes of death. Even with possible regional and cause-specific differences in the degree of underreporting, findings from this study suggest that the overall reporting of Maori deaths is gravely deficient.

In many Pacific countries vital event registration systems, if they exist, are often deficient. Cause of death data are frequently inaccurate and incomplete, and not disaggregated by age, sex, or ethnic group. In some countries cause of death information is reasonably extensive, while in others this type of data is only available for hospital deaths (Taylor *et al.*, 1989).

Although the overall effect of these peculiarities is impossible to measure, it is bound to play a role in distorting age- and cause-specific mortality patterns and the underlying similarities and differences in the mortality experiences of these indigenous populations. Even still attempting to make this type of comparison is a worthwhile task because so little is known about how Australian Aboriginal mortality levels differ from those exhibited by other indigenous populations.

2.3 An overview of indigenous mortality patterns

As already stated, existing information on Australian Aboriginal, American and Canadian Indian, New Zealand Maori, and Pacific Islander mortality patterns is limited. Current research studies suggest that the indigenous people in these countries as a rule have high age-specific death rates, especially during adulthood, moderately low life expectancies, and high cause-specific death rates from circulatory and respiratory system diseases and accidental and violent events.

2.3.1 Australia

By virtually every health status measure Australian Aborigines are much worse off than other Australians (Thomson, 1990). To begin, their life expectancy is much lower.

National figures indicate that during the 1981-1986 period they had a life expectancy at birth of 56 years for men and 64 years for women (Gray, 1990b). When states and territories are compared, life expectancy at birth for both Aboriginal men and women is lowest in the Northern Territory and highest in Victoria and Tasmania. Gray (1990b) indicates that Aboriginal mortality is higher in geographical areas of Australia which contain proportionately more remote communities, but the differences are actually quite small. Overall, Aboriginal expectation of life is well below that of the total Australian population in 1986 of 73 years for men and 79 years for women (Australian Bureau of Statistics, 1987a).

Country-wide estimates of Aboriginal mortality have also been produced by combining 1985 cause-specific mortality data for Queensland, Western Australia, South Australia, and the Northern Territory (Honari, 1990). Diseases of the circulatory system were the leading cause of death for Aboriginal men and women in 1985. Death rates from these diseases, including ischaemic heart disease and other heart disease, were more than twice those of the total Australian population in 1985, with the greatest excess risk occurring between the ages of 25 and 44 years (Thomson, 1990: 7). Included in the category called injury and poisoning are deaths from motor vehicle accidents, suicide and self-inflicted injury, and homicide and injury purposefully inflicted by others; these were the second most frequent cause of death for males and the third most frequent for females. The death rates from these causes were about four times higher than the expected rates for the total Australian population. Diseases of the respiratory system, including both acute and chronic conditions, constitute the third most frequent cause of death for Aboriginal males and the second for Aboriginal females. Death rates from respiratory system diseases were six to eight times higher than the expected rates for the total Australian population.

The pattern of Aboriginal mortality found in the states and territories is very similar to the 1985 country-wide pattern. For example, circulatory system diseases are the primary cause of death among Aborigines living in New South Wales (Smith *et al.*,

1983; Gray and Hogg, 1989), the Northern Territory (Khalidi, 1989; Plant, 1988), Queensland (Lincoln *et al.*, 1983), South Australia (Divakaran-Brown and Honari, 1990) and Western Australia (Health Department of Western Australia, 1985; Hicks, 1990). However, there are some notable differences in inter-regional cardiovascular death rates. A comparison of the mortality rates of Aborigines living in 12 Queensland Aboriginal reserves between 1976 and 1980 and other Queenslanders in 1978 by Lincoln and others (1983: 358-360) showed that deaths from cardiovascular diseases were well over twice as frequent on Aboriginal reserves. Among Aborigines living on Bloomfield River, Cherbourg, Mornington Island, Palm Island, and Woorabinda reserves, the death rates from heart disease were three to four times higher than the comparable rates for Queenslanders. Reserves which had significantly higher rates of death were those which were close to an urban centre and which had historically been established as receivers of Aborigines forcibly removed from other places. These reserves also tended to have a majority of people of mixed racial descent living a less traditional lifestyle and a majority of dwellings serviced by sewerage or septic units (Trigger *et al.*, 1983: 362).

2.3.2 United States

In 1940, the life expectancy of American Indians born in that year was 51 years. By 1980 it had increased to 71 years, an increase of almost 40 per cent. In the same period life expectancy for the total United States population increased from 64 to 74 years, an increase of only 16 per cent (Rhoades *et al.*, 1987a: 356-357). As the American Indian life span approaches that of the general population, the disease pattern of the Indian Health Service population also more closely resembles that of other Americans. For example, age-specific rates of heart disease in the service area population tend to resemble those in the general population. For the 1983-1985 period, diseases of the heart were the leading cause of death for Indians (as well as for the general American population) followed for men by external causes and for women by malignant neoplasms (Rhoades *et al.*, 1987b; U.S. Department of Health and Human Services,

Public Health Service, Indian Health Service, 1988). Mortality rates have declined for Indians living both on and off reservations. A recent study observed that between 1970 and 1978 there was a substantial decrease in age-specific mortality, especially for the zero to four years age group. However, this downward trend was not uniform; it was not experienced by females aged 65 years and over and by males aged 5 to 14 years and 65 years and over living on reservations. Overall, the study found that the mortality rates of Reservation Indians were higher than those of non-Reservation Indians, even though both rates have declined (Kenen and Hammerslough, 1987).

2.3.3 Canada

Canadian Indian causes of death show a distinctly different pattern from those of the total Canadian population. During the 1982-1985 period, the life expectancy of Registered (status) Indians was 64 years for men and 73 years for women — compared with 72 years for men and 80 years for women in the total Canadian population. Although circulatory system diseases are the primary cause of death in both populations, death rates for Indians from this cause are usually lower than national averages. For the 1980-1984 period, the Indian age-standardized death rate for diseases of the circulatory system was slightly lower than the total Canadian rate — 3.1 compared with 3.2 per 1,000. The Inuit (Canadian Eskimos) have a much lower rate of heart disease. In 1983 they had an age-standardized rate from all cardiovascular diseases of 1.2 per 1,000, suggesting that they have a lower risk of heart disease than either Registered Indians or the total Canadian population (Muir, 1988: 18). Indians also have much lower rates of cancer than the total Canadian population. When indirectly standardized rates for the 1980-1984 period are compared, the five-year average death rate is 1.2 per 1,000 for Indians and 1.7 per 1,000 for the total Canadian population. Inuit again appear to have even lower rates of death from cancer than the other two populations — in 1983, the age-standardized rate for the Inuit population was 0.7 per 1,000 (Muir, 1988: 18).

The most significant difference between the mortality rates of Indians and Inuit and those experienced by Canadians as whole is that the former groups are three to four times more likely to die by accidental or violent means than the latter. During the 1977-1982 period, violent and accidental deaths accounted for one-half of all the deaths among residents of Indian reserves. Among males, one-sixth of these deaths were from intentional means — suicide or homicide (Mao *et al.*, 1986: 267). The high rate of Indian deaths from motor vehicle accidents has been attributed to alcohol abuse, poorly maintained roads and motor vehicles, and unqualified drivers; while fires which lead to death have likewise been attributed to alcohol abuse, poorly maintained and cleaned stove pipes, unsafe fire-fighting practices, and children playing with matches (Schmitt *et al.*, 1966; Jarvis and Boldt, 1980; Young, 1983).

The mortality rates for Indians are greater than the rates for the general Canadian population in all provinces or territories. The greatest differences in age-standardized rates are found from Quebec westward, with Alberta having the greatest margin of difference (from 11.5 per 1,000 for Indians to 6.5 per 1,000 for all Albertans) and Manitoba appearing as an exception to the general pattern. The Atlantic Region and the Yukon and Northwest Territories have the smallest degree of difference. For cardiovascular diseases, there tends to be less difference between Indian and regional Canadian rates, except in Ontario and the Northwest Territories where Indians experience higher rates (Health and Welfare Canada, 1988: 8-9).

2.3.4 New Zealand

In New Zealand, significant mortality differentials exist between Maoris and non-Maoris. Based on 1980-1982 mortality experiences, Maori males at birth could expect to live 64 years while females could expect to live 68 years — six and eight years less respectively than their non-Maori counterparts. Although Maoris had higher age-specific death rates than non-Maoris at almost all ages, the Maori pattern of death displayed the same broad pattern of variation with age as that of non-Maoris. The only

two exceptions were that the Maori population had a higher rate of death in the first year of life and a more pronounced 'accident hump' in the late teens and early twenties (Department of Statistics, 1986).

Coronary heart disease is the single most important cause of death among Maoris and non-Maoris. Of particular concern is the excessive number of deaths from coronary heart disease occurring in Maori women, especially those aged 25 to 44 years (Pomare and de Boer, 1988). Other forms of heart disease, especially chronic rheumatic fever and hypertensive heart disease, are also a major concern in the Maori population because of their disproportionate mortality burden. For example, the rates of death for chronic rheumatic fever and hypertensive heart disease are four to five times higher in Maoris than in non-Maoris (Pomare and de Boer, 1988). Cancer is also a leading cause of death. While cancers of the lung and breast are numerically the most important cancers in Maoris, in comparison with the non-Maori population there is a striking excess risk of death from cancers of the stomach and cervix. Environmental factors such as cigarette smoking, obesity, and sexually transmitted diseases are important risk factors of cancer but additionally a genetic contribution should not be discounted (Pomare and de Boer, 1988: 82). Accidents are also an important cause of death. Motor vehicle accidents are the leading cause of death for the 1 to 24 years age group in both the Maori and non-Maori populations. Deaths from suicide, especially among young men, have increased in the past two decades. Between 1970-1974 and 1980-1984 suicide rates for Maori men aged 15 to 24 years more than doubled (Pomare and de Boer, 1988).

Smith and Pearce (1984: 101) have noted several important determinants of the high death rates found among Maoris aged 15 to 64 years during the 1974-1978 period. Compared with non-Maori death rates, 20 per cent of the Maori male excess risk of death is attributable to marked ethnic differences in socio-economic status. Of the remaining excess risk, approximately 15 per cent is related to cigarette smoking, 10 per cent to alcohol consumption (excluding accidental causes of death), and 17 per cent to

accidents. When differences in age and social class are standardized, Maori men are five times more likely than non-Maoris to die from rheumatic fever, hypertensive heart disease, nephritis, diabetes, and some respiratory system disease conditions. Findings for Maori women are similar to those for men with the following exceptions: mortality from coronary heart and cerebrovascular diseases also contributed significantly to Maori excess risk, and differences attributable to socio-economic status could not be estimated from available data.

2.3.5 The Pacific

Several Pacific Island populations appear to have a pattern of high young adult mortality similar to that found among Australian Aborigines. Fifty per cent or more of the deaths in Fiji, most of Polynesia (American and Western Samoa, Tonga, the Cook Islands, and French Polynesia), and eastern Micronesia (Guam, Palau, and the Northern Marianas) are attributable to heart disease. In Guam, American Samoa, and the Cook Islands the predominance of cardiovascular and other non-communicable diseases is a manifestation of high life expectancy; while in other countries and ethnic groups non-communicable diseases, particularly among males, are a significant cause of premature death. Nauru has a very marked pattern of premature adult mortality, while Tuvalu, Fiji (especially in Indians), Western Samoa, Tonga, French Polynesia, the Marshall Islands, and most likely New Caledonia Melanesians have less marked regimes of high young adult mortality (Taylor *et al.*, 1989: 639-645).

On the island of Nauru the rapid transition from a traditional to a Westernized way of life has resulted in drastic social and cultural changes which have had major health implications. In terms of mortality, a recent analysis of adult (aged 15 to 64 years) deaths by cause between 1976 and 1981 indicates that circulatory system diseases were a major cause of death among both males (28 per cent) and females (10 per cent). Calculations of the years lost by adult Nauruans aged 15 years and over through

premature death indicate that circulatory system diseases account for 20 per cent of the years lost among males and 30 per cent among females (Taylor and Thoma, 1985: 151).

This brief review of recent indigenous mortality studies demonstrates that heart disease is a major cause of death for Australian Aborigines, American and Canadian Indians, New Zealand Maoris, and Nauruans. However, because of the nature of this enquiry little can be said about specific differences which seem to exist in indigenous mortality trends. In the next section, I have attempted to partially rectify this problem of incompatibility by comparing age- and cause-specific standardized rates of death.

2.4 Comparison of indigenous mortality patterns

Data on indigenous mortality were obtained from a variety of published and unpublished sources. Australian Aboriginal mortality data were procured from studies conducted in the Northern Territory, 1979-1983 (Plant, 1988), Western Australia, 1983 (Health Department of Western Australia, 1985), and western New South Wales, 1984-1987 (Gray and Hogg, 1989). These studies were principally chosen because information on causes of death by age and sex and on the population at risk was readily available. Data on American Indians, Canadian Registered Indians, and New Zealand Maoris were obtained respectively from the Indian Health Service, Health and Welfare Canada, and the National Health Statistics Centre. For Canadian Registered Indians and New Zealand Maoris I was able to obtain unpublished data on all registered deaths by age, sex, and major cause, and the population at risk of death for the years 1980-1986. In contrast, for American Indians I was only able to obtain published summary data on American Indian mortality in Reservation states for the years 1983-1985. For the Pacific Islands, information on indigenous mortality was limited to what was available from published sources. For reasons of clarity and simplicity, I chose to use the data from a study of mortality in Nauru, 1976-1981 (Taylor and Thoma, 1985), because the pattern of death for the indigenous population of this Pacific Island was very similar to that of Australian Aborigines.

The mortality patterns of these populations were compared by looking at differences in life expectancy and age- and cause-specific patterns of death. Life expectancies and age-specific mortality rates were mainly derived from published estimates. However, as already stated, in the case of Canadian Registered Indians and New Zealand Maoris these measures were produced directly from the available six year totals of deaths.

Comparisons of cause-specific patterns of death were made by calculating both cause-specific death rates and potential years gained due to the removal of specific causes of death. Cause-specific death rates were calculated by directly standardizing, to the Australian age-structure in 1986, indigenous age- and sex-specific death rates for the major causes of death. Once rates were standardized, age-specific relative risk, a ratio of the indigenous rate to the Australian rate, and confidence intervals, at a 95 per cent level of confidence, around each ratio of relative risk were determined (Bailar and Ederer, 1964). To limit the effects of small numbers, relative risk ranges were only calculated for cause-specific mortality rates based on five or more deaths.

Estimates of the potential gain in life expectancy for each of the 17 major causes of death were calculated from a series of multiple decrement cause-deleted life tables (see Chiang, 1977) using a program called SURVIVAL (Smith, 1988). In this case, after producing a series of cause-deleted life tables, the program printed a summary table displaying the estimated potential gain in life expectancy to age 75 years that would result by the elimination of each specific cause. An interaction term was also printed as a residual, and was distributed to the various causes in proportion to their unadjusted levels.

As shown in Table 2.1, three distinct groups of male and female life expectancies are discernible: relatively high life expectancies (American and Canadian Indians and New Zealand Maoris); moderately low life expectancies (Australian Aborigines and female Nauruans); and low life expectancies (male Nauruans). American Indian men and women tend to have higher life expectancies at birth and age 15 years than any other

group, although differences between American Indian male and New Zealand Maori male and American and Canadian Indian female life expectancies are more difficult to discern because they are so small. Differences between Northern Territory, Western Australia and western New South Wales life expectancies at birth are probably partly due to the differing reference periods. Khalidi (1989) noted that in one region of the Northern Territory there was a marked increase in male and female life expectancy during the decade before the mid-1980s.

Indigenous patterns of age-specific mortality differ mainly in degree. As shown in Figure 2.1, they all show a characteristic peaking during early and middle adulthood. Although comparable sources of data are not available for Nauruans, available evidence suggests that their patterns are similar (see Taylor and Thoma, 1985). This characteristic peaking is due to a pattern of high adult mortality which is not found in developed countries even when life expectancies are similar. Figure 2.2 shows that even when the 1921-1925 total Australian population (which had life expectancies at birth of 59 years for males and 63 years for females) is compared to the 1981-1986 Aboriginal population (with life expectancies of 56 years for males and 64 years for females), there are notable differences in age-specific death rates. In particular, Aborigines have much lower infant and higher adult age-specific death rates than did this Australian population (see Gray, 1990b; Australian Bureau of Statistics, 1976). As a whole, indigenous age-specific death rates vary more at younger and older ages than during early and middle adulthood.

Even though indigenous groups have similar patterns of age-specific mortality, the contributions of the primary underlying causes of death are very different. For example, the prevalence of circulatory and respiratory system diseases is noticeably higher among Australian Aborigines than Canadian Indians or New Zealand Maoris; and deaths from external causes are higher among Australian Aborigines and Canadian Indians than among New Zealand Maoris (see Tables 2.2 and 2.3). Data available for American Indians suggest that they may have very low rates of heart disease — 1.3 and

0.9 per 1,000 respectively for males and females (U.S. Department of Health and Human Services, Public Health Service, Indian Health Service, 1988). However, among Nauruans, especially males, the prevalence of deaths due to heart disease and accidents and injury is very high (Taylor and Thoma, 1985).

When indigenous mortality rates are compared with 1986 Australian cause-specific rates, a slightly different pattern emerges among the prominent causes of death (see Tables A1.1 to A1.10 in Appendix 1). Among male and female Australian Aborigines and New Zealand Maoris, circulatory system diseases account for between 20 and 60 per cent of the excess risk; while among Canadian Indians they account for only a negligible amount. Additionally, although Canadian Indians and Australian Aborigines have comparable rates of deaths from external causes, among Canadian Indians these causes represent a much greater proportion of the excess risk — 77 and 45 per cent respectively for males and females.

Comparisons of potential gains in life expectancy again demonstrate that circulatory system disease death rates are considerably higher among Australian Aborigines than among other indigenous groups (see Tables 2.4 and 2.5). When circulatory system diseases are deleted as a cause of death, Aboriginal men have potential gains in life expectancy to age 75 years of between five and eight years, while women have potential gains of approximately four years. In comparison, Canadian Indians and New Zealand Maoris of both sexes have potential gains in life expectancy to age 75 of about two to three years. The removal of respiratory system diseases or external causes also significantly increases Aboriginal life expectancy. For Canadian Indians the removal of external causes substantially increases both male and female life expectancy. Although this type of exercise is hypothetical, it does demonstrate the underlying relative importance of different causes of death.

This comparative analysis has demonstrated that although Australian Aborigines, Canadian Indians, and New Zealand Maoris have similar patterns of high adult

mortality, the relative importance of some more notable causes of death is strikingly different. Underlying disparities in cause-specific death rates, especially from circulatory and respiratory system diseases and external causes, account for some of the differences in life expectancies between these indigenous groups. Although this pattern of high adult mortality is also most likely found among American Indians and Nauruans, the data available are not sufficient enough to be certain. Overall, this section has demonstrated that Australian Aborigines have remarkably high rates of death from circulatory system diseases; and that any substantial reduction in the incidence of circulatory system diseases would most likely be associated with a significant decrease in adult mortality and an increase in life expectancy.

Table 2.1 Life expectancy at birth and age 15 years for various indigenous populations worldwide, by sex.

Population	Males		Females	
	Age 0	Age 15	Age 0	Age 15
Australian Aborigines				
Australian Aborigines, 1981-86 ¹	56	N/A	64	N/A
Northern Territory, 1979-83 ²	51	40	58	47
Western Australia, 1983 ³	56	44	64	51
Western New South Wales, 1984-87 ⁴	54	41	65	51
American Indians				
Reservation Indians, 1979-81 ⁵	67	54	75	62
Canadian Indians				
Registered Indians, 1980-86 ⁶	64	53	73	61
New Zealand Maoris				
Maoris, 1980-86 ⁷	66	52	71	58
Other Pacific Island populations				
Nauru, 1976-81 ⁸	49	37	62	51

1. Source: Gray, (1990b).

2. Source: Plant (1988).

3. Source: Health Department of Western Australia (1985).

4. Source: Gray and Hogg (1989).

5. Source: Vital Events Staff, Program Statistics Branch, Evaluation and Legislation, Indian Health Service (1984).

6. Source: unpublished data from Health and Welfare Canada.

7. Source: unpublished data from National Health Statistics Centre.

8. Source: Taylor and Thoma (1985).

Table 2.2 Male cause-specific mortality rates¹ for Aborigines from the Northern Territory (NT)², 1979-1983, Western Australia (WA)³, 1983, and western New South Wales (WNSW)⁴, 1984-1987, Canadian Indians⁵, 1980-1986, and New Zealand Maoris⁶, 1980-1986.

Cause of death	Australian Aborigines			Canadian Indians	New Zealand Maoris
	NT	WA	WNSW		
All causes	28.4	21.0	23.3	11.2	12.3
Infectious and parasitic	1.1	0.3	0.2	0.2	0.2
Malignant neoplasms	2.2	1.9	3.0	1.2	3.2
Endocrine, nutritional	0.7	0.3	0.2	0.2	0.6
Blood and blood-forming organs	0.1	0.0	0.0	0.0	0.0
Mental disorders	0.3	0.6	0.3	0.1	0.1
Nervous system	0.5	1.2	0.2	0.1	0.1
Circulatory system	8.9	6.7	12.6	3.6	5.1
Respiratory system	6.2	3.7	1.3	1.0	1.5
Digestive system	0.8	1.0	1.7	0.5	0.3
Genito-urinary system	1.0	1.4	0.5	0.2	0.2
Skin and subcutaneous tissue	0.0	0.0	0.0	0.0	0.0
Musculoskeletal system	0.1	0.3	0.0	0.0	0.0
Congenital anomalies	0.1	0.0	0.2	0.1	0.1
Perinatal causes	0.0	0.0	0.2	0.2	0.1
Signs, symptoms and ill-defined conditions	2.1	0.4	1.0	0.4	0.1
Accidents, poisonings and violence	4.3	3.3	2.0	3.3	0.9

1. Rates are deaths per 1,000 population per year. Cause-specific rates were directly standardized to the 1986 Australian age structure.

2. Source: Plant (1988).

3. Source: Health Department of Western Australia (1985).

4. Source: Gray and Hogg (1989).

5. Source: unpublished data from Health and Welfare Canada.

6. Source: unpublished data from National Health Statistics Centre.

Table 2.3 Female cause-specific mortality rates¹ for Aborigines from the Northern Territory (NT)², 1979-1983, Western Australia (WA)³, 1983, and western New South Wales (WNSW)⁴, 1984-1987, Canadian Indians⁵, 1980-1986, and New Zealand Maoris⁶, 1980-1986.

Cause of death	Australian Aborigines			Canadian Indians	New Zealand Maoris
	NT	WA	WNSW		
All causes	22.4	15.3	14.6	8.8	10.3
Infectious and parasitic	1.2	0.2	0.2	0.2	0.2
Malignant neoplasms	2.0	2.3	1.8	1.4	2.7
Endocrine, nutritional	1.4	1.5	1.4	0.3	0.6
Blood and blood-forming organs	0.5	0.0	0.0	0.0	0.0
Mental disorders	0.2	0.4	0.1	0.1	0.0
Nervous system	0.3	0.0	0.1	0.1	0.1
Circulatory system	6.4	5.5	7.2	3.1	4.6
Respiratory system	5.0	1.6	1.5	0.9	1.3
Digestive system	0.8	0.4	0.5	0.6	0.2
Genito-urinary system	1.1	1.5	0.6	0.2	0.2
Pregnancy, childbirth,	0.1	0.0	0.0	0.0	0.0
Skin and subcutaneous tissue	0.1	0.0	0.0	0.0	0.0
Musculoskeletal system	0.1	0.0	0.0	0.0	0.0
Congenital anomalies	0.2	0.2	0.0	0.1	0.0
Perinatal causes	0.2	0.0	0.0	0.1	0.1
Signs, symptoms and ill-defined conditions	1.4	0.4	0.2	0.4	0.1
Accidents, poisonings and violence	1.5	1.4	1.0	1.3	0.3

1. Rates are deaths per 1,000 population per year. Cause-specific rates were directly standardized to the 1986 Australian age structure.

2. Source: Plant (1988).

3. Source: Health Department of Western Australia (1985).

4. Source: Gray and Hogg (1989).

5. Source: unpublished data from Health and Welfare Canada.

6. Source: unpublished data from National Health Statistics Centre.

Table 2.4 Potential gain in male life expectancy to age 75 years from a specific cause of death being eliminated for Aborigines from the Northern Territory (NT)¹, 1979-1983, Western Australia (WA)², 1983, and western New South Wales (WNSW)³, 1984-1987, Canadian Indians⁴, 1980-1986, and New Zealand Maoris⁵, 1980-1986.

Cause of death eliminated	Potential gain in life expectancy (in years)				
	Australian Aborigines NT	WA	WNSW	Canadian Indians	New Zealand Maoris
Infectious and parasitic	1.4	0.7	0.5	0.2	0.2
Malignant neoplasms	1.8	1.0	2.5	0.9	2.6
Endocrine, nutritional	0.6	0.0	0.3	0.2	0.5
Blood and blood-forming organs	0.1	0.0	0.0	0.0	0.0
Mental disorders	0.4	0.2	0.4	0.2	0.1
Nervous system	0.7	1.4	0.3	0.2	0.1
Circulatory system	6.5	5.3	8.0	2.4	3.8
Respiratory system	4.8	2.4	1.2	0.8	1.0
Digestive system	1.0	1.3	1.8	0.7	0.3
Genito-urinary system	0.8	1.1	0.1	0.1	0.1
Skin and subcutaneous tissue	0.0	0.0	0.0	0.0	0.0
Musculoskeletal system	0.2	0.2	0.0	0.0	0.0
Congenital anomalies	0.6	0.0	0.6	0.5	0.2
Perinatal causes	1.3	1.7	0.7	0.8	0.3
Signs, symptoms and ill-defined conditions	1.4	0.2	2.2	0.9	0.5
Accidents, poisonings and violence	5.7	4.2	3.5	6.8	1.9

1. Source: Plant (1988).

2. Source: Health Department of Western Australia (1985).

3. Source: Gray and Hogg (1989).

4. Source: unpublished data from Health and Welfare Canada.

5. Source: unpublished data from National Health Statistics Centre.

Table 2.5 Potential gain in female life expectancy to age 75 years from a specific cause of death being eliminated for Aborigines from the Northern Territory (NT)¹, 1979-1983, Western Australia (WA)², 1983, and western New South Wales (WNSW)³, 1984-1987, Canadian Indians⁴, 1980-1986, and New Zealand Maoris⁵, 1980-1986.

Cause of death eliminated	Potential gain in life expectancy (in years)				
	Australian Aborigines NT	WA	WNSW	Canadian Indians	New Zealand Maoris
Infectious and parasitic	1.6	0.5	0.7	0.3	0.1
Malignant neoplasms	1.8	1.7	1.0	1.2	2.5
Endocrine, nutritional	1.0	1.1	0.9	0.3	0.5
Blood and blood-forming organs	0.5	0.0	0.0	0.0	0.0
Mental disorders	0.2	0.4	0.3	0.2	0.0
Nervous system	0.6	0.1	0.4	0.1	0.1
Circulatory system	4.5	3.9	4.2	1.6	2.7
Respiratory system	3.7	1.3	0.9	0.6	1.0
Digestive system	0.7	0.5	0.5	0.7	0.1
Genito-urinary system	1.0	1.1	0.7	0.2	0.1
Pregnancy, childbirth,	0.3	0.0	0.0	0.0	0.0
Skin and subcutaneous tissue	0.0	0.0	0.0	0.0	0.0
Musculoskeletal system	0.1	0.0	0.0	0.0	0.0
Congenital anomalies	0.7	0.7	0.2	0.4	0.2
Perinatal causes	0.6	0.0	0.2	0.5	0.3
Signs, symptoms and ill-defined conditions	1.0	0.3	0.4	0.7	0.4
Accidents, poisonings and violence	2.6	3.0	1.8	2.9	0.6

1. Source: Plant (1988).

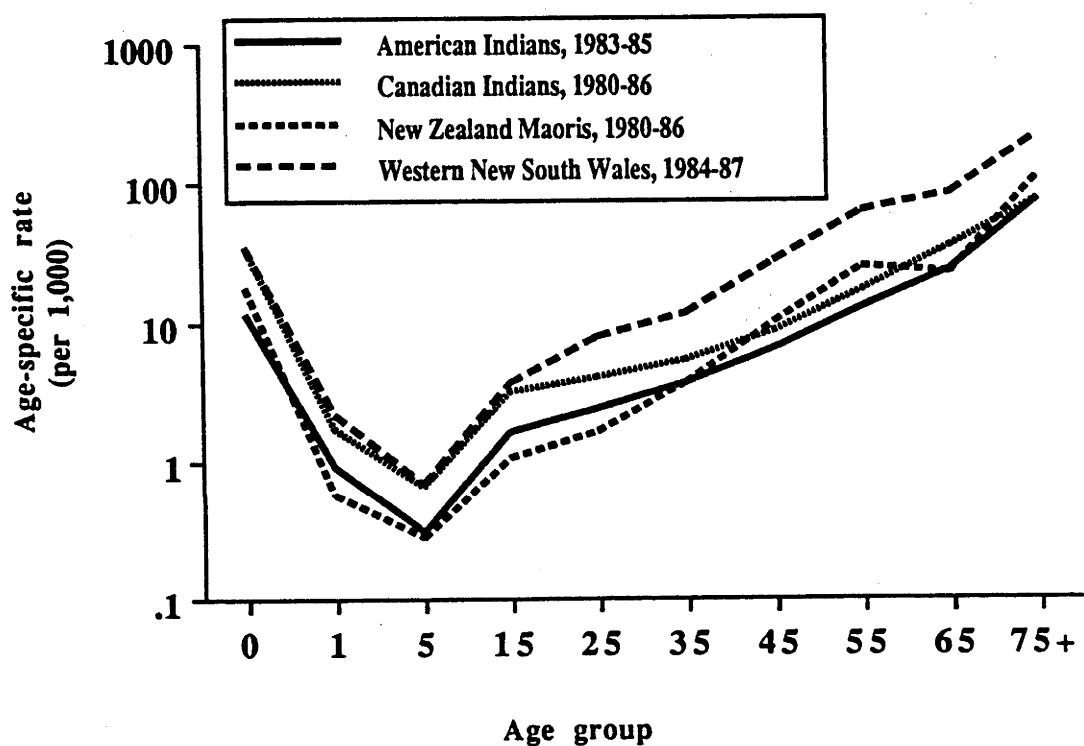
2. Source: Health Department of Western Australia (1985).

3. Source: Gray and Hogg (1989).

4. Source: unpublished data from Health and Welfare Canada.

5. Source: unpublished data from National Health Statistics Centre.

Figure 2.1 Age-specific mortality rates for Australian Aborigines from western New South Wales¹, 1984-1987, American Indians², 1983-1985, Canadian Indians³, 1980-1986, and New Zealand Maoris⁴, 1980-1986.



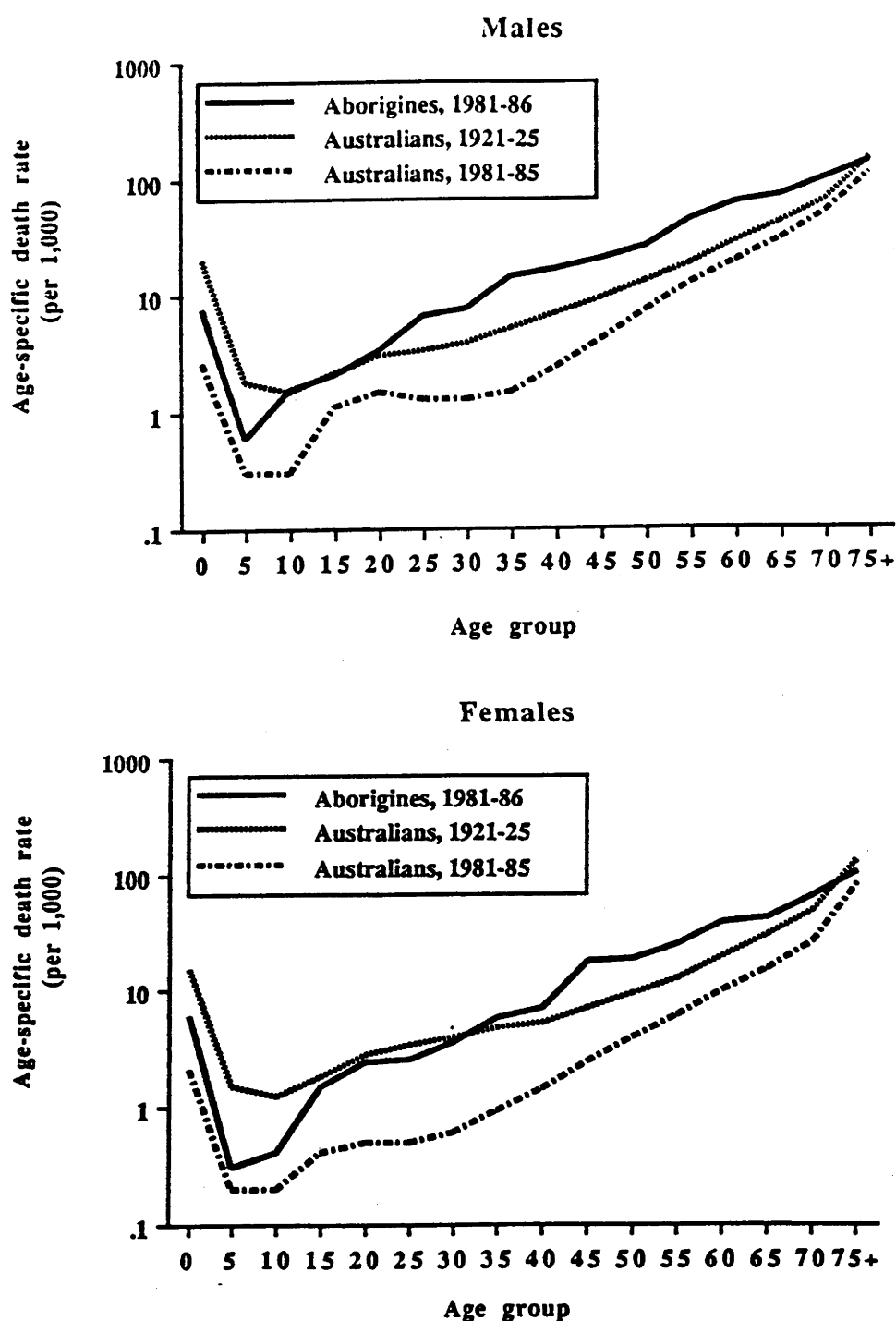
1. Source: Gray and Hogg (1989).

2. Source: U.S. Department of Health and Human Services, Public Health Service, Indian Health Service (1988).

3. Source: unpublished data from Health and Welfare Canada.

4. Source: unpublished data from National Health Statistics Centre.

Figure 2.2 Age-specific mortality rates for the total Australian population, 1921-1925¹ and 1981-1985², and Australian Aborigines³, 1981-1986.



1. Source: Australian Bureau of Statistics (1976).

2. Source: Australian Bureau of Statistics (1989b).

3. Source: Gray (1990b).

2.5 Risk factors of coronary heart disease

As discussed in chapter 1, there are two major types of risk factors of coronary heart disease — biological and behavioural. Biological risk factors are biological abnormalities detectable before the appearance of the first symptoms or clinical problems characteristic of the disease; and behavioural risk factors are characteristics which, as they appear before the onset of the disease, can be controlled by clinical, epidemiological, and other types of intervention (Péron and Strohmenger, 1985).

The purpose of this discussion is to highlight differences in the prevalence of several biological and behavioural risk factors of coronary heart disease between Australian Aborigines and other indigenous groups. The four biological risk factors examined are diabetes, hypertension, obesity, and high serum cholesterol; and the four behavioural risk factors examined are alcohol consumption, tobacco consumption, dietary change, and physical inactivity. However, because so much emphasis has been placed on diabetes in the literature, the review of this risk factor is much more extensive than the other seven risk factors. This is especially so with physical inactivity, where I was only able to find a few relevant studies.

2.5.1 Non-insulin dependent diabetes mellitus

Diabetes is a major problem among all the indigenous groups examined here. However, because of the underlying heterogeneity in the way diabetes studies are conducted, many problems arise in trying to review works on diabetes epidemiology critically. Some of the major problems are due to the way in which populations are sampled and screened, in which known diabetics are included in studies, and in which hospital admissions and mortality data are used (Zimmet, 1982: 400). In an attempt to minimize some of these problems, initial comparisons were made between risk factor prevalence studies which were based on diagnostic criteria set out by the World Health Organization (1980) for impaired glucose tolerance and diabetes mellitus.

Table 2.6 provides a selection of crude prevalence rates of diabetes in various indigenous populations worldwide. As shown in this table, low prevalence rates have been noted for some American Indian (Mouratoff *et al.*, 1967, 1969) and Pacific Island populations (Zimmet, 1979, 1982); moderately high prevalence rates have been reported for New Zealand Maoris (Prior and Davidson, 1966) and Australian Aborigines (Wise *et al.*, 1970; Cameron *et al.*, 1986; Williams *et al.*, 1987); and very high prevalence rates have been reported for Pima Indians (Bennett and Knowler, 1980) and the Micronesian population of Nauru (Zimmet *et al.*, 1977).

When country-specific rates are compared without regard for differing methodologies, crude diabetes² prevalence rates for Australian Aborigines aged 20 years and over are known to vary from 8 per cent (Williams *et al.*, 1987) to 19 per cent (Wise *et al.*, 1970). Kamien (1976) noted that diabetes was present in 8 per cent of Bourke Aboriginal subjects over 30 years of age and 13 per cent of those over 40 years of age. Another study conducted in Bourke and the nearby community of Enngonia noted that these Aborigines had an age-sex standardized (to the New South Wales population in 1981) diabetes prevalence rate of 16 per cent (Cameron *et al.*, 1986); while a comparable study in two coastal New South Wales communities showed that Aborigines there had an age-sex standardized diabetes prevalence rate of 8 per cent. The considerable difference between these two age-sex standardized rates was partly explained by the extent of non-Aboriginal genetic admixture in the two coastal communities (Williams *et al.*, 1987). In South Australia, the difference in diabetes prevalence rates between Aboriginal reserves seems to be influenced by body build and by non-Aboriginal genetic admixture (Wise *et al.*, 1976). An Aboriginal Health Organization survey (n.d.) has also shown that rural, tribal, and urban populations of Aborigines have similar levels of diabetes and that diabetes is more commonly found in persons of either sex who are obese. In Western Australia, Bastian (1979) found a diabetes prevalence rate

2. In most instances crude prevalence rates reported here refer to both types of diabetes mellitus — insulin and non-insulin dependent diabetes mellitus.

of 17 per cent for Aborigines living in west Kimberley. Finlay-Jones and McComish (1972) found an even higher rate among Aborigines in Derby, although this was a highly unusual population. Also, a large survey conducted in Western Australia on 1,218 diabetic subjects showed that the Aboriginal group had: a greater proportion of non-insulin dependent diabetics; a relative female dependence; and a tendency to present and be diagnosed at earlier ages of onset than the rest of the subjects (Stanton *et al.*, 1985: 16). Overall, the prevalence of diabetes seems to be higher among Aborigines who are obese and those who are of full Aboriginal descent.

In comparison with Australian Aborigines, the variation in the diabetes prevalence rates among American Indians is much more considerable. High crude prevalence rates are found among the Cherokee of North Carolina, the Comanche and Kiowa of Oklahoma, the Seneca of New York State, the Seminole of Florida, the Winnebago of Nebraska, as well as several tribes from Arizona, including the Pima, Papago, Maricopa, Hualapai, and Cocopah; and low crude prevalence rates are found among the Navajo of Arizona and the Athabaskan Indians and Eskimos of Alaska. Related tribes do not necessarily have similar prevalence rates, whereas only distantly related tribes may — which suggests that although the variation cannot be easily explained, environmental factors are of predominant importance (Bennett, 1983: 54).

Little is known about the prevalence of diabetes among Canadian Indians, as there are very few published reports on geographically defined Indian groups (Young *et al.*, 1985: 796). However, where diabetes prevalence rates have been reported they were generally low. A survey among the Dogrib Indians in the Northwest Territories, where clinical diabetes is unknown, showed that 13 per cent of males and 7 per cent of females over 20 years of age were hyperglycemic, which is one of the first indicators of diabetes (Szathmary and Holt, 1983). An unpublished study of Indians in Saskatchewan estimated that the prevalence rate of diabetes in that province was 3 per cent among those aged 20 years and over (Young *et al.*, 1985); while in eastern Manitoba, Dooley and Rodrigues (1984) estimated that the prevalence rate was 3 per cent for all ages.

Finally, among the Cree Indians living in northeastern Manitoba and Northwestern Ontario, Young and colleagues (1985: 796) estimated that the diabetes prevalence rate was 3 per cent overall, 5 per cent for those aged 15 to 64 years, and 10 per cent for those aged 65 years and over.

The prevalence of diabetes among New Zealand Maoris is relatively high. The prevalence rate is higher among women than men and rises for both sexes with increasing body mass index. In one study, 14 per cent of the men and 18 per cent of the women aged 25 years and over who were examined were either known to be or diagnosed as diabetics (Prior *et al.*, 1978).

One fact that has clearly emerged from studies of other Pacific Islanders is the wide disparity in the prevalence of diabetes (King *et al.*, 1984: 396). Age-specific standardized prevalence rates, based on the World Health Organization criteria for diabetes mellitus, have been found to range from less than 2 per cent in rural Melanesians in Fiji and New Caledonia (Zimmet, 1982) to 30 per cent in the affluent and urbanized Micronesian population of Nauru (Zimmet *et al.*, 1977).

Overall, however, Micronesians and Polynesians seem to have lower rates of diabetes than Melanesians, but results for Melanesians are often conflicting. Reports of minimal glucose intolerance (hyperglycemia) have been superseded by recorded diabetes prevalence rates of 5 to 15 per cent among some urban Melanesians (Eason *et al.*, 1987: 469). For example, a study of the predominantly Melanesian population in the Torres Strait region observed a diabetes prevalence rate of 19 per cent in the outer island area and a rate of 13 per cent in the Thursday Island and Bamaga areas. These prevalence rates, which are predominantly for non-insulin dependent diabetes mellitus, suggest that Melanesians, like other indigenous groups in the Pacific, are susceptible to the conditions which give rise to this disease (Duffy *et al.*, 1981: 11).

Researchers in the Pacific have also consistently noted a lower prevalence of diabetes in traditional-oriented rural communities. A study in Western Samoa noted that the

prevalence of diabetes in the urban population was more than twice that in the rural population — 10 versus 4 per cent for adults aged 20 years and over. While urban subjects were significantly more obese than their rural counterparts, the differences in their prevalence rates could not be wholly explained by this factor. Prevalence rates were still approximately double in urban subjects when obesity was controlled for, suggesting that other factors, like differences in diet, physical activity, or stress, might be important (Zimmet *et al.*, 1981).

Differences in the prevalence of non-insulin dependent diabetes mellitus among these indigenous populations are most probably due to a variety of environmental and genetic factors. The most important of these is obesity, which has been correlated with the incidence and prevalence of diabetes in various groups on an individual and total population basis (World Health Organization, 1980). The role of obesity as a risk factor of diabetes has generally been examined by assessing its duration or prevalence in a population. Measurements of the duration of obesity present great methodological difficulties and there are few reliable data with respect to non-insulin dependent diabetes mellitus (Zimmet, 1982: 406). One of the few more reliable studies was conducted among the Pima Indians in the southwestern United States, and it noted that the prevalence of diabetes was strongly related with preceding obesity. Age-sex adjusted prevalence rates rose steadily with increasing body mass index. However, as with the study in Western Samóa, these rates could not be attributed to obesity alone (Knowler *et al.*, 1982: 244). In this case they were also strongly related to whether an individual's parents were diabetic. When obese subjects were grouped according to whether they had no, one, or two parents with diabetes, the prevalence of diabetes increased synergistically, and obese subjects with two diabetic parents had the highest prevalence rate (Knowler *et al.*, 1983: 112).

How the association between obesity duration and parental diabetes contributes to an increased risk of diabetes in Pima Indians is currently unknown. Obesity may be either an environmentally induced factor which allows a diabetes genotype to express itself or

an early manifestation of a genetically determined obesity-diabetes syndrome (Knowler *et al.*, 1983: 112). Lillioja and Bogardus (1988: 103) propose that insulin resistance in Pima Indians may be due to a combination of a genetic defect and obesity-induced changes in the biophysical properties of the skeletal muscle. This combination, by slightly increasing the plasma glucose concentration and inducing pancreatic glucose insensitivity, may in turn lead to the development of non-insulin dependent diabetes mellitus.

Compared with incidence studies (those which only look at new cases of diabetes), prevalence studies show less of a relationship between diabetes and obesity. They indicate that although differences in obesity between two populations can explain some of the variation in the prevalence of diabetes, obesity is not the primary factor responsible for the variation (Zimmet, 1982: 406). Studies among the Pima and Dogrib Indians in North America have shown that little association exists between non-insulin dependent diabetes mellitus and concurrent obesity (Knowler *et al.*, 1981; Szathmary and Holt, 1983). Differences in the prevalence of diabetes may be related to the distribution of fat rather than to total fatness. Szathmary and Holt (1983: 507) observed that among the Dogrib Indians, hyperglycemic subjects differed from normal individuals not in the total amount of fat present, but in the location of that fat — more on the trunk and associated regions (chest and abdomen), and less on the distal extremities (arms and legs). In this case persons at risk of developing diabetes were those who began to deposit fat centripetally relative to appropriate members (same age and sex) of their own population. This distribution of fat may be a far more important risk factor than the presence of obesity in the early identification of those at risk of developing diabetes.

Acculturation may also be an important factor influencing the prevalence of diabetes. In a cross-cultural analysis of available literature on diabetes and culture change in Western and non-Western societies, Eaton (1977: 56) suggests that increased refined carbohydrate (especially sugar) consumption, coupled with decreased energy

expenditure and acculturational stress, correlate with an increased prevalence of non-insulin dependent diabetes mellitus. In non-Western societies the shift from a traditional to an urbanized lifestyle seems especially crucial in determining this susceptibility.

At an individual level, Australian Aborigines have been shown to be particularly prone to non-insulin dependent diabetes mellitus when they make such a transition. A comparison of lean young Aboriginal men from urban and rural communities has shown that there are a number of common metabolic characteristics which appear to be associated with this susceptibility: impaired glucose tolerance, high insulin response, and high serum and lipoprotein triglyceride levels (O'Dea *et al.*, 1982).

Current research also suggests that by having persons re-adopt specific components of a traditional lifestyle (adopting a traditional subsistence hunter-gatherer lifestyle for extended periods of time), non-insulin dependent diabetes mellitus can be a partly reversible or potentially preventable condition. O'Dea (1984: 602) has shown that when a group of diabetic Aborigines reverted temporarily to a traditional lifestyle, the major metabolic abnormalities of this condition were either greatly improved (glucose tolerance and insulin response to glucose) or completely normalized (plasma lipids, especially triglyceride concentrations). These observed improvements in insulin sensitivity and triglyceride levels were probably attributable to the low fat content of the traditional diet, weight loss, and increased physical activity.

The benefits of a traditional diet do not seem to be limited to diabetic Aborigines. A baseline study conducted in an outstation in northeastern Arnhem Land has shown that traditional-oriented Aborigines tend to have a better nutrition-related risk factor profile than urbanized Aborigines (O'Dea *et al.*, 1988: 179-180). The most striking difference is the extreme leanness of the Arnhem Land people — they were all underweight according to standard criteria for body mass index (less than 20 kg/metre²). In spite of this leanness they displayed no biochemical evidence of malnutrition. Their plasma

fatty-acid profiles were also consistent with a low dietary fat intake and a high consumption of lean meat. Fasting glucose and cholesterol levels were low relative to those of urbanized Aborigines and white Australians. However, their fasting insulin and triglyceride levels were inappropriately high for their very low body mass index and fasting glucose levels, which is consistent with insulin resistance and suggests that these Aborigines, like their urban counterparts, would be highly susceptible to obesity and diabetes when living a less traditional lifestyle.

Furthermore, findings from two recent investigations are consistent with the hypothesis that traditional starchy staple foods protect traditional-oriented indigenous people from developing diabetes. A study of traditional Aboriginal and Pacific Island foods has shown that these foods were as a rule digested by European subjects significantly more slowly than common Western foods; and that the starch in traditional Aboriginal bush foods was on average the least digested (Thorburn *et al.*, 1987a). Another study noted that while a slow-release food (Australian bush potato) did not produce significantly different glucose responses in Aborigines and Caucasians, a fast-release food (potato) produced much greater glucose and insulin responses in Aborigines (Thorburn *et al.*, 1987b). These research findings suggest that Aborigines and Pacific Islanders may have evolved gastrointestinal and endocrine systems better suited to metabolizing the slow-release carbohydrates in their traditional diets than the fast-release carbohydrates in Western diets.

In regard to heart disease, several microvascular and macrovascular complications of diabetes have been noted in indigenous people. A study of hospital admissions of Navajo Indians in the United States observed that the large number of admissions for specific complications of diabetes confirms that the Navajos suffer from typical microvascular and macrovascular complications, rather than from a benign form of hyperglycemia. Furthermore, the prevalence of diabetes among Navajos who had sustained a myocardial infarction increased from 34 per cent in 1976-1979 to 50 per cent in 1984-1986 (Sugarman *et al.*, 1990). Complications of diabetes also seem to be

strongly related to the duration of the disease. A study of Hopi and Navajo diabetic patients noted that the prevalence of retinopathy, peripheral neuropathy, and peripheral vascular disease increased with the known duration of diabetes (Rate *et al.*, 1983). Young and colleagues (1985: 795) also found that a duration of diabetes of five years or longer and the presence of hypertension at the time diabetes was diagnosed were associated with a statistically significant twofold increase of diabetic Cree and Ojibwa patients developing complications.

High insulin responses (hyperinsulinaemia), which have been noted in traditional- and urban-oriented Australian Aborigines (O'Dea *et al.*, 1982; O'Dea *et al.*, 1988), Pima Indians (Aronoff *et al.*, 1977), and some Pacific Islander groups (Zimmet *et al.*, 1979; Martin *et al.*, 1980) may also be an important risk factor of heart disease. Several studies have noted that individuals with high plasma insulin levels, in addition to being at risk of non-insulin dependent diabetes mellitus, are at an increased risk of coronary heart disease (Pyörälä, 1979; Welborn and Wearne, 1979; Ducimetiere *et al.*, 1980). In these studies the plasma insulin level was of better predictive value than plasma glucose in determining the risk of coronary heart disease and was independent of other coronary heart disease risk factors (O'Dea *et al.*, 1982: 152).

There may also be a link between maternal diabetes and heart disease. One study has shown that infants of diabetic mothers are prone to cardiomyopathy, an enlargement of the heart causing partial blockage of the blood flow (Way *et al.*, 1976; Wolfe and Way, 1977). The effect of this disease would mainly be felt not in childhood but in early adulthood. No study of which I am aware has tested the relationship between maternal diabetes and cardiomyopathy in indigenous people.

The significance of diabetes in terms of the prevalence of heart disease mortality in these indigenous populations is difficult to assess. Although this review is mainly a discussion of diabetes in its own right, it does suggest that the extremely high prevalence of heart disease among Australian Aborigines is unlikely to be associated

with the similarly high prevalence of diabetes. Clearly, non-insulin dependent diabetes mellitus is an important risk factor of heart disease, but other factors such as high insulin responses (hyperinsulinaemia) found among both diabetic and non-diabetic Aborigines may be even more important.

Table 2.6 Crude prevalence rates of diabetes for various indigenous populations worldwide¹.

Population	Age group (years)	Diagnostic criteria (Two-hour plasma glucose mmol/litre)	Diabetes prevalence (per cent)
Australian Aborigines			
South Australia ²	20+	≥ 8.3 (50g)	11.0
Bourke and Enngonia, N.S.W. ³	20+	≥ 11.0 (75g)	15.6
Purfleet and Foster, N.S.W. ⁴	20+	≥ 11.1 (75g)	7.8
American Indians			
Alaskan Eskimos ⁵	20+	≥ 8.9 (100g)	1.9
Athabaskan Indians ⁶	20+	≥ 8.9 (100g)	1.3
Pima Indians ⁷	20+	≥ 11.0 (75g)	34.1
New Zealand Maoris			
Maoris, Polynesian ⁸	20+	≥ 8.3 (100g)	7.5
Other Pacific Island populations			
Loyalty Islands, Melanesian ⁹	20+	≥ 11.0 (75g)	2.0
Nauru, Micronesian ¹⁰	20+	≥ 11.0 (75g)	30.3
Tuvalu, Polynesian ¹¹	20+	≥ 11.0 (75g)	3.9

1. Adapted from Zimmet (1982: 402-403, Tables 1 and 3).

2. Source: Wise *et al.* (1976).

3. Source: Cameron *et al.* (1986).

4. Source: Williams *et al.* (1987).

5. Source: Mouratoff *et al.* (1967).

6. Source: Mouratoff *et al.* (1969).

7. Source: Bennett and Knowler (1980).

8. Source: Prior and Davidson (1966).

9. Source: Zimmet, (1982).

10. Source: Zimmet *et al.* (1977).

11. Source: Zimmet (1979).

2.5.2 Hypertension

Indigenous people, as noted by the studies summarized here, have quite high levels of hypertension. Aboriginal women in Inverell, New South Wales, have been shown to have higher blood pressures than non-Aboriginal women (Simons *et al.*, 1981).

Another study conducted in the west Kimberley region of Western Australia showed that 37 per cent of all Aborigines aged 21 years and over were hypertensive (Bastian, 1979). However, the Aboriginal Health Organization in South Australia has noted that high blood pressure is no more a problem among Aborigines than among the general Australian population (Aboriginal Health Organization, n.d.).

Levels of hypertension among southwestern American Indians are higher than corresponding levels in the white population (Becker *et al.*, 1988). In Canada, findings from a three-communities study in northwestern Ontario suggest that 13 per cent of the Cree and Ojibwa Indian population in these communities are hypertensive; in the vast majority, but especially in females, the disease had been previously diagnosed (McIntyre and Shah, 1986).

In New Zealand, Maoris also have a different pattern of increasing blood pressure than the white population. On average, Maori youths have lower blood pressures than their Non-Maori counterparts, but from age 23 years and over their the blood pressures are distinctly higher than those of non-Maoris (Wood and Gans, 1986). High systolic blood pressure in Maoris may also be a good predictor of coronary heart disease. One recent five-year follow-up study noted an association between the age-adjusted rate of coronary heart disease and initial systolic blood pressure readings. In both men and women there was nearly a threefold increase in the rate of coronary heart disease between the lowest and highest blood pressure readings (Beaglehole *et al.*, 1978).

Studies of Pacific Islanders generally note an age-related rise in blood pressure and an increased prevalence of hypertension accompanying urbanization. This increase in

hypertension is associated with obesity, stress, physical inactivity, and a high salt intake (Patrick *et al.*, 1983; Taylor *et al.*, 1985; Eason *et al.*, 1987).

2.5.3 Obesity

The relationship between obesity and diabetes has been discussed in section 2.5.1.

Generally, however, obesity is a major health problem among Australian Aborigines, American and Canadian Indians, New Zealand Maoris, and other Pacific Islanders. The problem of obesity among Australian Aborigines has been noted country-wide: in Bourke (Cameron *et al.*, 1986); Kempsey (Sibthorpe, 1988); Wilcannia (Stephenson and Lenz, 1990); South Australia (Aboriginal Health Organization, n.d); and the west Kimberley region of Western Australia (Bastian, 1979)³. Furthermore, obesity has been significantly correlated with coronary heart disease in both men and women (Bastian, 1979).

Information from published sources on American Indian obesity prevalence rates is rather sparse. Becker and colleagues (1988) have noted that the prevalence of obesity among southwestern American Indians is very high compared with white Americans. West (1978: 201) has suggested that the high prevalence of non-insulin dependent diabetes mellitus among Oklahoma Indians is largely attributable to the high rate of obesity. However, as noted above, more recent studies of the Pima (Knowler *et al.*, 1981) and Dogrib Indians (Szathmary and Holt, 1983) suggest that there is little association between non-insulin dependent diabetes mellitus and concurrent obesity.

In Canada, a study among the Cree and Ojibwa Indians in northwestern Ontario noted that the proportion of the population who were obese increased with age. Over 70 per cent of women between the ages of 35 and 64 years were obese; and the highest rate of obesity in men was for those aged 35 to 44 years — approximately 50 per cent. For both sexes obesity was significantly related to hypertension ($p < 0.001$), but this

3. See section 6.3.1 in chapter 6 for a further discussion of these findings.

association was dependent on age (McIntyre and Shah, 1986). Young and Sevenhuysen (1989) noted that obese Cree and Ojibwa subjects had higher age-adjusted mean serum lipids, blood pressures, plasma glucose, and glycosylated haemoglobin, but lower total energy intake. They also found that obesity, as measured by body mass index, was an independent and significant predictor of both diabetes and hypertension.

In New Zealand, deaths and hospital admissions due to obesity-related conditions are strikingly more common among Maoris, especially Maori males. Although the number of cases is small, the rate of obesity-related hospital admissions for Maori men is 14 times that of non-Maori men. Obesity in Maori women is least prevalent among those less than 30 years of age and greatest in those over 45 years of age; overall, however, nearly three-quarters of Maori women are overweight (Pomare and de Boer, 1988; Birkbeck, 1977).

Obesity was historically regarded as a sign of high status and prosperity in Micronesian and Polynesian societies. Currently, a high prevalence of persons who are overweight or obese has been noted in some Melanesian, Micronesian, and Polynesian populations. Within populations there are some notable rural-urban and male-female differences in the prevalence of obesity (Zimmet and Whitehouse, 1981; King *et al.*, 1984; Taylor, *et al.*, 1985).

2.5.4 Serum cholesterol

As a rule, studies of cholesterol observe that indigenous people have lower total cholesterol levels than non-indigenous people. In Australia, a coronary heart disease risk factor study in Inverell, New South Wales, noted that the average serum cholesterol level in Aborigines was 0.4 to 1.0 mmol/litre lower than in non-Aborigines, and the difference increased with age. However, a large proportion of Aboriginal men and women had very high triglyceride levels (Simons *et al.*, 1981). A similar pattern was noted in Wilcannia, where Aboriginal total cholesterol levels were found to be slightly lower than those for non-Aborigines (Stephenson and Lenz, 1990). Among Bourke and

Enngonia Aborigines, serum cholesterol and triglyceride values have been shown to be significantly higher in men with previously undiagnosed glucose intolerance than in those with normal glucose tolerance (Cameron *et al.*, 1986). In Western Australia, serum cholesterol was found to be significantly higher in Aboriginal men with probable or suspected coronary heart disease (Bastian, 1979).

Indians in the southwestern United States were shown to have lower serum cholesterol levels than age- and weight-matched white Americans; furthermore, these levels rose very little with advancing age (Sievers, 1968). Indians living in the southwest were also shown to have a higher (more favourable) ratio of high-density to low-density lipoproteins compared with non-Indian controls (Garnick *et al.*, 1979).

In New Zealand, one investigation of serum lipid levels among a sample of Maoris observed notable associations and age- and sex-specific patterns (Beaglehole *et al.*, 1979). Although this investigation found no sex-specific differences in cholesterol levels by age group, notable differences in triglyceride levels were found. For either sex, high-density lipoprotein levels were found to be negatively correlated with body mass index and for men to be positively correlated with alcohol consumption. Overall, Maori men and women were found to have lower total cholesterol and high-density lipoprotein levels but higher triglyceride levels than non-Maoris.

2.5.5 Alcohol consumption

No comprehensive statistics of alcohol consumption are available for Australian Aborigines, American and Canadian Indians, New Zealand Maoris, or other Pacific Islanders. Generally, available information on alcohol consumption comes either directly from prevalence studies or indirectly from alcohol-related morbidity and mortality statistics.

A study conducted among Australian Aborigines in Bourke during the early 1970s noted that of the 124 men surveyed, only 10 per cent were non-drinkers while 53 per

cent were heavy drinkers consuming more than 80 grams of alcohol per day (Kamien, 1975a). Among males, drinking began between the ages of 14 and 19 years and most drinkers consumed some alcohol every day. Women were much lighter drinkers than men — only 4 per cent of the 128 women who were surveyed fit into the heavy drinking category. This study also noted that drinking was very much a social event which primarily occurred in two characteristic forms (Kamien, 1975a: 295). The first form of drinking tended to occur after work when men drank beer in the public bars of hotels in town, clearly enjoying the social benefits of drinking without trying to get drunk. By contrast, the aim of the second form of drinking was to get drunk. Cheap fortified wine was consumed by the riverbank or on the Bourke reserve over the space of a few hours. This form of drinking, the 'periodic binge', was the most common.

A study of Aboriginal mortality patterns in the country regions of New South Wales noted that in 34 per cent of the male and 15 per cent of the female deaths reported, alcohol was mentioned on the death certificate as a significant medical problem. Alcohol abuse was most frequently reported for male and female deaths between the ages of 35 and 44 years; and was most frequently associated with deaths from diseases of the digestive system (especially of the liver and pancreas) and respiratory system (especially pneumonia) (Smith *et al.*, 1983).

Alcohol is the leading and perhaps the most costly risk factor among American Indians. Alcohol abuse underlies many major causes of death and contributes to an array of physical conditions treated by the Indian Health Service. Four of the top ten causes of death among American Indians are alcohol-related — injuries (18 per cent of all deaths), chronic liver disease and cirrhosis (5 per cent), suicide (3 per cent), and homicide (3 per cent) (Rhoades *et al.*, 1987b: 365).

However, several studies have noted that not all American Indians can be stereotyped as being heavy drinkers. For example, although the Hopi and Navajo Indians in Arizona have adapted to the same environment, their differing patterns of social organization

and socialization have led to very different rates of death from alcoholic liver disease, homicide, and suicide (Kunitz *et al.*, 1971; Kunitz, 1983, 1990b). Also, two studies in Oklahoma have found significant differences in alcohol-related deaths by tribal area (Stratton, 1981; Christian *et al.*, 1989). For example, Christian and colleagues (1989: 275) noted that the Cheyenne-Arapaho, Comanche, and Kiowa areas (in western Oklahoma) have higher contemporary alcohol-related death rates than the Cherokee, Choctaw, Creek, Seminole, and Pawnee areas (in eastern Oklahoma). Overall, historical and cultural factors are assumed to be important in explaining this east-west dichotomy.

Alcohol abuse among Canadian Indians also seems to be an ever-increasing problem. A health survey conducted by Young (1982) in and around Sioux Lookout, Ontario, noted that 29 per cent of men and 14 per cent of women over 15 years of age drank alcohol. The proportion of drinkers declined sharply among the older age groups; and drinking was more prevalent among the better educated. However, these response rates may not actually reflect the true state of affairs. Many northern Indian reserves have passed by-laws banning alcohol, and although drinking still occurs in these 'dry' communities, the typical pattern is one of binge drinking when residents visit southern towns.

Also, a study of deaths among Canadian Indians living in 35 communities or reserves in Alberta estimated that a large majority of accidental and violent deaths (over 40 per cent) were directly or indirectly attributable to alcohol abuse. That is, four out of five deaths not due to natural causes were in part attributable to alcohol abuse (Jarvis and Boldt, 1980: 20).

Alcohol abuse occurs among all human populations, including those where its use is forbidden. Among indigenous populations alcohol abuse seems to be directly associated with the environmental and social-cultural context rather than due to any underlying biological or genetic susceptibility (Fisher, 1987: 81). As Kamien (1975a: 297) states about Bourke Aborigines:

In this community, drinking was due more to group psychological pressures rather than to individual psychological need. Because of this, any logical approach aimed at diminishing the alcohol problem needs to be community directed, and to influence the norms of peer groups rather than those of individuals. Heavy alcohol intake was a partially adaptive response to the life problems of Aborigines and until other available solutions are available to them, they will continue to drink in excess.

Thus, Aboriginal drinking habits, like those of other indigenous people, are tied to the general lack of employment, housing, education opportunities, proper health facilities, and general recreational outlets (see Perkins, 1976; Brody, 1977; Stratton, 1981; Fisher, 1987).

2.5.6 Cigarette smoking

As with alcohol consumption, information on the smoking habits of Australian Aborigines, American and Canadian Indians, New Zealand Maoris, and other Pacific Islanders is very limited. Kamien's (1976) study of Bourke Aborigines during the early 1970s observed that the vast majority of them were either current or ex-smokers — 78 and 7 per cent respectively. Forty-nine per cent of the men and 11 per cent of the women who smoked were regular smokers by the age of 12 years; and 94 per cent of the men and 53 per cent of the women who smoked were regular smokers by the age of 16 years. On average, Aboriginal men and women started smoking ten years earlier than their counterparts in the white population. The coronary heart disease risk factor study in Inverell, New South Wales, noted that a greater proportion of Aborigines than non-Aborigines smoked at least ten cigarettes per day (Simons *et al.*, 1981). Additionally, Stephenson and Lenz (1990) noted that there are approximately two times more Aboriginal than non-Aboriginal current smokers in Wilcannia.

A recent study of the smoking behaviour of Indians living in the western United States noted that cigarette smoking is more prevalent among Indians than non-Indians. Also, the use of smokeless tobacco is widespread among males, especially those under the age of 25 years (Goldberg and Warren, 1990). Another study conducted by the Alaskan

Native Health Board observed that 54 per cent of adults and 41 per cent of youths aged 12 to 18 smoked (Lee, 1984).

A survey conducted among Canadian Indians in the Northwest Territories in 1985 noted that 49 per cent of a representative sample of 1,000 native and non-native residents currently smoked. Of the native portion of the sample, 62 per cent of the Dene and 76 per cent of the Inuit smoked. Furthermore, native smoking rates were higher among those under 30 years of age (Health and Welfare Canada, 1988). McIntyre and Shah's (1986) survey among the Cree and Ojibwa Indians in northwestern Ontario noted that 65 per cent of the men and 47 per cent of the women were smokers. The highest rate of smoking was among men and women aged 15 to 24 years, and few people over 65 years of age smoked. The vast majority of male smokers were daily smokers, while among women there were about the same number of daily and occasional smokers.

The 1981 New Zealand census indicates that a greater proportion of Maoris than non-Maoris smoke cigarettes regularly. Of the total Maori respondents aged 15 years and over, 54 per cent of males and 58 per cent of females smoked, as compared with 33 per cent of non-Maori males and 27 per cent of non-Maori females. This difference is seen at all age groups and especially among females. Of notable significance is the large proportion of Maori compared to non-Maori women of childbearing age (15 to 44 years of age) who are smoking: 63 versus 31 per cent (Pomare and de Boer, 1988; Pool, 1983).

Additionally, information in the 1981 New Zealand census suggest that a large number of migrant Pacific Islanders smoke. Forty-six per cent of the male and 24 per cent of the female respondents were current smokers; and 10 and 7 per cent respectively of the males and females were ex-smokers. Furthermore, data from a study by Prior and Tasman-Jones (1981) support the idea that migrant Islanders are at a greater risk of taking up smoking when they arrive in New Zealand.

2.5.7 Dietary change

The increased prevalence of coronary heart disease, diabetes, and hypertension among indigenous people can probably be partly explained by an increasing shift in dietary habits away from locally available food sources (bush foods) towards non-local refined and packaged foodstuffs. This factor among Australian Aborigines has been linked to the prevalence of malnutrition and infections in Aboriginal infants and children, maternal malnutrition and health problems, cardiovascular diseases, and diabetes (Gracey, 1986). A survey conducted among Aborigines living in six missions, five government settlements, and six cattle stations in the Northern Territory during the 1950s noted that the amount of indigenous foods consumed was not large. However, the mean consumption of bread, cereals, and flour was approximately twice the average Australian consumption; while the consumption of meat was 30 per cent higher. Milk products, fruit, vegetables, fats, and eggs were on average consumed less often than by other Australians (Wilson, 1953).

Furthermore, significant differences in dietary habits seem to exist even among urbanized Aborigines. A study of dietary patterns in an Aboriginal community in southwestern Australia noted that nutrient intakes were closely related to several social factors including employment, education, and the standard of housing. Caloric and other nutrient intakes were the lowest in households on the reserve; while households in town and between town and the reserve had much higher intakes (Hitchcock and Gracey, 1975). Although Kamien and colleagues (1975a) also noted similar differences in the nutrient intakes of Bourke Aborigines, they concluded that the families living both on the reserve and in town had unsatisfactory diets. Overall, the high costs of food in remote towns and communities may help to encourage these unsatisfactory dietary patterns and socio-economic differentials (Sullivan *et al.*, 1987).

Not all Aboriginal dietary intake studies support the view that dietary intakes are unsatisfactory or inconsistent with the wider society. A recent dietary recall study

conducted by Sibthorpe (1988) among Kempsey Aborigines noted that fat, sugar, and vitamin intakes were on the whole not inconsistent with results for all Australians.

Evidence from the United States supports Sibthorpe's findings. A dietary intake study of Pima Indians aged 25 to 44 years showed that dietary habits met or exceeded American recommended allowances for calories, protein, calcium, iron, and magnesium. The ratio of polyunsaturated to saturated fat was also lower. Furthermore, no link could be made between the intake of selected nutrients and the high prevalence of gall bladder disease and diabetes among the Pima (Reid *et al.*, 1971).

In Canada, several researchers argue that large-scale development quickens the acculturation of traditional Indian dietary habits. For example, in northern Manitoba the Churchill-Nelson River Hydro Project has been shown to have indirectly increased the dependence of local Indians on non-local food sources. This dietary void has been filled by an increased consumption of store-bought meats and other foodstuffs which are generally less nutritious than locally available bush foods (Waldram, 1985). A similar pattern of change towards store-bought foods has occurred among the Cree and Inuit of northern Quebec. Again these changes were brought on by a major hydroelectric development in the area (Thouez *et al.*, 1989).

A dietary intake analysis among the Dogrib Indians in Canada noted that dietary differences by community and age demonstrate a consistent but different pattern of dietary acculturation. The traditional food base is stable between communities, as neither total grams of traditional foods consumed nor the total calories derived from them vary significantly. However, non-traditional intakes do differ, producing a pattern of variable amounts of 'new food' being added to a traditional food base. The net result by community or age is more a matter of change in the quantity of food eaten than the substitution of new foods for native foods (Szathmary *et al.*, 1987: 801).

In New Zealand, a study conducted by the National Heart Foundation during the mid-1970s (Birkbeck, 1977) remains to this day the only comprehensive dietary study

undertaken. Overall, the findings were that Maoris consume more energy and fat, particularly saturated fat, and significantly less fruit and vegetables than the non-Maori population (Pomare and de Boer, 1988).

Economic and social changes brought about by migration also have an important bearing on the availability and use of food and ultimately on nutrition and health status. For example, in New Zealand the dietary habits of migrants from the Cook Islands have been shown to shift from a diet high in fibre content and low in refined carbohydrates and salt to one low in fibre content and high in refined carbohydrates and salt. These dietary changes reflect the social adaptations the migrants have to make in the new environment, including adjustments to an increased income, the availability of new and familiar foods, and shifting work patterns (Fitzgerald, 1986).

The high prevalence rates of hypertension, heart disease, and cerebrovascular disease among Nauruans are presumed to be related to the high prevalence of obesity and high salt intake (Taylor *et al.*, 1985); while the prevalence of obesity is probably related to high caloric intake coupled with reduced physical activity (Ringrose and Zimmet, 1979). The move from a traditional diet of coconut products and fish to a reliance on store-bought foods (particularly polished rice, sugar, condensed milk, and canned meat and fish) became significant by the mid-1920s. Such changes in diet, suggest Taylor and Thoma (1985: 153), are usually associated with increased salt intake, both from purchased salt and from highly salted foods.

Although this literature review makes no clear link between diet and heart disease in indigenous people, the evidence presented does indicate that acculturation can greatly alter dietary habits. Overall, there seems to be a shift away from traditional bush foods to prepackaged store-bought foods which are higher in salt, fat, and refined carbohydrates and lower in dietary fibre, all of which have been shown to be dietary risk factors of coronary heart disease.

2.5.8 Physical inactivity

Data on the physical fitness levels of indigenous people are very meagre. In Canada, Rode and Shephard (1984) observed that after ten years of exposure to a non-traditional way of life, 344 male and female Inuit (aged 9 to 76 years) of Igloolik had experienced a 15 per cent decrease in predicted volume of oxygen intake; a two to four kilogram increase in body mass; an accumulation of subcutaneous fat; and a decrease in leg extension strength. In New Zealand, data on levels of physical fitness are negligible, although anecdotal evidence suggests that the proportion of adults now engaging in regular physical activity is increasing. What proportion of these exercisers are Maori is unknown but it is likely to be small, suggest Pomare and de Boer (1988: 154), as exercising is more a middle-class activity. Information on Maori fitness will probably become widely known as results from the Hillary Commission (1990) survey on the lifestyles of New Zealanders are published.

2.6 Summary

Evidence presented so far demonstrates that although Australian Aborigines, American and Canadian Indians, New Zealand Maoris, and Nauruans have similar patterns of high adult mortality, they are generally not characterized by similar life expectancies at birth and age- and cause-specific death rates. For example, Australian Aborigines, especially those from western New South Wales, have cardiovascular disease death rates two to three times higher than comparable Canadian Indian and New Zealand Maori rates. American Indian death rates from heart disease are also probably much lower than those of Australian Aborigines even though they cannot be directly compared. Existing but rather incomplete information on risk factor prevalence levels does not really explain why Australian Aborigines as compared to other indigenous groups have such high rates of death from circulatory system diseases, or why so many of these deaths occur at relatively young ages. However, this literature review does emphasize that certain risk factors, such as obesity, are more important than others, such as high cholesterol; and that other previously unrecognized risk factors, such as hyperinsulinaemia (a high insulin response), may be significant.

CHAPTER THREE

ABORIGINES IN WESTERN NEW SOUTH WALES

The word Aborigine is best used the way the dark people of southeastern Australia themselves use it: to include any person of Aboriginal ancestry. To be Aboriginal, in their own view, is to belong to, to be loyal to, the small closed community of kin and friends who are fellow Kooris, scattered enclaves in a world of whites.

(Barwick, 1988: 27)

3.1 Introduction and overview

Australian Aborigines are divided between those who live in remote and those who live in settled regions of the continent (Altman and Niewenhuysen, 1979: 101-102). In remote regions — the Northern Territory and northern and inland areas of South and Western Australia — Aborigines often form the majority of discernible population groups and live a more traditional-oriented lifestyle. By contrast, in settled regions — the Australian Capital Territory, New South Wales, Queensland, Victoria, and Tasmania (southeastern Australia), and coastal areas of South and Western Australia — Aborigines are usually in the minority and live a non-traditional-oriented lifestyle in or on the fringes of rural towns or urban centres. The concern in this study is with those Aborigines who live in settled regions, especially those living in southeastern Australia.

Of particular interest to this study are those Aborigines living in western New South Wales. My involvement with the study grew out of Aboriginal mortality research done in this region which demonstrated that coronary heart disease is a major cause of death (Gray and Hogg, 1989). In an attempt to understand why this is so, the present study re-examines regional Aboriginal mortality levels and estimates coronary heart disease risk factor prevalence levels in one community. The purpose of this chapter is to describe the setting for the study and to examine some of the underlying demographic and social-cultural characteristics of Aborigines in western New South Wales: Aboriginal identity,

family and household formation, employment opportunities, labour force participation and occupation, and income levels. The following four analytical chapters examine Aboriginal mortality patterns in this region and the biological and behavioural risk factor prevalence levels of Aborigines in Bourke.

3.2 The setting

Western New South Wales encompasses a number of geographical regions within the state including the central west, far west, and northwest. The study area, which is approximately 200,000 square kilometres in area, is defined by Goodooga, Lightning Ridge, and Moree to the north, by Murrin Bridge, Lake Cargelligo and Condobolin to the south, by Moree, Narrabri, and Wellington to the east, and by Wilcannia and Menindee to the west (see Figure 3.1). In total, the study focuses on 19 communities¹ which vary from medium sized urban centres, like Dubbo and Wellington, where a small proportion of the total population is Aboriginal, to small and medium sized country towns, like Bourke, Brewarrina, Moree, and Walgett, where a much larger proportion of the total population is Aboriginal.

Although the region's terrain is varied, from mountain ranges in the southeast to a vast, flat, semi-arid plain in the northwest, the rivers remain a common feature throughout. These rivers, including the Darling, Castlereagh, Lachlan, and Macquarie, are part of the westward flowing Murray-Darling river system that in prehistoric times was the lifeline to Aboriginal existence (see Dargin, 1976). This river system is the longest in Australia and drains part of Queensland, most of New South Wales, and a substantial part of Victoria before finally flowing into the sea on the eastern side of the South Australian coast. Its rivers provide irrigation to a rich agricultural and pastoral area.

1. These communities were selected for the study because in each case the enumeration of Aboriginal deaths was believed to be complete (see section 4.2 in chapter 4). They include: Bourke, Brewarrina, Collarenebri, Condobolin, Coonamble, Dubbo, Gilgandra, Gingie, Gulargambone, Goodooga, Lake Cargelligo, Lightning Ridge, Menindee, Moree, Murrin Bridge, Narrabri, Walgett, Wellington, and Wilcannia.

The largest concentrations of Aboriginal people in western New South Wales live in communities along these rivers and probably always have. Other families were moved, or drifted, from small remote settlements to live on the riverbanks, missions, housing settlements or, more recently, to live in the town areas (see Beckett, 1958a, 1958b; Long, 1970; Kamien, 1975b). Currently, country towns along the Darling River and its tributaries, like Bourke, Brewarrina, Walgett, and Wilcannia, all have large Aboriginal populations.

The 1986 census of population and housing estimates the Aboriginal population in the 19 study communities to be approximately 9,300 — 4,600 males and 4,700 females (see Table 3.1). This figure represents over 16 per cent of the total New South Wales^o Aboriginal population in 1986, or 30 per cent of the state's Aboriginal population living in rural and urban communities of between 200 and 100,000 persons.

The age structure of this population is relatively young. Approximately 45 per cent of the Aboriginal people living in these communities are less than 15 years of age and under 5 per cent are 65 years of age and over. This compares with the total New South Wales population in which 21 per cent are less than 15 years of age and over 10 per cent are 65 years of age and over (see Table 3.2). These substantial differences in age structure can be easily explained by the fact that Aboriginal fertility and mortality rates in this region are much higher than those experienced by the total New South Wales population (see section 4.3 in chapter 4).

There is also a marked imbalance in the sex structure of these two populations — they both have a deficit of males (see Table 3.2). For every 100 females in the Aboriginal and total New South Wales populations there are 98 and 99 males respectively. In the Aboriginal population this imbalance becomes more marked at 40 years of age and over, while in the total New South Wales population it is not noticeable until 60 years of age and over. However, regardless of when the imbalance occurs, in both populations it is due to differences in age-specific mortality rates between the sexes.

Figure 3.1 A map of western New South Wales showing the locations mentioned in this study.

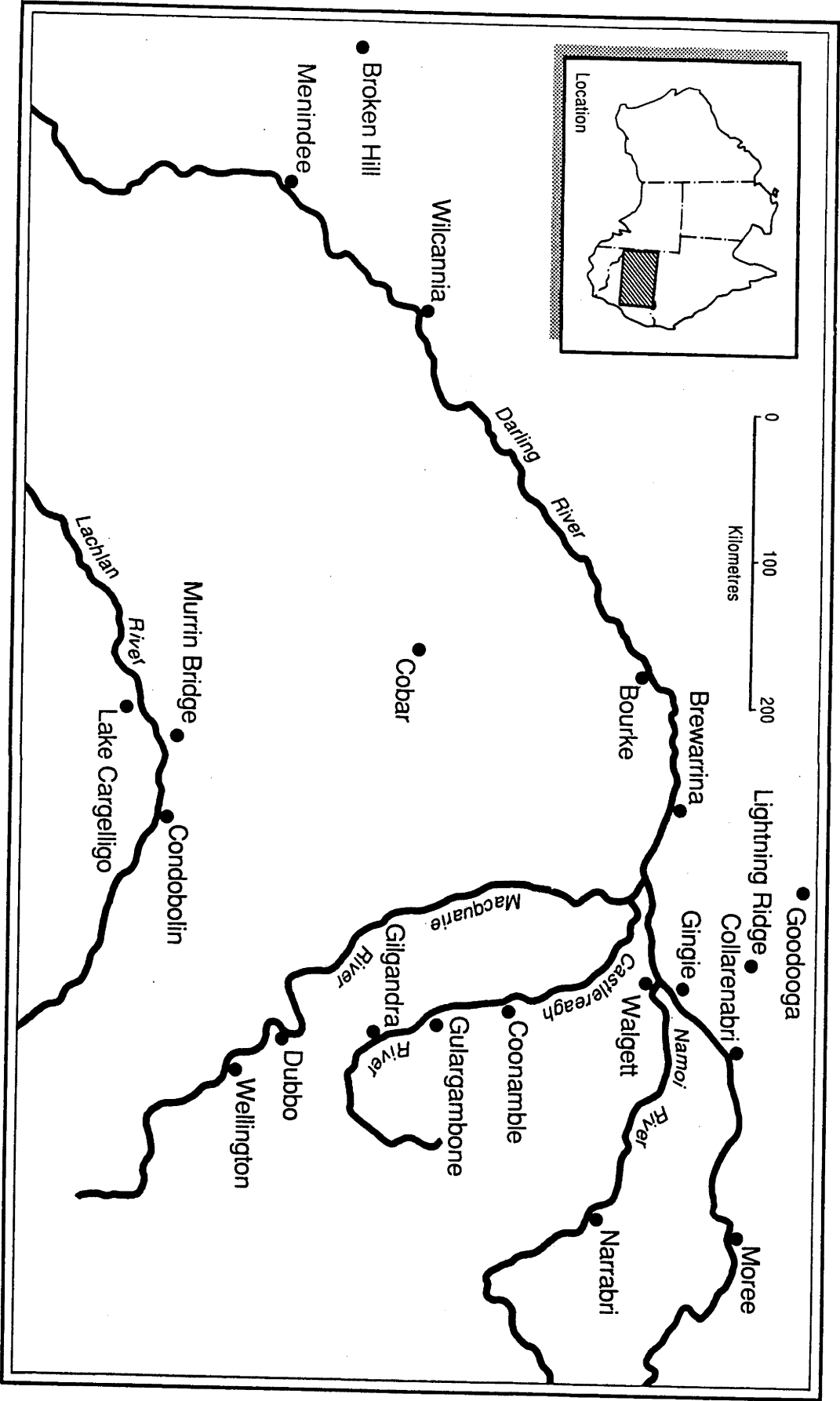


Table 3.1 Estimated number of Aborigines living in and around 19 western New South Wales communities¹, 30 June 1986, by sex and age.

Age group	Males	Females
0-4	694	669
5-9	609	600
10-14	639	641
15-19	626	629
20-24	448	487
25-29	353	390
30-34	270	272
35-39	251	232
40-44	192	206
45-49	160	171
50-54	129	132
55-59	76	97
60-64	45	75
65 plus	101	108
All ages	4,593	4,709

1. Source: Australian Bureau of Statistics 1986 census of population and housing. The communities include: Bourke, Brewarrina, Collarenebri, Condobolin, Coonamble, Dubbo, Gilgandra, Gingie, Gulargambone, Goodooga, Lake Cargelligo, Lightning Ridge, Menindee, Moree, Murrin Bridge, Narrabri, Walgett, Wellington, and Wilcannia.

Table 3.2 Comparison of the age and sex structure of the Aboriginal population in western New South Wales¹ and the total New South Wales population².

Age group	Aboriginal		New South Wales	
	Males (per cent) ³	Females (per cent) ³	Males (per cent) ³	Females (per cent) ³
0-4	15.0	14.1	7.7	7.3
5-9	13.2	12.7	7.5	7.1
10-14	13.8	13.6	8.2	7.8
15-19	13.5	13.3	8.2	7.8
20-24	9.7	10.3	8.3	7.9
25-29	7.7	8.3	8.4	8.2
30-34	5.9	5.8	7.9	7.8
35-39	5.5	4.9	8.0	7.7
40-44	4.2	4.4	6.6	6.2
45-49	3.5	3.6	5.5	5.2
50-54	2.9	2.8	4.8	4.6
55-59	1.7	2.1	5.0	4.8
60-64	1.0	1.6	4.6	4.8
65 plus	2.5	2.5	9.1	12.8
Total	100	100	100	100
Total population	4,593	4,709	2,756,990	2,774,536
Sex ratio ⁴	97.5		99.3	

1. Based on the estimated Aboriginal population living in and around 19 western New South Wales communities, 30 June 1986; source: Australian Bureau of Statistics 1986 census of population and housing.

2. Source: Australian Bureau of Statistics (1987b).

3. Percentages may not add up because of rounding.

4. The sex ratio is calculated by dividing the total number of males by females and is expressed as males per 100 females.

3.3 Social and demographic characteristics

Until recently social scientists have expressed little interest in studying the Aboriginal way of life in southeastern Australia. In western New South Wales, only a few intensive social-cultural or historical anthropological studies have been conducted and most of these were prior to the mid-1960s (see Reay, 1945, 1949; Reay and Sitlington, 1948; Fink, 1955, 1957; Beckett, 1958a, 1964; Bell, 1964; Kitaoji, 1976; Mathews, 1977; Goodall, 1982; Gray, 1984b; Cunneen and Robb, 1987; Cowlshaw, 1988; Read, 1988). The research that was done, especially the earlier studies, was often marred by the fact that anthropologists and other social scientists thought of non-traditional-oriented Aborigines as having no distinctive culture. In fact, anthropologists who did fieldwork among non-traditional-oriented Aboriginal groups, it has been said, were often considered by their colleagues to be apprentices — practicing for real anthropological work (Cowlshaw, 1988: 280).

3.3.1 Aboriginal identity

The modern Aboriginal subculture in western New South Wales, as in other parts of southeastern Australia, is based upon its members having a strong emotional allegiance to their kin and to the land, to the region of their forebears, and is a composite of both contemporary and traditional elements. In this cultural context, Aborigines define themselves not just in terms of genetic inheritance, but also in terms of a common set of beliefs, reciprocal obligations, and values. As Barwick (1988: 27) states:

Members of each regional population are knit together by reciprocal obligations of hospitality and help, by lifelong bonds of affection, duty and loyalty to the relatives and friends who alone provide complete acceptance and security in a seemingly hostile world. To publicly identify oneself as an Aborigine is a matter of defiant pride for many who could readily 'pass' and be lost as anonymous citizens. Their refusal 'to be ashamed of our blood and our people' is an explicit demonstration of allegiance to a small community bonded by shared experience, common memories and inherited legends of oppression as a despised indigenous minority.

Today, these people identify themselves as being either 'Aborigines', 'dark people', 'coloured', or 'blacks'; and employ a variety of regional labels, such as 'Murrie' and

'Koori', and tribal or language names, like Wiradujuri and Gooreng Gooreng, to mark regional and cultural differences (Keen, 1988: 3).

This Aboriginal identity is fostered by a number of internal and external mechanisms which engender group cohesion. Internally, Aboriginal solidarity is maintained through enforced allegiance to the group's ways and beliefs, and to the elders from whom these values are obtained. Those who detach themselves from the group are 'shamed' — punished through gossip and public humiliation (Barwick, 1988: 28).

Externally, prejudice and disapproval of Aborigines serve to reinforce their cultural beliefs and identity. Cowlshaw describes this as 'oppositional culture' and states (1988: 233-234) about one western New South Wales community:

To describe Aboriginal social life . . . as oppositional culture is to accept that certain of its features are an immediate trigger to white hostility. Rather than being accorded dignity, many Aboriginal practices . . . are disliked, disdained or pitied. Were Aborigines passive and silent in the face of such judgements there would be little need for the vilification to continue. There would be no point in continuing to actively dominate a population that had accepted subordination. Rather than showing shame, oppositional culture acts as both a challenge to those who despise Aborigines, as well as a defence against them.

In this sense, the division between Aborigines and non-Aborigines is based on the premise that there are two different kinds of people — Aborigines and whites. Despite the arbitrary nature and imaginary quality of this racial dichotomy, the dividing line is kept in place by a whole series of processes which operate to reinforce each other (see Kitaoji, 1976: VII 41-43; Cowlshaw, 1988). For example, people in one group generally do not even make casual contact with people in the other except for institutionalized interaction, such as contact through government services, hospitals and other health services, and schools. These processes help to enforce group cohesion and identity and to create a situation of mutual distrust, fear, and racial tension.

3.3.2 Family and household formation

Descriptions of non-traditional-oriented Aborigines have often dealt with family and household formation in a cursory fashion. This treatment, Gray (1985: 140) suggests, has helped to perpetuate the myth that the functioning of the contemporary Aboriginal family in settled Australia is disorderly and unstable. However, contrary to this myth, the integrity of existing Aboriginal family roles and values is sound and has evolved through the continued resistance and adaptation to a hostile social-cultural environment.

In southeastern Australia, kinship is a major determinant of contemporary Aboriginal social organization — their behaviours, values, and ideas (Kitaoji, 1976: VII 11).

Kinship is used as a means of classifying a regional network of relatives and friends.

As Barwick (1974: 154-155) states:

... kinship terms, like 'sis', 'brother', and 'cousin', are used incessantly in daily conversation as symbols of group membership. They can also be used deliberately as 'passwords among the dark people' when soliciting favours from distant kinsmen or mere acquaintances, or when wishing to insinuate that the persons addressed are 'getting too proud and ought to remember they are Aborigines'.

In this sense, kinship networks are formed on the basis of biological, social, and marital relations. However, the actual levels of interaction and intimacy between kin are determined not only through the closeness of kinship ties and marital relations, but also through geographic proximity, living arrangements, and personal attachments (Kitaoji, 1976: VII 12-14; Beckett, 1988).

Within this context, Aboriginal family and household formation is an orderly process which has adapted to a variety of cultural and situational constraints. The most outstanding feature of Aboriginal family formation is the number of different types of sexual unions that partners can enter — such as casual liaisons, *de facto* unions, and legal marriage. As a rule, partners enter these unions in a logically ordered sequence. As Gray (1984b: 385) states:

... casual liaison and pregnancy usually precedes the first consensual marriage, which may or may not be permanent, and consensual marriage usually precedes legal union if the marriage is legalized at all. Childbirth does not necessarily result in the formation of a consensual union between the parents, and it is not always a necessary condition.

Casual liaisons are more typical of adolescents; and *de facto*, common law, unions tend to be more unstable than marriage. However, Aborigines generally do not distinguish between long-lived *de facto* unions and marriage. Barwick (1974: 161) states that enduring unions from which several children have been born have a status equal to marriage.

The transition from a *de facto* union to marriage is dependent on the stability of the union and on a variety of other social factors. The most important factor is the man's occupational status or income. Additionally, factors such as the place of residence, house type, education, and religion can play a role in this transition (Kitaoji, 1976: VII 15-16). Marriage may also be regulated by certain traditional considerations. Beliefs still operate to forbid first and second cousin marriages and to confine marriage choices to a particular group of kin or locality (Barwick, 1988: 29).

In comparison with the New South Wales population, figures from the 1986 census suggest that a greater proportion of adult Aborigines in western New South Wales are never married: 67 and 57 per cent of Aboriginal males and females respectively, compared with 33 per cent of the males and 24 per cent of the females in the total New South Wales population (see Table 3.3). However, what this demonstrates is that Aborigines tend to remain in casual liaisons and *de facto* unions rather than becoming legally married. Although the numbers of formerly married people are similar in the two populations, in the Aboriginal population most people included in this category are widowed, especially women, while in the total New South Wales population most are separated or divorced. As a rule the larger proportion of widowed Aborigines is due to the higher levels of adult mortality.

An Aboriginal household most often consists of a group of kin, usually consisting of a household head with or without a spouse, the head's own, step, or adopted children, the adult children's partners, and other coresident members, who share a common dwelling and all its facilities. Men typically have nominal status as household heads, but older women are the effective managers and usually decide who may join the household. Overall, the structure of an Aboriginal household tends to be matrifocal (Barwick, 1974: 156-157; Kitaoji, 1976: VII 18).

The composition of a household can change rapidly as temporary visitors come and go (Barwick, 1974: 156-157). Notions of right conduct and decent behaviour require household heads to give help and accommodation to any relative who asks, especially those with young children. These obligations are seen as reciprocal and help to cement ties between networks of friends and relatives from the same region.

Because dwellings which are constructed for Aborigines are generally built to better suit the functioning of a nuclear household than a large Aboriginal composite household, overcrowding is a major problem. For example, a recent survey of household conditions on the north coast of New South Wales observed that deficiencies in the size of dwellings, such the number of bedrooms or toilets, are exacerbated by problems of overcrowding (Gray 1987b; Gray and Vesper, 1987). Furthermore, the steady growth of the Aboriginal population in this region will continue to maintain excessive pressure on housing stock.

Although there has been a reduction in the overall size of Aboriginal households in New South Wales during the past three decades (Rowley, 1982: 27), Aboriginal households in western New South Wales are still much larger than those in the total New South Wales population. In 1986, the proportion of Aborigines living in households of six or more people was over four times higher than in the total New South Wales population — 52 versus 12 per cent (see Table 3.4).

Table 3.3 Comparison of the marital status of the Aboriginal population in western New South Wales¹ and the total New South Wales population², by sex and age, for persons aged 15 years and over.

Age group	Aboriginal			New South Wales		
	Never Married (per cent in each age group) ³	Married	Formerly Married (per cent in each age group) ³	Never Married (per cent in each age group) ³	Married	Formerly Married (per cent in each age group) ³
Males						
15-19	98	1	1	100	0	0
20-29	84	15	2	66	31	3
30-39	51	38	11	18	72	10
40-49	31	50	19	9	80	12
50-59	34	47	19	9	79	12
60 plus	19	46	35	7	75	18
All ages	67	25	8	33	58	9
Total ⁴	1,755	657	224	667,109	1,188,323	191,566
Females						
15-19	97	2	1	98	2	0
20-29	76	18	7	46	48	6
30-39	41	42	17	10	72	17
40-49	19	53	28	5	79	16
50-59	19	42	40	4	75	20
60 plus	8	35	56	6	47	47
All ages	57	27	16	24	56	20
Total ⁴	1,626	756	462	510,493	1,190,753	430,014

1. Based on the estimated Aboriginal population living in and around 19 western New South Wales communities, 30 June 1986.

2. Source: Australian Bureau of Statistics 1986 census of population and housing.

3. Percentages refer to an age group and may not add up because of rounding.

4. Total number of individuals in each category.

Table 3.4 Comparison of the Aboriginal population in western New South Wales¹ and the total New South Wales population², by household size³.

Household size (members)	Aboriginal		New South Wales	
	Number of members	Per cent ⁴	Number of members	Per cent ⁴
1	141	2	360,053	7
2	433	5	1,087,646	21
3	941	10	926,496	18
4	1,496	16	1,324,368	26
5	1,458	16	857,670	17
6	1,298	14	378,594	7
7	1,138	12	140,805	3
8 or more	2,493	26	104,616	2
Total	9,398	100	5,189,248	100

1. Based on the estimated Aboriginal population living in and around 19 western New South Wales communities, 30 June 1986.

2. Source: Australian Bureau of Statistics 1986 census of population and housing.

3. Excludes households living in caravan parks and persons usually resident but absent on census night; and includes persons not usually resident but present on census night.

4. Percentages may not add up because of rounding.

3.3.3 Economic activity and Aboriginal employment opportunities

Economic activity in the western region of New South Wales is based mainly on agricultural industries and mining. In the 1986-1987 growing season, the region's agricultural industries accounted for about 18 per cent or \$860 million of the total gross value of the agricultural commodities produced in the state (Australian Bureau of Statistics, 1988a). During that season, hay and cereal crops like wheat, oats, and barley were grown in most local government areas; citrus fruits were grown in the Bourke, Brewarrina and Wellington shires; and cotton, which accounted for over 80 per cent of the total production in New South Wales, was grown in the Bourke, Narrabri, and Moree plains shires. Well over 9,000,000 sheep and lambs were shorn; 500,000 beef cattle were produced, while less than 1,000 cows were used for milk production; and pigs were produced mainly in the eastern and northeastern local government areas. Another important industry in the region is mining; however, most of the larger and more extensive operations exist in communities such as Broken Hill and Cobar which have been excluded from this study. Finally, country towns in the region are often service centres for retail trade and local, state, and commonwealth government agencies.

The employment opportunities for Aborigines in the region are limited. Their access to employment on agricultural properties has declined in the last few decades because of increasing labour costs, reduced profits, and the move towards a greater mechanisation of agricultural techniques (Castle, 1987: 62; Cass, 1988: 241; Cowlshaw, 1988: 119; Ross, 1988: 1). In addition, virtually no jobs are available to them in the private sector — in shops or small businesses. Small businesses are the norm in country towns and are most often family operated; self-employment is much more common here than in Australia as a whole. Any jobs that are available are usually filled by a word-of-mouth process which tends to exclude Aborigines (Castle, 1987: vi). Increasingly, therefore, the major source of employment for Aborigines in this region and other rural areas of New South Wales is the public sector (Castle, 1987: 62). Commonwealth government-

sponsored services, such as the Aboriginal Health Services, and existing social service networks, such as the Department of Social Security, offer limited employment opportunities. Other public sector positions include support staff in schools, nursing and domestic staff in hospitals, and labourers in shire councils. Most of these positions have been recently created and are designated for Aborigines, in areas where 'Aboriginalisation' is policy (such as the Department of Aboriginal Affairs), or are traineeships of a set duration (Cowlshaw, 1988: 127).

The lack of employment opportunities for Aborigines is also compounded by the fact that Aborigines have low levels of formal education. As Cass (1988: 245) states:

The prospects for long-term improvement in the position of Aboriginal people in the labour market remain poor unless there are substantial improvements in their level and standard of schooling. These low levels of education and training have obvious implications for Aboriginal access to employment and for the nature of that employment.

Although very limited data are available to assess education and skill levels in western New South Wales, several broad-based studies have highlighted numerous ways in which Aborigines throughout the state are educationally disadvantaged. In Australia as a whole, 1986 census figures suggest that there are much lower levels of education among Aborigines of working age than among the general Australian population (see Cass, 1988: 245). Approximately 11 per cent of the Aboriginal working age population have never attended school in contrast to less than 1 per cent of the general Australian working age population. A much smaller proportion of Aboriginal people continued their schooling beyond 15 years of age and an even larger disparity exists in the proportion who have tertiary qualifications. Only 4 per cent of the Aboriginal working age population have some form of tertiary education compared with 24 per cent of the general Australian population. Similarly, in non-metropolitan areas of New South Wales, Young (1982: 16) noted that while all Aboriginal children between 5 and 14 years of age were attending school, the proportion of them between 15 and 19 years of age who were still at school had dropped to 35 per cent. She also found that almost 30 per cent of Aboriginal adults had only a primary education and that only 4 per cent had

more than four years of secondary education. Aborigines with more education tend to fare better in the labour force. Another study (Ross, 1988) in non-metropolitan areas of New South Wales observed that Aboriginal individuals with at least a Year 11 education tend to have significantly higher employment levels and lower unemployment levels than the Aboriginal population as a whole. However, apart from the few who have sufficient levels of formal education to get jobs, the employment prospects for the majority of Aborigines are not good (see Castle, 1987).

3.3.4 Labour force participation and occupation

In comparison with the total New South Wales population, Aboriginal labour force participation (defined as those who are working and those who are not working but seeking employment) and employment rates in western New South Wales are rather low. As indicated by the 1986 census, only 62 per cent of men and 33 per cent of women over 15 years of age are in the labour force; however, less than half of these men and women are employed — 44 and 49 per cent respectively. These figures compare with the total New South Wales population where 73 per cent of men and 46 per cent of women are in the labour force; and the majority of them are employed — 90 per cent of both men and women (see Table 3.5).

There are also noteworthy age-specific disparities between the Aboriginal labour force participation and employment rates and those of the total New South Wales population. As shown in Table 3.5, Aboriginal labour force participation decreases sharply after the 20 to 29 year old age group, and the proportion of employed Aborigines increases with age as the labour force gets smaller. Although total New South Wales age-specific rates follow a similar pattern, both rates are much higher at all ages than those for Aborigines.

Aborigines in the region are principally employed in unskilled or semi-skilled occupations (see Table 3.6). Over 75 per cent of the men who are employed work as labourers, tradespersons, or machine operators; while over three-quarters of the women

who are employed work as clerks, labourers, or in the personal service. Furthermore, only 9 per cent of employed males and 10 per cent of employed females are working in professional or semi-professional occupations, which compares with 32 per cent of employed males and 26 per cent of employed females in the total New South Wales population.

Although Table 3.5 does not compare regional Aboriginal and non-Aboriginal labour force participation rates², it does highlight some basic disparities in these rates between the regional Aboriginal and total New South Wales populations. In particular, compared with the total New South Wales population, the proportion of Aborigines not in the labour force and unemployed is very high. Unemployment rates among both men and women are over 50 per cent of the potential work force (although they are much higher if one considers those who are not in the labour force). Additionally, there are increasing economic and social divisions between those Aborigines who have secure full-time work, a small minority, and those who have part-time work or no work at all, the majority. This is a major problem, because the potential benefits of full-time employment — economic independence and better health brought about by improved health care attitudes and opportunities (see Verbrugge, 1983) — can be expected to benefit only a small minority of individuals.

2. I was unable to find a comparable set of data for non-Aborigines in this region.

Table 3.5 Comparison of the labour force participation rates of the Aboriginal population in western New South Wales¹ and the total New South Wales population², by sex and age, for persons aged 15 years and over.

Age group	Aboriginal			New South Wales		
	Employed (per cent) ³	Unemployed (per cent) ⁴	In labour force (per cent) ⁵	Employed (per cent) ³	Unemployed (per cent) ⁴	In labour force (per cent) ⁵
Males						
15-19	28	72	48	78	22	51
20-29	42	58	79	87	13	90
30-39	50	50	74	92	8	93
40-49	54	46	63	94	6	91
50-59	53	47	41	93	7	82
60 plus	61	39	12	91	9	22
All ages	44	56	62	90	10	73
Total ⁶	718	928	1,646	1,349,311	148,318	1,497,629
Females						
15-19	24	76	38	78	22	48
20-29	47	53	40	88	12	67
30-39	65	35	35	91	9	60
40-49	69	31	30	93	7	62
50-59	73	27	16	94	6	40
60 plus	100	0	4	96	4	6
All ages	49	51	33	90	10	46
Total ⁶	451	467	918	871,338	100,820	972,158

1. Based on the estimated Aboriginal population living in and around 19 western New South Wales communities, 30 June 1986.

2. Source: Australian Bureau of Statistics 1986 census of population and housing.

3. Proportion in the labour force who are employed.

4. Proportion in the labour force who are unemployed.

5. Proportion who are in the labour force (employed and unemployed).

6. Total number of individuals in each category.

Table 3.6 Comparison of the occupational status of employed Aborigines in western New South Wales¹ and employed persons in the total New South Wales population², by sex, for persons aged 15 years and over.

Occupation	Aboriginal		New South Wales	
	Males (per cent) ³	Females (per cent) ³	Males (per cent) ³	Females (per cent) ³
Managers and administrators	1	1	14	7
Professionals	4	4	12	12
Para-professionals	4	5	6	7
Tradespersons	25	6	22	4
Clerks and salespersons	7	33	8	34
Personal service workers	2	21	8	18
Plant and machine operators	15	2	11	3
Labourers	36	22	16	12
Not stated ⁴	5	6	3	2
Total	100	100	100	100
Total number employed	704	472	1,349,309	871,338

1. Based on the estimated Aboriginal population living in and around 19 western New South Wales communities, 30 June 1986.

2. Source: Australian Bureau of Statistics 1986 census of population and housing.

3. Percentages may not add up because of rounding.

4. Insufficient information or not stated.

3.3.5 Income levels

Income for the support of Aboriginal families comes mainly from two sources: wages and salaries earned through employment; and pensions and benefits paid through the Department of Social Security (Young, 1982). However, because so few Aborigines have regular paid employment, there is a high level of dependency on social security payments. As Cowlshaw (1988: 203) notes about one community in western New South Wales:

In my experience virtually all [Aborigines] on the reserve and the majority at the Aboriginal end of town do not have jobs but rely on the income of those members of the family who receive pensions or unemployment benefits. It is very common for no one in a large household to be in regular paid employment.

Those Aborigines who do have paid employment are generally working in jobs characterized by low pay. A recent survey in non-metropolitan areas of New South Wales noted that gross average weekly earnings (before taxes) for those in full-time employment were approximately \$235 for males and \$225 for females; while for those in part-time employment estimates of gross average weekly earnings were \$175 for males and \$210 for females (Ross, 1988). The small difference between part-time and full-time earnings is in part due to the fact that the lack of job security and non-monetary benefits associated with part-time employment is compensated for by higher hourly wages.

Annual income levels are also very low. For example, compared with the 1986 total New South Wales population, Aborigines in western New South Wales are much more likely to have annual incomes under \$9,001 and much less likely to have annual incomes above \$22,000 (see Table 3.7). The difference between the two populations is much greater among males than females. Aboriginal males are two times more likely to have incomes under \$9,001 and substantially less likely to have incomes over \$22,000 than males in the total New South Wales population. Although this comparison is rather crude, it does help to highlight the fact that Aborigines in this region have very low individual incomes.

Table 3.7 Comparison of individual income levels of the Aboriginal population in western New South Wales¹ and the total New South Wales population², by sex, for persons aged 15 years and over.

Individual income (annual)	Aboriginal		New South Wales	
	Males (per cent) ³	Females (per cent) ³	Males (per cent) ³	Females (per cent) ³
\$0 - \$9,000	64	78	32	64
\$9,001 - \$15,000	23	16	17	17
\$15,001 - \$22,000	11	5	26	13
\$22,000 - 32,000	2	1	16	5
\$ 32,001 plus	0	0	9	1
Total	100	100	100	100
Total number of individuals ⁴	2,224	2,324	1,937,078	1,957,586

1. Based on the estimated Aboriginal population living in and around 19 western New South Wales communities, 30 June 1986.

2. Source: Australian Bureau of Statistics 1986 census of population and housing.

3. Percentages may not add up because of rounding.

4. Excluding not stated.

3.4 Summary

This chapter has briefly described the setting for the study and has examined some of the underlying demographic and social-cultural characteristics of Aborigines in western New South Wales. In particular, aspects of Aboriginal identity, family and household formation, employment opportunities, labour force participation and occupation, and income levels have been examined. Although these descriptions are rather brief, they do highlight some basic ways in which the contemporary way of life of Aboriginal people is different from that of other Australians.

CHAPTER FOUR

ABORIGINAL MORTALITY IN WESTERN NEW SOUTH WALES

4.1 Introduction and overview

Only two major studies¹ of Aboriginal mortality in the country areas of New South Wales have been undertaken in the past ten years — both were conducted for the Aboriginal Health Unit of the New South Wales Department of Health. The first study noted that for all country areas, the relative risk of death for male and female Aborigines in 1980-1981 was four times higher than that of the New South Wales population in 1981, with the relative risk of death in middle adulthood being up to 12 times higher (Julienne *et al.*, 1983; Smith *et al.*, 1983; Thomson and Smith, 1985). The second study observed that for western New South Wales in particular, Aboriginal sex-specific death rates from 1984 to 1987 were three to four times higher than those of the New South Wales population in 1986, with the relative risk of death in middle adulthood being at least five times higher (Gray and Hogg, 1989). Both studies have drawn attention to the acute level of adult mortality endured by Aborigines in the country areas of New South Wales, especially during middle adulthood.

Using mortality data from both of these studies as well as from other sources, I have attempted to evaluate the level of contemporary Aboriginal mortality in the western region of New South Wales by making a series of comparisons. In particular, this assessment reviews the quality of available sources of data and compares current mortality trends at regional, community, and individual levels. At a regional level, comparisons are made between: Aboriginal and total New South Wales mortality rates and causes of death; Aboriginal mortality trends from 1981-1983 to 1984-1987; and

1. Another minor study was conducted by the New South Wales Health Commission, Aboriginal Policy Committee (1979).

Aboriginal mortality rates and those of other Australian- and overseas-born birthplace groups. At a community level, a comparison is made between Aboriginal mortality rates and demographic and social-cultural conditions. At an individual level, a comparison is made between the observed and expected number of joint occurrences of various disease conditions which were mentioned as adult Aboriginal causes of death.

4.2 Sources of mortality data

The mortality data used in this study were compiled from three state-wide data bases, and include the listings of deaths produced from the two previous Aboriginal Health Unit studies. Individually these sources of data are incomplete, but together they provide a reliable body of information on most Aboriginal and some non-Aboriginal deaths which have occurred in selected western New South Wales communities for varying periods of time during the 1980s.

The first and most substantial source of data on Aboriginal and non-Aboriginal deaths was the records held by local registry offices throughout the state. This information was gathered by visiting registries, which are located in small and medium sized communities and in larger centres such as Broken Hill and Sydney, and searching through the records. Aboriginal informants were employed to identify the records on Aboriginal deaths and sometimes to supplement these records with additional background information. In particular, informants were asked questions about whether the deceased: was single, married or in a *de facto* union, divorced, or widowed; was predeceased by parents, spouse, and/or children; owned or rented his or her house or flat; had a job in the twelve months previous to death; and lived in town or on a mission (former reserve).

Although the listing of most Aboriginal and non-Aboriginal deaths was compiled from registry records, two other sources of mortality data were used. This additional data was necessary because the completeness of the listing obtained from local registry

records was often difficult to determine. In the past, country registries in New South Wales were required to record the deaths of individuals who died locally or who were buried locally after dying elsewhere. Now, however, with the computerization of the New South Wales Principal Registrar's central data base in Sydney, this registration requirement has been relaxed (Gray and Hogg, 1989).

The first supplementary source of data used was the yearly listing of Aboriginal deaths produced by the New South Wales Principal Registrar. These listings have been produced since 1980 and are generated from the question on the death record notification form (which is completed by the funeral director) asking whether the deceased was considered to be Aboriginal. The main drawback of this data source is that a large proportion of Aboriginal deaths are missed because they are recorded as non-Aboriginal (Gray, 1983). Gray and Hogg (1989) observed that one in three Aboriginal deaths occurring in selected western New South Wales communities between 1984 and 1987 were not found on these yearly listings, and this was an area of good reporting. As noted in chapter 2, a similar situation arises in New Zealand (see section 2.2.4).

The second supplementary source of data used was the system put in place by the Aboriginal Health Unit in 1978 whereby health workers complete notification forms for all Aboriginal deaths occurring in their health district. The basic details found on these forms are then forwarded to regional and central offices. Unfortunately this listing contained few of the actual number of Aboriginal deaths which occurred in western New South Wales; however, it did contain a few deaths that were not found in the other two sources.

In total, information was recorded on 673 Aboriginal and 319 non-Aboriginal deaths which occurred between 1979 and 1988 (a copy of the coding sheet can be found in Appendix 2). This data set included many deaths which occurred outside the study area or in communities where the enumeration of deaths was believed to be incomplete.

Therefore, instead of basing estimates of Aboriginal and non-Aboriginal mortality on the total number of deaths, calculations were restricted to three overlapping data subsets made up of deceased individuals from communities where the enumeration of deaths was believed to be complete. The first subset consists of 315 Aboriginal deaths (205 male and 110 female) which occurred in 19 communities² between 1984 and 1987. The second subset consists of 408 Aboriginal deaths (258 male and 150 female) which occurred in Bourke, Brewarrina, Dubbo, Gilgandra, Gingie, Goodooga, Lightning Ridge, Moree, and Walgett between 1980 and 1987. The third subset consists of 171 Aboriginal (116 female and 55 male) and 279 non-Aboriginal (184 male and 95 female) deaths which occurred in Bourke and Brewarrina between 1979 and 1988. The first subset was used to examine recent regional and community Aboriginal mortality trends; the second to explore the changes in Aboriginal mortality rates between 1980-1983 and 1984-1987; and the third to compare community patterns of Aboriginal and non-Aboriginal mortality in Bourke and Brewarrina (as discussed in section 5.5.1 of chapter 5).

Although the enumeration of deaths in these three subsets is thought to be relatively complete, there still may be some degree of underenumeration, especially of non-Aboriginal deaths. For example, the number of deaths that were recorded in the Brewarrina and Bourke registry offices from 1979 to 1988 is about 10 per cent less than the number of deaths that were officially listed by the Principal Registrar (see Table 4.1). This apparent underenumeration could be due to deaths being registered in a year in which they did not occur or deaths of local residents being recorded elsewhere. The first type of error did not affect this study, because listings of deaths were based on the year of death and not the year of registration; however, the second type of error did have an effect on analysis as it disproportionately influenced the estimation of non-

2. These communities are Bourke, Brewarrina, Collarenebri, Condobolin, Coonamble, Dubbo, Gilgandra, Gingie, Gulargambone, Goodooga, Lake Cargelligo, Lightning Ridge, Menindee, Moree, Murrin Bridge, Narrabri, Walgett, Wellington, and Wilcannia.

Aboriginal mortality. This problem could not be rectified because there was a lack of supplementary data sources which could be compared with the local registry listings.

Estimates of the population at risk of death were obtained by applying a limited cohort-component method of reverse survival to 1986 census figures for various selected western New South Wales communities (Shryock and Siegel, 1971: 740-741). Using life table survivor ratios, this technique projected backwards the Aboriginal population living in and around the 19 study communities on 30 June 1986 to 31 December 1985 — the mid-point of the four-year study period for the first data subset. Other mid-point population estimates, using this reverse survival technique, were produced for the Aboriginal population living in the nine communities of the second data subset over two periods, 1980-1983 and 1984-1987; and for the Aboriginal and non-Aboriginal populations living in Bourke and Brewarrina from 1979 to 1988.

A reverse survival technique was used, as opposed to an intercensal survival technique, because of the inherent inaccuracies in the 1981 census figures. As indicated by the 1986 figures, and as already discussed in chapter 2 (see section 2.2.1), there was a large intercensal increase between 1981 and 1986 in the total number of Australian Aborigines. For Aborigines in New South Wales there was an increase of 91 per cent in major urban areas, 70 per cent in other urban areas, and 28 per cent in rural areas. Although these results are also indicative of a long-term trend towards increased urbanization, the very dramatic intercensal growth rate in the Aboriginal population is probably due to an increased propensity of people to record themselves and their households as being Aboriginal (Australian Bureau of Statistics, 1989a).

The coding of Aboriginal and non-Aboriginal underlying causes of death (the disease or injury initiating the sequence of events which led to death) was based on criteria outlined in the ninth revision of the Manual of the International Statistical Classification of Diseases, Injuries, and Causes of Death (World Health Organization, 1977). Most causes of Aboriginal death included in this study were already coded by the Principal

Registrar and were found on the yearly listings of Aboriginal deaths. However, for Aboriginal or non-Aboriginal deaths that were not recorded on the Principal Registrar's listings, the underlying cause of death was coded by myself in accordance with the World Health Organization's criteria.

To test the accuracy of my coding methods, a comparison was made of the causes of death coded by myself, as part of a survey of Aboriginal and non-Aboriginal deaths in Bourke and Brewarrina, and those coded by the Principal Registrar. In regards to this comparison, the Principal Registrar's coding was found on a list of causes of death for all deaths in Bourke and Brewarrina which was produced for the Australian Bureau of Statistics. As shown in Table 4.1, the most noticeable discrepancy was between the proportion recorded as dying from respiratory system diseases. However, this is not a true comparison because my survey of deaths included many Aboriginal deaths that were recorded on the Principal Registrar's yearly listings and for these I simply used the Principal Registrar's code for cause of death. I only coded those deaths that I compiled from registry records which were not recorded on the yearly listings — some Aboriginal deaths and all non-Aboriginal deaths.

Table 4.1 Comparison of survey¹ and registry² death records from Bourke and Brewarrina, 1979-1988, by sex, age, and major cause of death.

	Males		Females	
	Survey (per cent) ³	Registry (per cent) ³	Survey (per cent) ³	Registry (per cent) ³
Age group				
0	3	5	4	6
1-14	1	2	1	1
15-24	5	5	4	4
25-44	13	13	9	9
45-64	34	32	21	23
65 plus	44	43	60	57
All ages	100	100	100	100
Cause of death				
Infectious and parasitic	1	1	2	2
Malignant neoplasms	15	15	14	16
Circulatory system	46	46	49	48
Respiratory system	10	8	9	5
Congenital anomalies	0	1	2	2
Perinatal causes	1	2	2	2
Signs, symptoms and ill-defined conditions	2	2	1	1
Accidents, poisonings and violence	13	12	8	8
All other causes	11	12	13	15
All causes	100	100	100	100
Total cases ⁴	359	386	180	198

1. A survey of death registries in Bourke and Brewarrina conducted in February and March 1989.

2. Source: Mortality tabulations based on a short list of causes of death produced by the New South Wales Principal Registrar for the Australian Bureau of Statistics.

3. Percentages may not add up because of rounding.

4. Excluding a female death record from the Brewarrina Registry Office in which the underlying cause of death was not stated.

4.3 Comparisons at a regional level

As noted above, comparisons at a regional level are made between: Aboriginal and total New South Wales mortality rates and causes of death; Aboriginal mortality trends from 1981-1983 to 1984-1987; and Aboriginal mortality rates and those of other Australian- and overseas-born birthplace groups. The purpose of these regional comparisons is to assess whether Aboriginal mortality rates are different from those exhibited by other Australians and whether there have been noticeable changes in Aboriginal mortality trends during the 1980s.

4.3.1 Comparison with the total New South Wales population

Aboriginal mortality in western New South Wales from 1984 to 1987 is markedly higher than the level exhibited by the total New South Wales population in 1986. However, if only crude rates of mortality are compared, female Aborigines have a death rate which is markedly lower than that for the total New South Wales population. The Aboriginal crude death rate was 8.4 per 1,000, or 11.1 per 1,000 for males and 5.8 per 1,000 for females. This compares with a crude death rate for the total New South Wales of 7.6 per 1,000, or 8.2 per 1,000 for males and 7.1 per 1,000 for females (see Table 4.2).

The problem with comparing crude death rates is that the above rates have been seriously affected by the age structure of the two populations. As noted in chapter 3, the total New South Wales population has an older age structure than the Aboriginal population in this region (see Table 3.2). Therefore, the total New South Wales population, with a larger proportion of older people, will tend to have higher sex-specific crude death rates, simply because older people are at a much greater risk of death than younger people. As shown in Table 4.2, if the Aboriginal population had the same age structure as the total New South Wales population, its directly standardized

death rates³ would have been 19.5 per 1,000, or 24.0 per 1,000 for males and 15.1 per 1,000 for females. Put in these terms, the Aboriginal mortality rate for males is about three times and for females about two times the rate for their counterparts making up the total New South Wales population.

4.3.1.1 Age-specific death rates and life expectancy

These death rates have little meaning unless they are related to specific age groups. Therefore, to make these measures more meaningful, Aboriginal age-specific death rates were compared to rates for the total New South Wales population (Australian Bureau of Statistics, 1988b, 1988c). The calculations involved in this comparison took place in various stages. First, age intervals were grouped so as to limit the effects of small numbers. Second, age-specific relative risks, a ratio of the Aboriginal rate to the total New South Wales rate, were determined. Third, confidence intervals, at a 95 per cent level of confidence, were constructed around each ratio to provide a more reliable estimate of relative risk (Bailar and Ederer, 1964). However, in an additional effort to limit the effects of small numbers, relative risk ranges were only calculated for age-specific mortality rates based on five or more deaths.

The comparison between Aboriginal and total New South Wales age-specific death rates reveals a consistently higher relative risk of death experienced by both Aboriginal men and women at most age groups, but especially between the ages of 25 and 44 years (see Table 4.3). The lower confidence intervals of relative risk vary from about one to over five, and the upper intervals vary from two to nearly ten. Overall, this analysis suggests a pattern of gradually increasing relative risk until the age of 45 years, and then a progressive decline in relative risk with increasing age intervals.

3. In this chapter Aboriginal crude and cause-specific death rates have been directly standardized to the New South Wales age structure; and therefore they differ in magnitude from the indirectly standardized rates previously published in the report to the New South Wales Department of Health (Gray and Hogg, 1989).

Another important descriptive measure of overall mortality is life table analysis. This type of analysis provides a way of linking observable events to the consequences these events would have on a hypothetical cohort, free of the effects caused by demographic disturbances (Howell, 1979: 74). For each age interval these tables generally give information on: the mid-year population, P_x ; the number of observed deaths, D_x ; the age-specific death rate, ${}_nM_x$; the probability of dying, ${}_nq_x$; the number in a hypothetical cohort at that age interval, l_x ; the number of person-years lived by the hypothetical cohort in the interval, ${}_nL_x$; the number in the hypothetical cohort dying in the interval, d_x ; the total number of person-years lived by the hypothetical cohort beyond the age interval, T_x ; and life expectancy at that age interval, e_x° . However, for the purpose of brevity, only the information on P_x , D_x , ${}_nM_x$, ${}_nq_x$, l_x , and e_x° is included in the abridged life tables presented here (see Table 4.4). Additionally, a measure of standard error was constructed around each e_x° value for a closed age interval (see Chiang, 1977: 133-140, 1984:161-167). In this case, these estimates of standard error are used to assess the variability in life expectancy caused by small numbers.

Although life tables can provide a range of descriptive measures, the expectation of life is the one most commonly used. As shown in Table 4.4, the life expectancy at birth for Aboriginal males was 53 years and for females was approximately 66 years⁴. With the computation of standard error, life expectancy for either sex at birth and other ages was shown to vary by only about one year. However, Aboriginal men and women live on average 15 to 20 years less than men and women in the total New South Wales population — in 1986 the life expectancies at birth for men and women in New South Wales were 73 and 79 years respectively. The gap between Aboriginal male and female life expectancy is also remarkably large in comparison to the total New South Wales figures (13 versus 6 years). This gap occurs because Aboriginal males have much

4. Life expectancies reported in this chapter differ slightly from those published in the report to the New South Wales Department of Health (Gray and Hogg, 1989), because age intervals were further combined to reduce the effects of small numbers (see Table 4.4). However, in all cases these differences are not statistically different.

higher death rates than females at all ages, but especially at birth and between the ages of 25 and 64 years.

Table 4.2 Crude and standardized death rates¹ and the standardized mortality ratio² for Aborigines in western New South Wales³, 1984-1987, and the total New South Wales population⁴, 1986, by sex.

	Males	Females
Aboriginal, 1984-1987		
Total number of deaths	205	110
Crude death rate	11.1	5.8
Standardized death rate	24.0	15.1
New South Wales, 1986		
Total number of deaths	22,571	19,596
Crude death rate	8.2	7.1
Standardized mortality ratio		
Aboriginal standardized rate to New South Wales crude rate	292 (254 - 337)	215 (177 - 264)

1. Rates are deaths per 1,000 population per year. Aboriginal crude death rates were directly standardized to the New South Wales age structure, 30 June 1986.
2. A ratio of expected Aboriginal deaths to the total number of deaths in New South Wales in 1986. Confidence intervals, at a 95 per cent level of confidence, are designated by the ratios shown in brackets. The ratios have been multiplied by 100.
3. Based on mortality data for 19 western New South Wales communities (see section 4.2).
4. Source: Australian Bureau of Statistics (1988b).

Table 4.3 Age-specific mortality rates (ASMR)¹ and relative risk² for Aborigines in western New South Wales³, 1984-1987, and the total New South Wales population⁴, 1986, by sex.

Age group	Aboriginal		New South Wales		Relative risk range
	Deaths	ASMR	Deaths ⁵	ASMR	
Males					
0	21	39.8	436	10.3	(2.5 - 6.2)
1-24	24	2.1	843	0.8	(1.8 - 4.1)
25-44	43	10.0	1,338	1.6	(4.7 - 8.8)
45-54	32	27.1	1,428	5.0	(3.8 - 7.9)
55-64	37	72.8	3,843	14.5	(3.6 - 7.1)
65 plus	48	105.3	14,671	58.0	(1.4 - 2.5)
Females					
0	10	14.9	323	8.0	(1.0 - 3.9)
1-24	12	1.0	350	0.3	(1.7 - 5.9)
25-44	19	4.3	624	0.8	(3.7 - 9.5)
45-54	17	13.9	810	3.0	(2.9 - 8.0)
55-64	18	25.9	2,079	7.8	(2.1 - 5.6)
65 plus	34	73.3	15,408	43.6	(1.2 - 2.4)

1. Rates are deaths per 1,000 population per year.

2. A ratio of the observed Aboriginal rate to the total New South Wales rate. The relative risk range represents confidence intervals, at a 95 per cent level of confidence, around each ratio. A relative risk range was calculated for age-specific mortality rates based on five or more deaths.

3. Based on mortality data for 19 western New South Wales communities (see section 4.2).

4. Source: Australian Bureau of Statistics (1988b).

5. Excluding 'age not stated'.

Table 4.4 Sex-specific abridged life tables for Aborigines in western New South Wales, 1984-1987¹.

x	n	P_x	D_x	${}_nM_x$	${}_nq_x$	l_x	e_x°	S.E.
Males								
0	1	132	21	0.0398	0.0386	1.000	53	1.1
1	4	564	6	0.0027	0.0106	0.961	54	1.1
5	10	1,248	2	0.0004	0.0040	0.951	51	1.0
15	10	1,077	16	0.0037	0.0397	0.947	41	1.0
25	10	626	22	0.0088	0.0846	0.910	33	1.0
35	10	446	21	0.0118	0.1137	0.833	25	1.0
45	10	295	32	0.0271	0.2477	0.738	18	0.9
55	10	127	37	0.0728	0.5229	0.555	12	0.9
65	+	114	48	0.1053	1.0000	0.265	10	-
All ages		4,629	205					
Females								
0	1	168	10	0.0149	0.0147	1.000	66	1.2
1	4	501	1	0.0005	0.0020	0.985	66	1.2
5	10	1,241	3	0.0006	0.0060	0.983	62	1.2
15	10	1,118	8	0.0018	0.0175	0.977	52	1.2
25	10	664	9	0.0034	0.0351	0.960	43	1.2
35	10	440	10	0.0057	0.0548	0.927	34	1.1
45	10	306	17	0.0139	0.1379	0.876	26	1.0
55	10	174	18	0.0259	0.2286	0.755	19	0.9
65	+	116	34	0.0733	1.0000	0.582	14	-
All ages		4,728	110					

1. Based on mortality data for 19 western New South Wales communities (see section 4.2). For each age interval x this table yields information on: the duration of the interval, n ; the population at risk, P_x ; the number of deaths, D_x ; the age-specific death rate, ${}_nM_x$; the probability of dying, ${}_nq_x$; the proportion of people who survived, l_x ; the life expectancy at age x , e_x° ; and the standard error around each e_x° value for a closed age interval, S.E..

4.3.1.2 Cause-specific death rates

Comparisons were also made between Aboriginal and total New South Wales cause-specific death rates for major underlying causes of death. As before, the calculations involved directly standardizing the Aboriginal cause-specific death rates to the New South Wales age structure, grouping age intervals, and constructing relative risk ranges for major causes based on five or more deaths. In addition, a measure of the proportion of excess risk for each major underlying cause of death was determined. Excess risk was calculated by subtracting cause-specific death rates for the total New South Wales population from the rates for the Aboriginal population. The proportion of excess risk was then derived by producing a ratio of the Aboriginal cause-specific excess risk to the Aboriginal excess risk for all causes.

This analysis shows that major differences exist between Aboriginal and total New South Wales cause-specific death rates (see Tables 4.5 to 4.9). The most apparent difference is in the incidence of circulatory system diseases as an underlying cause of death. In comparison with the total New South Wales population, diseases of the circulatory system made up over 40 per cent of the excess risk experienced by Aboriginal men and women with the relative risk of death being the highest between the ages of 25 and 64 years. The relative risk range suggests that after the age of 64 years, Aboriginal sex-specific circulatory system disease death rates approach the total New South Wales rates. As shown in Table 4.9, the greatest proportion of excess risk due to circulatory system diseases was from ischaemic heart disease, cerebrovascular disease, and other forms of circulatory system disease including diseases of the pulmonary circulation. When Aboriginal rates for these diseases are compared to total New South Wales rates, ischaemic heart disease accounted for 30 per cent of the total excess risk for males and 26 per cent for females; and cerebrovascular disease accounted for 14 per cent of the total excess risk for males and 22 per cent for females.

External causes, another major underlying cause of death, accounted for approximately 8 per cent of the excess risk experienced by both Aboriginal men and women. For both sexes, the relative risk of death from these causes was at least two times higher than that experienced by the total New South Wales population. The largest proportion of the 45 deaths from external causes, which disproportionately occurred between the ages of 15 and 29 years, were from motor vehicle accidents (22 deaths or 49 per cent) and homicide and injuries purposely inflicted on others (nine deaths or 20 per cent). A small but significant proportion were due to several separate house fires (five deaths or 11 per cent) and self-inflicted injuries (two deaths or 4 per cent) — a suicide by hanging and a death in police custody.

Among Aboriginal men, other important causes of death include diseases of the digestive system, malignant neoplasms (cancer), and respiratory system diseases. In total, these diseases made up over 20 per cent of the excess risk. Most of the 17 deaths caused by digestive system diseases were from chronic liver disease and alcoholic cirrhosis (13 deaths or 76 per cent). Nine deaths or 42 per cent of the 21 deaths from cancer were from lung, pancreas, and prostate cancers. Finally, seven of the nine deaths from respiratory system diseases were from chronic obstructive airways disease (a respiratory disease which is often attributable to the effects of smoking cigarettes).

Among women, other notable causes of death include diseases of the endocrine and respiratory systems. These causes of death made up approximately 30 per cent of the excess risk. Seven of the eight deaths from endocrine disorders were from diabetes; while six of the nine deaths from respiratory system diseases were from chronic obstructive airways disease.

Infant deaths, those occurring in individuals under one year of age, were generally from four specific conditions: congenital anomalies, infectious diseases, perinatal causes, and sudden infant death syndrome. However, sudden infant death syndrome is not actually a cause of death, but rather a category used by physicians to indicate that despite

exhausting all possibilities a cause of death could not be found. All seven reported cases of this condition were baby boys.

Finally, there are other notable causes of death in this data set which should be mentioned because of their implications for Aboriginal health. These are deaths caused by tuberculosis (1 active case), epilepsy (6 deaths), narcotics dependency (3 deaths), acquired immune deficiency syndrome (1 case from blood transfusion), and complications of measles (1 death).

Table 4.5 Male cause-specific mortality rates (CSMR)¹, relative risk², and proportion of excess risk³ for Aborigines in western New South Wales⁴, 1984-1987, as compared with the total New South Wales population (NSW)⁵, 1986.

Cause of death	Aborigines Deaths	Aborigines CSMR	NSW CSMR	Relative risk range	Proportion of excess risk
All causes	205	24.0	8.2	(2.6 - 3.4)	100
Infectious and parasitic	4	0.2	0.0	-	1
Malignant neoplasms	21	3.1	2.1	(1.0 - 2.4)	7
Endocrine, nutritional, and metabolic	2	0.1	0.2	-	0
Mental disorders	3	0.3	0.1	-	2
Nervous system and sense organs	2	0.2	0.1	-	0
Circulatory system	84	13.0	3.8	(2.8 - 4.3)	58
Respiratory system	10	1.4	0.6	(1.2 - 4.7)	5
Digestive system	17	1.8	0.3	(10.0 - 27.6)	10
Genito-urinary system	3	0.5	0.1	-	2
Congenital anomalies	5	0.2	0.0	(1.2 - 8.8)	1
Perinatal causes	6	0.2	0.1	(1.3 - 7.7)	1
Signs, symptoms and ill-defined conditions	18	1.0	0.1	(10.0 - 26.6)	6
Accidents, poisonings and violence	30	2.0	0.7	(2.1 - 4.5)	9

1. Rates are deaths per 1,000 population per year. Aboriginal cause-specific rates were directly standardized to the New South Wales age structure, 30 June 1986.

2. A ratio of the directly standardized Aboriginal rate to the total New South Wales rate. The relative risk range represents confidence intervals, at a 95 per cent level of confidence, around each ratio. A relative risk range was calculated for cause-specific mortality rates based on five or more deaths.

3. Excess risk was calculated by subtracting cause-specific death rates for the total New South Wales population from directly standardized Aboriginal rates. The proportion of excess risk was then derived by producing a ratio of the Aboriginal cause-specific excess risk to the Aboriginal excess risk for all causes. Percentages may not add up because of rounding.

4. Based on mortality data for 19 western New South Wales communities (see section 4.2).

5. Source: Australian Bureau of Statistics (1988c).

Table 4.6 Female cause-specific mortality rates (CSMR)¹, relative risk², and proportion of excess risk³ for Aborigines in western New South Wales⁴, 1984-1987, as compared with the total New South Wales population (NSW)⁵, 1986.

Cause of death	Deaths	Aborigines CSMR	NSW CSMR	Relative risk range	Proportion of excess risk
All causes	110	15.1	7.1	(1.8 - 2.6)	100
Infectious and parasitic	6	0.2	0.0	(3.5 - 20.7)	2
Malignant neoplasms	10	1.9	1.6	(0.6 - 2.4)	3
Endocrine, nutritional, and metabolic	8	1.4	0.1	(4.8 - 21.8)	15
Mental disorders	2	0.1	0.1	-	0
Nervous system and sense organs	3	0.1	0.2	-	0
Circulatory system	41	7.5	3.7	(1.5 - 2.8)	45
Respiratory system	9	1.5	0.5	(2.2 - 9.0)	14
Digestive system	4	0.5	0.3	-	3
Genito-urinary system	5	0.7	0.1	(2.7 - 19.4)	7
Congenital anomalies	2	0.0	0.0	-	0
Perinatal causes	2	0.0	0.0	-	0
Signs, symptoms and ill-defined conditions	3	0.2	0.0	-	2
Accidents, poisonings and violence	15	1.0	0.3	(2.0 - 5.8)	8

1. Rates are deaths per 1,000 population per year. Aboriginal cause-specific rates were directly standardized to the New South Wales age structure, 30 June 1986.

2. A ratio of the directly standardized Aboriginal rate to the total New South Wales rate. The relative risk range represents confidence intervals, at a 95 per cent level of confidence, around each ratio. A relative risk range was calculated for cause-specific mortality rates based on five or more deaths.

3. Excess risk was calculated by subtracting cause-specific death rates for the total New South Wales population from directly standardized Aboriginal rates. The proportion of excess risk was then derived by producing a ratio of the Aboriginal cause-specific excess risk to the Aboriginal excess risk for all causes. Percentages may not add up because of rounding.

4. Based on mortality data for 19 western New South Wales communities (see section 4.2).

5. Source: Australian Bureau of Statistics (1988c).

Table 4.7 Male cause-specific mortality rates (CSMR)¹, relative risk², and proportion of excess risk³ for Aborigines in western New South Wales⁴, 1984-1987, as compared with the total New South Wales population (NSW)⁵, 1986, by major age group and cause of death.

Cause of death	Aborigines Deaths	Aborigines CSMR	NSW CSMR	Relative risk range	Proportion of excess risk
Under 1 year					
All causes	21	39.8	10.3	(2.5 - 6.2)	100
Signs, symptoms and ill-defined conditions	9	17.0	2.9	(3.1 - 12.8)	48
Perinatal causes	6	11.4	4.0	(1.3 - 7.7)	25
Congenital anomalies	4	7.6	2.4	-	18
1 to 24 years					
All causes	24	2.1	0.8	(1.8 - 4.1)	100
Accidents, poisonings and violence	14	1.2	0.6	(1.3 - 4.0)	52
25 to 44 years					
All causes	43	10.0	1.6	(4.7 - 8.8)	100
Circulatory system	15	3.5	0.3	(7.4 - 21.8)	38
Accidents, poisonings and violence	9	2.1	0.7	(1.6 - 6.5)	16
Digestive system	7	1.6	0.3	(2.7 - 13.9)	16
45 to 64 years					
All causes	69	18.2	6.1	(2.4 - 3.9)	100
Circulatory system	36	8.9	2.0	(3.2 - 6.4)	56
Malignant neoplasms	11	2.6	0.1	(10.3 - 37.0)	20
Digestive system	6	1.6	2.8	(0.3 - 1.5)	-10
Respiratory system	5	1.6	0.3	(2.1 - 14.8)	10
65 years and over					
All causes	48	105.3	58.0	(1.2 - 2.4)	100
Circulatory system	32	70.2	30.7	(1.6 - 3.3)	84
Malignant neoplasms	7	15.4	14.6	(0.5 - 2.6)	2

1. Rates are deaths per 1,000 population per year. Aboriginal cause-specific rates were directly standardized to the New South Wales age structure, 30 June 1986.

2. A ratio of the directly standardized Aboriginal rate to the total New South Wales rate. The relative risk range represents confidence intervals, at a 95 per cent level of confidence, around each ratio. A relative risk range was calculated for cause-specific mortality rates based on five or more deaths.

3. Excess risk was calculated by subtracting cause-specific death rates for the total New South Wales population from directly standardized Aboriginal rates. The proportion of excess risk was then derived by producing a ratio of the Aboriginal cause-specific excess risk to the Aboriginal excess risk for all causes. Percentages may not add up because of rounding.

4. Based on mortality data for 19 western New South Wales communities (see section 4.2).

5. Source: Australian Bureau of Statistics (1988c).

Table 4.8 Female cause-specific mortality rates (CSMR)¹, relative risk², and proportion of excess risk³ for Aborigines in western New South Wales⁴, 1984-1987, as compared with the total New South Wales population (NSW)⁵, 1986, by major age group and cause of death.

Cause of death	Aborigines Deaths	Aborigines CSMR	NSW CSMR	Relative risk range	Proportion of excess risk
Under 1 year					
All causes	10	14.9	8.0	(1.0 - 3.9)	100
Infectious and parasitic	4	6.0	0.1	-	86
Congenital anomalies	2	3.0	2.1	-	13
Perinatal causes	2	3.0	3.4	-	-6
1 to 24 years					
All causes	12	1.0	0.3	(1.7 - 5.9)	100
Accidents, poisonings and violence	9	0.8	0.2	(2.0 - 9.0)	85
25 to 44 years					
All causes	19	4.3	0.8	(3.7 - 9.5)	100
Circulatory system	4	0.9	0.1	-	22
Accidents, poisonings and violence	3	0.7	0.2	-	14
45 to 64 years					
All causes	35	18.2	6.1	(2.2 - 4.3)	100
Circulatory system	17	8.9	2.0	(2.8 - 7.7)	56
Endocrine, nutritional and metabolic diseases	5	2.6	0.1	(7.9 - 57.2)	20
Respiratory system	3	1.6	0.3	-	10
Malignant Neoplasms	3	1.6	2.8	-	-10
65 years and over					
All causes	34	73.3	43.6	(1.2 - 2.4)	100
Circulatory system	19	40.9	26.3	(1.0 - 2.6)	50
Malignant neoplasms	5	10.8	8.2	(0.6 - 4.1)	9
Respiratory system	4	8.6	2.4	-	21
Endocrine, nutritional and metabolic diseases	3	6.5	0.9	-	19

1. Rates are deaths per 1,000 population per year. Aboriginal cause-specific rates were directly standardized to the New South Wales age structure, 30 June 1986.

2. A ratio of the directly standardized Aboriginal rate to the total New South Wales rate. The relative risk range represents confidence intervals, at a 95 per cent level of confidence, around each ratio. A relative risk range was calculated for cause-specific mortality rates based on five or more deaths.

3. Excess risk was calculated by subtracting cause-specific death rates for the total New South Wales population from directly standardized Aboriginal rates. The proportion of excess risk was then derived by producing a ratio of the Aboriginal cause-specific excess risk to the Aboriginal excess risk for all causes. Percentages may not add up because of rounding.

4. Based on mortality data for 19 western New South Wales communities (see section 4.2).

5. Source: Australian Bureau of Statistics (1988c).

Table 4.9 Cause-specific mortality rates from circulatory system diseases (CSMR)¹, relative risk², and proportion of excess risk³ for Aborigines in western New South Wales, 1984-1987⁴, as compared with the total New South Wales population (NSW)⁵, 1986, by sex.

Cause of death	Aborigines Deaths	Aborigines CSMR	NSW CSMR	Relative risk range	Proportion of excess risk
Males					
All circulatory system diseases	84	13.0	3.8	(2.8 - 4.3)	58
Rheumatic fever and heart disease	1	0.1	0.0	-	1
Hypertensive disease	2	0.4	0.0	-	2
Ischaemic heart disease	46	7.2	2.4	(2.3 - 4.1)	30
Other forms of heart disease ⁶	15	2.2	0.4	(3.5 - 10.8)	11
Cerebrovascular disease	20	3.0	0.7	(2.7 - 6.8)	14
Other diseases	0	-	0.2	-	0
Females					
All circulatory system diseases	41	7.5	3.7	(1.5 - 2.8)	45
Rheumatic fever and heart disease	1	0.1	0.0	-	0
Hypertensive disease	0	-	0.1	-	0
Ischaemic heart disease	24	4.0	1.9	(1.4 - 3.2)	26
Other forms of heart disease ⁶	3	0.3	0.5	-	-3
Cerebrovascular disease	12	2.8	1.0	(1.6 - 5.3)	22
Other diseases	1	0.3	0.2	-	1

1. Rates are deaths per 1,000 population per year. Aboriginal cause-specific rates were directly standardized to the New South Wales age structure, 30 June 1986.

2. A ratio of the directly standardized Aboriginal rate to the total New South Wales rate. The relative risk range represents confidence intervals, at a 95 per cent level of confidence, around each ratio. A relative risk range was calculated for cause-specific mortality rates based on five or more deaths.

3. Excess risk was calculated by subtracting cause-specific death rates for the total New South Wales population from directly standardized Aboriginal rates. The proportion of excess risk was then derived by producing a ratio of the Aboriginal cause-specific excess risk to the Aboriginal excess risk for all causes. Percentages may not add up because of rounding.

4. Based on mortality data for 19 western New South Wales communities (see section 4.2).

5. Source: Australian Bureau of Statistics (1988c).

6. Includes diseases of the pulmonary circulation.

4.3.2 Comparison of mortality trends over time

The comparative framework employed at the regional level thus far has demonstrated that there are major age- and sex-specific differences in the Aboriginal and total New South Wales mortality regimes. The purpose of this section is to assess whether the Aboriginal mortality rates in this region have improved or deteriorated during the 1980s.

Potential changes in Aboriginal mortality trends in nine communities — Bourke, Brewarrina, Dubbo, Gilgandra, Gingie, Goodooga, Lightning Ridge, Moree, and Walgett — were assessed by constructing life tables and age- and cause-specific mortality rates for two periods, 1980-1983 and 1984-1987. Changes in life expectancy were evaluated by comparing differences at each age interval using a ratio of the differences in life expectancy to the difference in standard error over the two periods. If this ratio, called the critical ratio, exceeded a value of 2.33 then the difference in life expectancy was significant at a 0.01 probability level (see Chiang, 1984: 164-165). Changes in age- and cause-specific rates were appraised by constructing relative risk ranges based on the ratio of the Aboriginal rate in 1980-1983 or 1984-1987 to the New South Wales rate in 1986. As before, relative risk ranges were only calculated for age- and cause-specific rates based on five or more deaths. Finally, a measure of the proportion of excess risk was calculated for all cause-specific death rates.

As a rule, few noticeable differences in Aboriginal mortality trends were found by using the above comparative framework. Between 1980-1983 and 1984-1987, directly standardized rates of overall mortality increased slightly for men at all ages; while for women they remained constant (see Table 4.10).

Differences in life expectancy between the two periods were not found to be significant. In 1980-1983 life expectancy at birth was 58 years for males and 66 years for females; while in 1984-1987 life expectancy at birth was 54 years for males and 68 years for females. What this analysis does show, however, is that the gap between male and

female life expectancy at all ages seems to be getting larger — for example, the gap in life expectancy at birth grew from 8 years in 1980-1983 to 14 years in 1984-1987 (see Tables A1.11 to A1.13 in Appendix 1).

Furthermore, there is little difference in age- and cause-specific mortality rates over the two periods. For males, age- and cause-specific death rates tended to rise, especially during adulthood and from circulatory system diseases; while for females they remained constant (see Tables 4.11 to 4.13).

Very little is actually known about Aboriginal longitudinal mortality trends. A regional study conducted by Khalidi (1989) suggests that there was a steady decline in Aboriginal mortality rates in central Australia between 1975 and 1986. This decline was associated with an improvement in: life expectation at birth; the gap between male and female life expectancy; and the death rates of most leading causes of death. In contrast, Gray (1990b) has argued that Aboriginal mortality patterns in the 1980s were not only similar throughout Australia, but were also similar to the patterns exhibited by Aborigines in the Northern Territory during the 1960s. This similarity was especially apparent after the age of 40 years. The analysis presented here for Aborigines in western New South Wales tends to support the theory that mortality patterns have remained relatively constant. However, the analysis of male mortality rates also demonstrates that age-specific death rates during adulthood and cause-specific rates from circulatory disease have likely increased during the 1980s.

Table 4.10 Crude and standardized death rates¹ and standardized mortality ratios² for Aborigines living in nine western New South Wales communities³, 1980-1983 and 1984-1987, and the total New South Wales population⁴, 1986, by sex.

	Males	Females
Aboriginal, 1980-1983		
Total number of deaths	122	81
Crude death rate	9.3	6.1
Standardized death rate	18.5	13.8
Aboriginal, 1984-1987		
Total number of deaths	136	69
Crude death rate	10.8	5.4
Standardized death rate	24.8	13.2
New South Wales, 1986		
Total number of deaths	22,571	19,596
Crude death rate	8.2	7.1
Standardized mortality ratio		
Aboriginal, 1980-1983 standardized rate to New South Wales crude rate	226 (189 - 273)	195 (157 - 246)
Aboriginal, 1984-1987 standardized rate to New South Wales crude rate	303 (2.56 - 360)	188 (148 - 241)

1. Rates are deaths per 1,000 population per year. Aboriginal crude death rates were directly standardized to the New South Wales age structure, 30 June 1986.
2. A ratio of expected Aboriginal deaths to the total number of deaths in New South Wales in 1986. Confidence intervals, at a 95 per cent level of confidence, are designated by the ratios shown in brackets. The ratios have been multiplied by 100.
3. Based on mortality data for nine western New South Wales communities (see section 4.2).
4. Source: Australian Bureau of Statistics (1988b).

Table 4.11 Age-specific mortality rates (ASMR)¹ and relative risk² for Aborigines living in nine western New South Wales³, 1980-1983 and 1984-1987, and the total New South Wales population⁴, 1986, by sex.

Age group and sex	Aboriginal		New South Wales		Relative risk range
	Deaths	ASMR	Deaths ⁵	ASMR	
Males	1980-1983		1986		
0	12	31.9	436	10.3	(1.8 - 6.0)
1-24	9	1.1	843	0.8	(0.8 - 3.1)
25-44	26	8.8	1,338	1.6	(3.8 - 8.6)
45-54	19	19.5	1,428	5.0	(2.5 - 6.5)
55-64	22	34.8	3,843	14.5	(1.6 - 3.8)
65 plus	34	170.0	14,671	58.0	(2.1 - 4.2)
Males	1984-1987		1986		
0	11	30.2	436	10.3	(1.6 - 5.8)
1-24	15	1.9	843	0.8	(1.5 - 4.3)
25-44	28	9.9	1,338	1.6	(4.4 - 9.5)
45-54	20	23.6	1,428	5.0	(3.1 - 7.7)
55-64	22	65.5	3,843	14.5	(3.0 - 7.2)
65 plus	40	128.2	14,671	58.0	(1.6 - 3.0)
Females	1980-1983		1986		
0	10	20.3	323	8.0	(1.4 - 5.3)
1-24	10	1.3	350	0.3	(2.0 - 7.7)
25-44	19	6.1	624	0.8	(5.2 - 13.5)
45-54	5	5.6	810	3.0	(0.8 - 5.8)
55-64	9	17.7	2,079	7.8	(1.2 - 5.0)
65 plus	28	70.0	15,408	43.6	(1.1 - 2.4)
Females	1984-1987		1986		
0	9	18.6	323	8.0	(1.2 - 5.1)
1-24	7	0.9	350	0.3	(1.3 - 6.5)
25-44	13	4.3	624	0.8	(3.4 - 10.8)
45-54	11	12.7	810	3.0	(2.4 - 8.5)
55-64	10	21.0	2,079	7.8	(1.5 - 5.6)
65 plus	19	63.3	15,408	43.6	(0.9 - 2.4)

1. Rates are deaths per 1,000 population per year.

2. A ratio of the observed Aboriginal rate to the total New South Wales rate. The relative risk range represents confidence intervals, at a 95 per cent level of confidence, around each ratio. A relative risk range was calculated for age-specific mortality rates based on five or more deaths.

3. Based on mortality data for nine western New South Wales communities (see section 4.2).

4. Source: Australian Bureau of Statistics (1988b).

5. Excluding 'age not stated'.

Table 4.12 Male cause-specific mortality rates (CSMR)¹, relative risk², and proportion of excess risk³ for Aborigines living in nine western New South Wales communities⁴, 1980-1983 and 1984-1987, as compared with the total New South Wales population (NSW)⁵, 1986.

Cause of death	Aborigines Deaths	CSMR	NSW CSMR	Relative risk range	Proportion of excess risk
1980-1983					
All causes	122	18.5	8.2	(1.9 - 2.7)	100
Malignant neoplasms	5	1.2	2.1	(0.2 - 1.7)	-9
Circulatory system	51	8.7	3.8	(1.7 - 3.1)	47
Respiratory system	18	3.8	0.6	(3.7 - 10.0)	30
Digestive system	12	1.5	0.3	(3.0 - 10.2)	12
Accidents, poisonings and violence	11	1.0	0.7	(0.8 - 3.0)	3
Other causes	25	2.4	0.8	(2.2 - 5.0)	16
1984-1987					
All causes	136	24.8	8.2	(2.6 - 3.6)	100
Malignant neoplasms	16	3.6	2.1	(1.1 - 3.0)	9
Circulatory system	61	14.3	3.8	(3.0 - 5.0)	64
Respiratory system	4	1.0	0.6	-	2
Digestive system	12	1.7	0.3	(3.3 - 11.2)	8
Accidents, poisonings and violence	17	1.7	0.7	(1.6 - 4.5)	7
Other causes	26	2.5	0.8	(2.3 - 5.1)	11

1. Rates are deaths per 1,000 population per year. Aboriginal cause-specific rates were directly standardized to the New South Wales age structure, 30 June 1986.

2. A ratio of the directly standardized Aboriginal rate to the total New South Wales rate. The relative risk range represents confidence intervals, at a 95 per cent level of confidence, around each ratio. A relative risk range was calculated for cause-specific mortality rates based on five or more deaths.

3. Excess risk was calculated by subtracting cause-specific death rates for the total New South Wales population from directly standardized Aboriginal rates. The proportion of excess risk was then derived by producing a ratio of the Aboriginal cause-specific excess risk to the Aboriginal excess risk for all causes. Percentages may not add up because of rounding.

4. Based on mortality data for nine western New South Wales communities (see section 4.2).

5. Source: Australian Bureau of Statistics (1988c).

Table 4.13 Female cause-specific mortality rates (CSMR)¹, relative risk², and proportion of excess risk³ for Aborigines living in nine western New South Wales communities⁴, 1980-1983 and 1984-1987, as compared with the total New South Wales population (NSW)⁵, 1986.

Cause of death	Aborigines Deaths	CSMR	NSW CSMR	Relative risk range	Proportion of excess risk
1980-1983					
All causes	81	13.8	7.1	(1.6 - 2.5)	100
Malignant neoplasms	8	1.4	1.6	(0.4 - 2.0)	-3
Circulatory system	30	7.7	3.7	(1.4 - 3.0)	59
Respiratory system	6	1.2	0.4	(1.5 - 8.6)	12
Digestive system	6	0.5	0.3	(0.9 - 5.2)	3
Accidents, poisonings and violence	11	0.7	0.3	(1.3 - 5.0)	6
Other causes	20	2.3	0.8	(1.9 - 4.9)	23
1984-1987					
All causes	69	13.2	7.1	(1.5 - 2.4)	100
Malignant neoplasms	5	1.5	1.6	(0.4 - 2.8)	-2
Circulatory system	23	6.2	3.7	(1.1 - 2.6)	40
Respiratory system	4	0.8	0.4	-	8
Digestive system	4	0.7	0.3	-	8
Accidents, poisonings and violence	10	1.0	0.3	(1.8 - 6.9)	11
Other causes	23	3.0	0.8	(2.5 - 6.0)	35

1. Rates are deaths per 1,000 population per year. Aboriginal cause-specific rates were directly standardized to the New South Wales age structure, 30 June 1986.

2. A ratio of the directly standardized Aboriginal rate to the total New South Wales rate. The relative risk range represents confidence intervals, at a 95 per cent level of confidence, around each ratio. A relative risk range was calculated for cause-specific mortality rates based on five or more deaths.

3. Excess risk was calculated by subtracting cause-specific death rates for the total New South Wales population from directly standardized Aboriginal rates. The proportion of excess risk was then derived by producing a ratio of the Aboriginal cause-specific excess risk to the Aboriginal excess risk for all causes. Percentages may not add up because of rounding.

4. Based on mortality data for nine western New South Wales communities (see section 4.2).

5. Source: Australian Bureau of Statistics (1988c).

4.3.3 Comparison with other birthplace groups

The final comparison at the regional level is made between the mortality experiences of Aborigines in western New South Wales from 1984 to 1987 and other Australian- and overseas-born birthplace groups in 1986 by using standardized mortality ratios. In this case the standardized mortality ratio is a ratio of the observed to the expected number of deaths for persons aged 15 to 74 years. The expected number of deaths was calculated by applying Australian age- and cause-specific mortality rates in 1986 to the age and sex structure of each birthplace group and summing the results. Standardized mortality ratios for all birthplace groups, except Aborigines, are based on figures produced by the Australian Bureau of Statistics from one year totals of deaths (1988d).

For all causes of death, Aborigines have sex-specific standardized mortality ratios significantly higher than those of any Australian- or overseas-born birthplace group (see Table 4.14). Standardized mortality ratios for males range from 46 for Vietnamese-born to 424 for Aborigines; and for females from 76 for Yugoslavian-born to 368 for Aborigines. Taking all ratios into account, the standardized mortality ratios for Aborigines are four times higher than those for Australian-born and all overseas-born birthplace groups in Australia. When standardized circulatory system disease mortality ratios are compared, a similar pattern emerges — they are about five times higher for Aborigines than for other birthplace groups (see Table 4.14).

Overall, this table provides a striking reminder of how different Aboriginal mortality patterns in this region are from those exhibited by other Australians. No other birthplace group in Australia has similar levels of overall and circulatory system disease mortality. The table also provides evidence of significant differences in Australian- and overseas-born mortality patterns, which are most probably attributable to differences in lifestyle and to prospective immigrants being required to pass a medical screening before immigrating to Australia (Dasvarma, 1980; Young, 1986a).

Table 4.14 Standardized mortality ratios¹ for all causes and circulatory system diseases for Aborigines in western New South Wales², 1984-1987, and other birthplace groups in Australia³, 1986, by sex and birthplace.

Birthplace group	All causes		Circulatory system diseases	
	Males	Females	Males	Females
All Australians ⁴	100	100	100	100
Australian-born	105	104	105	105
Western New South Wales Aborigines	424	368	535	483
Africa	80	85	88	96
America	84	78	84	79
Canada and USA	96	91	96	94
Other	59	62	57	58
Asia	68	72	76	70
Lebanon	58	69	79	87
Vietnam	46	71	25	39
Europe	91	90	89	88
Greece	56	67	56	64
Italy	68	69	59	63
UK and Ireland	97	99	93	93
Yugoslavia	88	76	77	74
Other	100	92	105	95
Oceania	95	88	93	88
New Zealand	92	84	89	83
Other	110	108	114	112

1. In this case the standardized mortality ratio is the ratio of the observed to the expected number of deaths for persons aged 15 to 74 years if Australian age-specific mortality applied.
2. Based on mortality data for 19 western New South Wales communities (see section 4.2).
3. The standardized mortality ratios shown in this table for birthplace groups other than Australian Aborigines are based on the ratios from an Australian Bureau of Statistics publication (1988d: 74, Table 2.4). The ratios have been multiplied by 100.
4. Including Australian-born and all other overseas-born birthplace groups.

4.4 Comparisons at a community level

The purpose of this community level⁵ comparison is twofold: to highlight the community differences in mortality rates from 1984 to 1987; and to examine the relationship between these community mortality differentials and several demographic and social-cultural variables.

Differences in the social environments of various communities in western New South Wales are evident even to the most casual observer (Gray and Hogg, 1989). In the largest communities, like Dubbo and Wellington, Aborigines make up a small proportion of the total population and are generally urban residents. By contrast, in smaller country towns, like Bourke, Brewarrina, Moree, and Walgett, Aborigines make up a substantial proportion of the total population and often live within former reserves or fringe settlements alongside or in these communities.

Somewhat less obvious are the community differences in mortality rates. After standardizing for different age structures, the communities in this study have average death rates (standardized mortality ratios) ranging from over half to twice the regional average crude death rate for Aborigines (see Table 4.15). Additionally, after a simple chi square test was employed, the observed number of deaths was shown to be significantly different from the expected number in half of these communities. Overall, the communities can be divided into three broad categories: very high mortality in Bourke, Brewarrina, and Coonamble; moderately high mortality in Walgett and Wilcannia; and below average mortality, but still high by non-Aboriginal standards, in Condobolin, Dubbo, Moree, Wellington, and other communities (Collarenebri, Gilgandra, Goodooga, Lightning Ridge, Menindee, and Narrabri).

5. As already stated, data on 315 Aboriginal deaths which occurred in 19 New South Wales communities between 1984 and 1987 were used to make this comparison (see section 4.2).

Four demographic and social-cultural variables have been used to help explain why these community mortality differentials exist (see Table 4.16). The first variable is household size and the second is employment. Community mortality rates are negatively ($p < 0.1$) but marginally correlated with the proportion of Aboriginal households with less than five members; and are negatively ($p < 0.05$) correlated with the proportion of the Aboriginal labour force employed. These correlations suggest that Aboriginal mortality is lower in communities where households are smaller and where more Aborigines are employed.

The third and fourth variables are the Aboriginal dependency ratio and the proportion of Aboriginal people in each community's total population. The dependency ratio is defined as the ratio of Aboriginal individuals in each community aged 14 years and under and 65 years and over to those between the ages of 15 and 64 years. Mortality rates are strongly negatively ($p < 0.01$) correlated with these ratios, which range in value from 93 for Wellington to 68 for Brewarrina. In contrast, mortality rates are positively ($p < 0.05$) correlated with the proportion of Aboriginal people in each community. These two correlations suggest that the low mortality communities in this study have a smaller proportion of Aboriginal people in the total population and/or a smaller proportion of Aboriginal people between the ages of 15 and 64 years.

Additionally, three of these four demographic and social-cultural variables — employment, household size, and the proportion of Aboriginal descent — are moderately correlated with each other. The proportion employed is negatively ($p < 0.2$) correlated with the proportion of Aboriginal descent and positively ($p < 0.1$) correlated with the proportion of households with less than five persons; while the proportion of Aboriginal descent is somewhat positively ($p < 0.1$) correlated with the proportion of households with less than five persons. These interrelationships are especially noticeable in low mortality communities, which are often characterized by higher levels of employment, a smaller proportion of individuals of Aboriginal descent, and a greater

proportion of Aboriginal households with less than five persons. In contrast, the association between these three variables in high and moderately high mortality communities is much more variable.

Overall, these demographic and social-cultural variables are important because they highlight a number of underlying community and individual characteristics which can either promote or discourage differences in the levels of community mortality.

Although no causal model is offered here, they seem to be more important in describing why different levels of low mortality exist in some communities than in describing the existence of high mortality rates in others.

Table 4.15 Standardized mortality ratios¹ for Aborigines of both sexes living in selected western New South Wales communities, 1984-1987, by level of community mortality.

Community mortality level	Observed deaths	Expected deaths	Standardized mortality ratio ²
High			
Coonamble ³	27	13	210*
Bourke	37	20	190*
Brewarrina	36	21	170*
Moderate			
Walgett ⁴	49	36	140*
Wilcannia	22	17	130
Low			
Condobolin ⁵	25	29	90
Moree	45	53	80
Other communities ⁶	35	46	70
Wellington	14	22	60
Dubbo	25	45	60*

1. In this case the standardized mortality ratio is the ratio of the observed to the expected number of deaths for Aborigines of both sexes. Observed deaths are the actual number of deaths which occurred in the community between 1984 and 1987. Expected deaths are the number of deaths expected if Aboriginal age-specific mortality for western New South Wales, 1984-1987, applied. The ratios have been multiplied by 100.
2. An asterisk represents a significant difference between the observed and expected number of deaths at a probability level of 0.05.
3. Includes deaths from Gulargambone.
4. Includes deaths from Gingie.
5. Includes deaths from Lake Cargelligo and Murrin Bridge.
6. Includes deaths from Collarenebri, Gilgandra, Goodooga, Lightning Ridge, Menindee, and Narrabri.

Table 4.16 Comparison of community characteristics¹ by level of community mortality.

Community mortality level	Community characteristics				
	SMR ² (per 100)	HSE (per cent)	EMP (per cent)	DPR (per 100)	PAB (per cent)
High					
Coonamble ³	210	31	40	69	15
Bourke	190	28	34	72	23
Brewarrina	170	26	52	68	54
Moderate					
Walgett ⁴	140	24	42	78	39
Wilcannia	130	20	38	80	53
Low					
Condobolin ⁵	90	31	42	75	17
Moree	80	26	41	79	16
Other communities ⁶	70	34	48	70	11
Wellington	60	31	54	93	13
Dubbo	60	42	53	82	6
Average	100	30	54	77	13
Rank correlation with SMR⁷		-0.44 (0.19)	-0.68 (0.03)	-0.77 (0.00)	0.65 (0.04)

1. Community-specific data on four variables were obtained from the 1986 Australian census of population and housing.

These variables include: proportion of households with less than five persons (HSE), proportion employed (EMP), dependency ratio (DPR), and proportion of Aboriginal descent (PAB)

2. In this case the standardized mortality ratio is the ratio of the observed to the expected number of deaths for Aborigines of both sexes if Aboriginal age-specific mortality for western New South Wales, 1984-1987, applied.

The ratios have been multiplied by 100.

3. Includes deaths from Gulargambone.

4. Includes deaths from Gingie.

5. Includes deaths from Lake Cargelligo and Murrin Bridge.

6. Includes deaths from Collarenebri, Gilgandra, Goodooga, Lightning Ridge, Menindee, and Narrabri.

7. Spearman's rank correlations with confidence intervals in brackets underneath.

4.5 Comparisons at an individual level

In this section a comparison is made between the observed and expected number of joint occurrences of various disease conditions which were mentioned as causes of death. This analysis is restricted to all the death records of Aborigines aged 15 years and over in the total data set — a subset of 375 male and 211 female deaths.

In total, information was gathered from the death records on the incidence of 17 specific disease classes: ischaemic (coronary) heart disease; other circulatory system diseases; liver disease; other digestive system diseases; kidney disease; diabetes; other pancreatic diseases; alcohol-related conditions; chronic obstructive airways disease; other respiratory system diseases; malignant neoplasms (cancer); external causes (accidents, poisonings or violence); infectious diseases; epilepsy; broken bones or osteoporosis; malnutrition or cachexia; and old age or senility.

A measure of association between two disease conditions was obtained by dividing the observed number of joint occurrences by the expected number. The expected number was obtained by multiplying the number of individual records which mention the first disease condition by those which mention the second for each age group, and then dividing this sum by the total number of deaths which occurred at that same age group. A total was obtained by summing over the various age groups. If this measure of association is more than one, the observed number of joint occurrences is higher than expected, and if it is less than one, the observed number of joint occurrences is lower than expected. For example, for males the measure of the association between ischaemic heart disease and liver disease was calculated to be 1.00 (19 observed divided by 19 expected occurrences). In this and some other cases, the expected number is equal to the observed number of joint occurrences of two disease conditions. Measures of association were only calculated for observed frequencies numbering five or more in an effort to limit the effects of small numbers.

A chi square test at one degree of freedom was used to test the statistical significance of the measures of association obtained. Statistical significance was evaluated by determining whether the calculated chi square values exceeded a critical value at a 5 per cent, 1 per cent, or 0.1 per cent level of significance. However, because of the repetitive nature of this test, there is a high probability that some significant associations will occur purely by chance.

Tables 4.17 and 4.18 highlight various associations between 11 prevalent types of disease conditions mentioned in the death records as causes of Aboriginal death. In particular, these matrices summarize the association and joint occurrences between: ischaemic heart disease; other circulatory system diseases; liver disease; other digestive system diseases; kidney disease; diabetes; alcohol-related conditions; chronic obstructive airways disease; other respiratory system diseases; infectious diseases; and malnutrition or cachexia. The most notable associations seem to occur between ischaemic heart disease, other circulatory system diseases, liver disease, alcohol-related conditions, chronic obstructive airways disease, other respiratory system diseases, and infectious diseases. No notable associations were observed between the six disease conditions that were dropped from these tables.

Generally, the measures of association between heart disease (both ischaemic heart disease and other circulatory system diseases) and other disease conditions are either less than one or not much greater than one and not statistically significant. However, some associations were statistically significant — heart disease was associated with kidney disease and diabetes for males, and diabetes and alcohol-related conditions for females. For males, the most important association was between kidney disease and other circulatory system diseases, where the observed number of joint occurrences is twice the expected number ($p < 0.001$). For females, there were negative associations between both ischaemic heart disease and other circulatory system diseases and alcohol-related conditions ($p < 0.05$).

One of the more striking associations, for either sex, was found between alcohol-related conditions and liver disease. In this case the observed number of joint occurrences is for males (35 cases) nearly three times and for females (9 cases) nearly four times the expected number ($p < 0.001$). This strong association is not surprising considering that most deaths due to liver disease in the data set were from alcoholic cirrhosis of the liver. Also, alcohol-related conditions were shown to be associated with infectious diseases, other digestive system diseases, and malnutrition or cachexia for males, and infectious diseases for females; and liver disease was shown to be associated with kidney disease and infectious diseases for males.

The associations between other respiratory system diseases and a range of other conditions simply seem to be due to the fact that people of both sexes, often the elderly, eventually die of pneumonia after fighting diseases of all types.

What is interesting about this analysis is that ischaemic heart disease, or for that matter other circulatory system diseases, was not positively associated with alcohol-related conditions. As shown in Tables 4.17 and 4.18, joint occurrences of heart disease and alcohol-related conditions were mentioned in many death records. However, what is being established here is that the observed number of joint occurrences of these two disease conditions is not significantly higher than what would be expected if the two conditions were independent of one another. What this analysis indicates is that circulatory system diseases would most probably retain their importance as causes of death among Aborigines even without the existence of alcohol-related conditions.

Table 4.17 Measure of association¹ between 11 disease conditions mentioned as causes of death for male Aborigines² who died in western New South Wales, 1979-1989.

	Disease condition ³										
	1	2	3	4	5	6	7	8	9	10	11
1	-	1.0 (44)	1.0 (19)	- (2)	- (3)	1.4 (17)	1.0 (36)	1.1 (17)	0.9 (37)	0.7 (13)	0.6 (6)
2		-	1.1 (12)	- (3)	1.9** (7)	1.5* (10)	1.2 (25)	0.8 (6)	0.8 (17)	1.1 (5)	- (4)
3			-	- (1)	2.4** (5)	- (2)	2.7*** (35)	- (1)	1.1 (8)	1.6* (6)	- (2)
4					- (1)	- (1)	2.1** (5)	- (0)	3.1** (5)	- (2)	- (2)
5					-	- (1)	- (4)	- (0)	- (2)	- (3)	- (1)
6						-	1.0 (5)	- (1)	1.4 (8)	- (3)	- (0)
7							-	1.0 (5)	1.0 (15)	1.7* (12)	2.1** (9)
8								-	1.4 (11)	- (4)	- (2)
9									-	2.6*** (24)	1.5* (7)
10										-	- (3)
11											-

1. The measure of association is the observed number of joint occurrences of the two conditions divided by the expected number based on independent distributions. The measure was only calculated for observed frequencies numbering five or more. The figures in brackets show the observed number of joint occurrences.

2. Comprising a sample of 375 deaths of males aged 15 years and over.

3. This matrix compares the joint occurrences between types of disease conditions mentioned as causes of death including: ischaemic heart disease (1); other circulatory system diseases (2); liver disease (3); other digestive system diseases (4); kidney disease (5); diabetes (6); alcohol-related conditions (7); chronic obstructive airways disease (8); other respiratory system diseases (9); infectious diseases (10); and malnutrition or cachexia (11). An asterisk represents a significant difference between the observed and expected number of deaths at a 0.05 probability level; two asterisks represent a significant difference at a 0.01 probability level; and three asterisks represent a significant difference at a 0.001 probability level.

Table 4.18 Measure of association¹ between 11 disease conditions mentioned as causes of death for female Aborigines² who died in western New South Wales, 1979-1989.

	Disease condition ³										
	1	2	3	4	5	6	7	8	9	10	11
1	-	1.0 (34)	- (4)	- (1)	1.2 (15)	1.3 (26)	0.2* (5)	0.8 (7)	0.9 (18)	0.6 (7)	- (1)
2		-	- (1)	- (1)	0.9 (8)	1.6* (24)	0.3* (5)	- (2)	0.8 (12)	1.1 (9)	- (1)
3			-	- (1)	- (3)	- (1)	3.9*** (9)	- (1)	- (2)	- (9)	- (1)
4					- (2)	- (1)	- (1)	- (2)	- (2)	- (1)	- (0)
5					-	1.2 (6)	- (2)	- (2)	0.9 (5)	- (4)	- (0)
6						-	- (3)	- (2)	1.0 (9)	- (3)	- (0)
7							-	- (1)	1.5 (6)	2.7*** (7)	- (0)
8								-	2.0** (9)	2.8*** (6)	- (0)
9									-	3.4*** (19)	- (0)
10										-	- (0)
11											-

1. The measure of association is the observed number of joint occurrences of the two conditions divided by the expected number based on independent distributions. The measure was only calculated for observed frequencies numbering five or more. The figures in brackets show the observed number of joint occurrences.

2. Comprising a sample of 211 deaths of females aged 15 years and over.

3. This matrix compares the joint occurrences between types of disease conditions mentioned as causes of death including: ischaemic heart disease (1); other circulatory system diseases (2); liver disease (3); other digestive system diseases (4); kidney disease (5); diabetes (6); alcohol-related conditions (7); chronic obstructive airways disease (8); other respiratory system diseases (9); infectious diseases (10); and malnutrition or cachexia (11). An asterisk represents a significant difference between the observed and expected number of deaths at a 0.05 probability level; two asterisks represent a significant difference at a 0.01 probability level; and three asterisks represent a significant difference at a 0.001 probability level.

4.6 Summary

This chapter has attempted to evaluate current Aboriginal mortality trends in western New South Wales by making comparisons at regional, community, and individual levels. At a regional level, Aboriginal mortality rates have been shown to differ significantly from those exhibited by the total New South Wales population. In particular, the analysis has demonstrated that in comparison to the total New South Wales population: the relative risk of death for Aborigines of both sexes was at least two times higher; Aboriginal life expectancy at birth was 15 to 20 years lower; and Aboriginal cause-specific death rates were much higher for most underlying causes of death. Deaths from circulatory system diseases accounted for the greatest proportion of the excess risk between the Aboriginal and total New South Wales cause-specific rates.

Additionally, regional comparisons noted few differences in the level of Aboriginal mortality between 1980-1983 and 1984-1987, but very substantial disparities between Aboriginal mortality patterns and those exhibited by other Australian- and overseas-born birthplace groups. During the 1980s, Aboriginal life expectancy and age- and cause-specific mortality rates did not change significantly. However, among males there has been a small increase in age-specific mortality during adulthood and cause-specific mortality from circulatory system diseases. When Aboriginal sex- and cause-specific standardized mortality ratios were compared with those of other birthplace groups, Aboriginal ratios were found to be considerably higher.

At a community level, significant differences were found between the mortality rates of a select sample of communities. As a rule, mortality rates in these communities tend to fall into three broad categories — very high, moderately high, and low — and to be somewhat correlated with household size, employment, the proportion of Aboriginal descent, and the dependency ratio.

At an individual level, there are two notable findings from the analysis of the joint occurrences of various disease conditions. The first and most important finding is the

lack of association between circulatory system diseases and alcohol-related conditions. This suggests that alcohol-related conditions are not a major cause of heart disease. The second finding is the significant association between alcohol-related conditions and liver disease, which is not at all surprising.

These comparisons have demonstrated that the Aboriginal mortality rates in western New South Wales are remarkably higher than those exhibited by other Australians. Furthermore, as among other Aboriginal groups in Australia, circulatory system diseases account for the greatest proportion of this difference (see section 2.4 in chapter 2). Without substantial reductions in the incidence of heart disease, Aboriginal mortality rates in this and other regions of Australia will remain high.

CHAPTER FIVE

RISK FACTOR PREVALENCE SURVEY

5.1 Introduction and overview

In addition to gathering information on Aboriginal deaths in western New South Wales, I conducted a risk factor prevalence survey in Bourke during April and May of 1989. This survey was designed to assess the prevalence of and level of association between various behavioural and biological risk factors of coronary heart disease among Aborigines aged 18 years and over. The purpose of the survey, apart from gathering information for my own research needs, was to provide the community, especially the Aboriginal Health Service, with an assessment of the risk factors among Bourke Aborigines so that appropriate health promotion programs could be designed and implemented.

This was not the first such survey conducted in Bourke. Over the past 20 years Bourke Aborigines have been the subject of various health and social research projects. Many of the local research endeavours have been conducted by the town's health care practitioners. There is the well-known work conducted by Kamien and associates on Aboriginal health and nutrition in Bourke during the late 1960s and early 1970s as part of a project on the Human Ecology of the Arid Zone (Kamien *et al.*, 1974, 1975a; Kamien, 1975a, 1975b, 1975c, 1976); the morbidity survey by Coolican (1971, 1974) on his medical practice in 1968-69; the work by Cameron and colleagues (1986) on Aboriginal diabetes; and the work by Harris and colleagues on Aboriginal respiratory health and living conditions (Harris and Davies, 1984; Harris *et al.*, 1984).

Additionally, the community has been studied by external researchers. Among others, there is the work of Castle and Hagan on Aboriginal employment (Castle and Hagan, 1983; Castle, 1987); Parkinson (1982) on the Aboriginal history of Bourke; Rowley

(1982) on Aboriginal health and living conditions; Gray and Hogg (1989) on Aboriginal mortality; and the New South Wales Ministry of Aboriginal Affairs (1987) on the Bourke disturbances in August of 1986.

5.2 The setting

Bourke is situated on the Darling River, about 800 kilometres northwest of Sydney in the far northwest of New South Wales (see Figure 3.1). In 1986, the town and surrounding area had approximately 4,260 residents of whom 900 or 23 per cent were of Aboriginal descent (Australian Bureau of Statistics 1986 census of population and housing). The Aboriginal population resides in two main areas — the town settlers live in the east and northeast parts of town, and the fringe and former reserve settlers live west of town in an area of mostly old and run-down housing stock. Few Aborigines in Bourke are descendants of the Ngjamba tribe whose territory at contact included the present site of Bourke. Rather, most are descendants of a wide variety of Aboriginal groups from the surrounding region who arrived in Bourke 50 to 60 years ago (Kamien, 1975a). The descendants of the Ba:gundji tribe came from Wilcannia, the Marawari tribe from around the Paroo River, and from Peak Hill came descendants of the Wiradjuri and Wongaibon tribes. Other local inhabitants are descendants of the Wangkumara people who in 1935 were herded into cattle trucks and taken to Brewarrina from the Tibooburra region and who ended up residing in Bourke (see Beckett, 1958a; Long, 1970).

While the Aborigines in Bourke form a large proportion of the total population, they have little economic or political control (New South Wales, Ministry of Aboriginal Affairs, 1987). Although local Aboriginal services, such as the Aboriginal Health Service, the Widjeri Housing Co-operative, and the Land Council and legal office, have been established in recent years, real political control remains entirely in the hands of the non-Aboriginal population. The local shire council and chambers of commerce are made up entirely of non-Aborigines. In addition, the local court administration, the

banks, the police, the Community Employment Service, Australia Post, Telecom, and the schools and various other government agencies and services are generally staffed by non-Aborigines. Despite the fact that most businesses in town depend to a large extent on the Aboriginal population, especially on their social security payments, no local businesses are owned or operated by Aborigines and few, if any, have Aboriginal staff (see section 3.3.3 in chapter 3).

Economically, like other country towns in the region, Bourke is basically a service, retail, and distribution centre for the northwest part of the state (Castle, 1987: 26). Until relatively recently, sheep and wool production formed the primary industry; however, cotton, citrus fruit, grain, and some beef cattle production have recently broadened this economic base. In the 1986-87 growing season, the gross value of agricultural commodities produced in the shire was \$38 million. Over 23,000 beef cattle were produced and 1,000,000 sheep and lambs were shorn. In addition, the shire produced over 1,000 tonnes of cereal grains, 15,000 tonnes of cotton, and 200 tonnes of citrus fruit (Australian Bureau of Statistics, 1988a). Until 1987, the major secondary industry in Bourke was an abattoir. Now, however, there are only a few engineering and motor repair shops. The tertiary sector, or service industry, which expanded considerably in the early 1980s, is made up of an array of local, state, and commonwealth government agencies and services and local retail and tourist enterprises.

Since the closing of the abattoir in 1987, employment prospects for both Aborigines and non-Aborigines have deteriorated considerably (Castle, 1987: 27-28). There is no other large industry in town and no realistic chance of any being established. The main source of employment (as for Aborigines in other rural areas of New South Wales) remains the public sector, especially the shire council and hospital. However, with the past and probable future consolidation of government services, it is unlikely that this sector will provide future growth in employment. As discussed in chapter 3 (see section 3.3.3), Aborigines' access to employment on agricultural properties has declined during

the 1980s because of a reluctance on the part of farmers and pastoralists to hire during a period of increasing costs and debt levels.

5.3 Community approval and ethics

Before the survey was conducted, the approval and co-operation of the Bourke Aboriginal Health Service's Board of Governors and health workers was sought. As part of this successful consultation process we agreed that: all information about individuals and families would be kept confidential; no photocopies of questionnaires would be made and the interview reports and questionnaire key would remain the property of the Aboriginal Health Service; the questionnaires and log books would be destroyed by incineration after the survey was completed; a copy of the coding sheets and schedules would be left with the service on completion of the survey; computer diskettes and printout records of the survey data would be left with the service once they were available; and the service would be supplied with a copy of my thesis and any resulting publications. In addition, an individual's participation in the survey was seen as being totally voluntary. He or she was free to withdraw at any time. The privacy of individuals who did participate was protected in various ways by: restricting the access to questionnaires when the survey was taking place; placing appropriate safeguards on the handling, storing, and final disposal of questionnaires and log books; and not identifying the names or circumstances of any participants in this thesis or subsequent papers. The study was conducted with the full approval of the Aboriginal Health Service and the Ethics Committee of the Australian National University and was only concerned with the patterns exhibited by the total sample and not with the identification of particular individuals.

5.4 The survey

As already mentioned, this survey was concerned with assessing the prevalence of and level of association between various risk factors of coronary heart disease among

Aborigines aged 18 years and over. More specifically, I aimed to examine the interrelationships between alcohol and tobacco consumption, blood pressure, body composition, diet, physical activity, and serum cholesterol and the influence of various demographic, environmental, and social-cultural conditions (risk markers) on these risk factors.

5.4.1 Questionnaire design

Two questionnaires were developed for this study — one for households and the other for individuals. They were designed by drafting the above research aims into specific objectives, and then matching each objective to a set of questions and variables; additionally, they were designed in such a way that the approaches taken and questions used would be comparable to those of the National Heart Foundation's risk factor prevalence study of adults living in various capital cities throughout Australia (National Heart Foundation, 1985; Department of Health, 1986; Department of Health and Community Services, 1987). To minimize errors and problems of content and layout, the questionnaires were pretested on Aboriginal health workers and interviewers, as well as on other health researchers.

The household questionnaire was primarily used to elicit information on adult survivorship and on family relationships and living conditions within Aboriginal households, and to determine which persons in the household were eligible to be interviewed on an individual basis (see Appendix 2). A cover sheet was used to denote household members by name and by unique household and individual identification numbers. Once the cover sheet was completed, information was collected on household members: their sex and age, whether their parents were still alive, and their relationship to the head of the household. Information about the type and size of dwelling and its general condition was also recorded.

The individual questionnaire was used to gather information on each participant's health, lifestyle, and diet (see Appendix 2). Clinical and anthropometric data, such as a person's height, weight, blood pressure, and serum cholesterol, were gathered at the Aboriginal Health Service using its facilities. Individuals who had participated in the survey were asked, and often repeatedly reminded, to go to the Health Service and get their height, weight, and blood pressure measured. Height and weight were measured in a standing position with shoes and clothes on. Height was measured from the floor with a stadiometer and weight was measured using a balance type of scale. Systolic and diastolic blood pressures were measured in a sitting position using a sphygmomanometer. These measurements were taken by either an Aboriginal health worker or the nursing sister and were recorded in both the questionnaire and the Health Service's patient records.

Additionally, the serum cholesterol of non-fasting adults was measured by means of the Reflotron dry-chemistry system — a rather simple method requiring little training. The Health Service's nursing sister obtained a drop of blood from a finger prick, and this blood, which was collected in a capillary tube, was then placed on the Reflotron reagent strip and inserted into the previously-calibrated Reflotron analyser according to the manufacturer's instructions. The results, which were obtained in only a few minutes, were then recorded in the questionnaire and the Health Service's patient records. When high cholesterol readings were obtained (greater than 5.5 mmol/litre), the respondent was counselled on how his or her cholesterol level could be reduced. The results obtained on the Reflotron system, as shown by several recent studies, are comparable to those obtained from more commonly used laboratory instruments (James *et al.*, 1988; Kinlay, 1988; Pain, 1988; Phillips *et al.*, 1988).

In addition to gathering clinical and anthropometric data, the questionnaire asked general morbidity and specific diagnostic questions concerning an individual's health. Questions on morbidity included a set about whether respondents had been told by a

doctor that they had or had had: high blood pressure, high blood cholesterol, diabetes, a heart attack, or a stroke. There were also questions about whether respondents had been in the hospital during the past year and about how they would rate their health status against a set scale and against other Aborigines of the same age. A series of diagnostic questions designed by the London School of Tropical Medicine and Hygiene (Rose, 1962) were also included and were used to identify the following conditions: angina pectoris, a possible myocardial infarction, and intermittent claudication.

Lifestyle questions focused on eliciting information about a respondent's social and economic conditions, familial and social support mechanisms, exercise habits, and alcohol and tobacco consumption. In regard to social and economic conditions, the questions were designed to gather information on residence (which was defined in terms of how long a person had lived in Bourke), education, and marital and employment status. The proposed questions on pension benefits were not asked (see questions 9a and b on data sheet 3 in Appendix 2) because the pretest showed that they were likely to be found offensive.

Familial and social support mechanisms were examined by looking at whether respondents would seek advice from their own relations or would make use of existing health services in the community. These questions were all open-ended and involved the interviewer asking the respondent a particular question and then offering a series of prompts.

Exercise habits were assessed by questioning respondents about their participation in certain types of activities. Respondents were asked if in the two weeks prior to the interview they had participated in any type of physical activity for health fitness, recreation, or sport; or had in the course of their daily activities been involved in moderate to heavy physical exertion.

Alcohol and tobacco consumption was measured by questioning respondents about their regular habits. Smoking status was defined in terms of whether the respondent was a current smoker, ex-smoker, or non-smoker. Respondents were also asked questions about the type of product and how much they smoked. Drinking habits were categorized in terms of daily and weekly consumption patterns. Only a few general questions on alcohol consumption were included because of the possibility that more specific questions would be found offensive.

Dietary information was collected using a 24-hour dietary recall method and a food frequency questionnaire on salt intake. Respondents were asked to recall what they ate and drank on the day prior to the interview. Each meal or snack, beginning with the first one in the morning, was seen as a distinct recall event and was recorded on a separate data record sheet. For each recall event, respondents were asked: to describe what they ate and drank, including information on the cooking methods and portion sizes of the food items they consumed; and to remember where the meal or snack was eaten and whether it was prepared at home or purchased (see section 7.2 in chapter 7 for further details about the dietary recall methods employed).

Salt intake was measured using a simple questionnaire (Millar and Beard, 1988). The foods named in this checklist were chosen to represent a comprehensive list of sources of added sodium in a Western diet. A respondent's salt intake was assessed by summing the scores of the 21 items on the checklist — a score of zero would indicate that salt had not been added in cooking or at the table and no prepared food with added salt had been eaten.

Finally, respondents were given the opportunity to have information on their cardiovascular fitness passed on to their physician or to add any further comments relevant to the interview.

5.4.2 Survey operation

The survey aimed at interviewing as many Aboriginal households and individuals aged 18 years and over as possible. With this goal in mind, a non-random sample¹ of households and individuals was obtained by using local Aboriginal interviewers who themselves chose the people they interviewed. In total, information was collected on 62 households and 273 individuals, of whom 159 were male and 114 were female. Based on 1986 census figures, this non-random sample represents 70 per cent of the men and 48 per cent of the women aged 18 years and over in the total Bourke Aboriginal population (see Table 5.1).

The hiring of Aboriginal interviewers was seen as the most efficient and least intrusive way of surveying the Aboriginal community. Competent interviewers were selected by the staff of the Aboriginal Health Service from as many of the more prominent families in the community as possible in the hope of maximizing the total number of possible respondents and the overall representativeness of the sample. After appointment, each interviewer attended a training workshop which focused on teaching the interviewers about various interviewing procedures, such as first and last contact with a respondent, interviewing techniques, and confidentiality. Interviewers were paid per interview and worked at their own pace. Although there seemed to be little variation in the quality of interviews, interviewers tended to choose individuals of the same age, sex, and family relations as themselves. The fact that more male than female and younger than older interviewers were hired may in part explain why more young men than any other group were interviewed.

Households were first made aware of the survey through contact with the interviewer. The purpose of this contact was to inform the household and potential individual respondents about: the purpose of the survey and how people could participate; the type

1. In these types of surveys researchers generally opt for non-random rather than random sampling techniques (see Sibthorpe, 1988: 161-164).

of questions that would be asked and what would be done with the information; and the rights of respondents and how confidentiality would be maintained. If a household (generally the household head) or individual agreed to participate in the survey, an interview time was arranged at their convenience. During the interview, the interviewer asked the questions in sequence and watched to see if the respondent fully understood the question. At all times during the interview the respondent had the right to look at the interview schedule and to correct mistakes. Additionally, at the end of the interview respondents were read back what they had said to the interviewer.

All the questionnaires remained in Bourke as property of the Aboriginal Health Service. Preliminary coding was done on coding sheets while I was in Bourke, and final data entry was done at the Australian National University from these coding sheets using two standard data entry computer programs. The dietary data were entered onto a program called DIET / 1 (Xyris Software, 1989a) and the household and remaining individual data were entered onto a program called dSURVEY (Corner, 1989). Once the coding of interviews and data entry had been completed, the questionnaires and log books were destroyed as had been planned. The Aboriginal Health Service was left with copies of the coding sheets and appropriate disk copies and printouts of the data files.

5.4.3 Sampling and selection biases

The non-random sampling approach taken in this study is a major source of potential bias, as the background characteristics and responses of those who participated may not be truly representative of the total Bourke Aboriginal population. Sampling bias affects external validity and will occur if the subjects in the non-random sample (the survey population) are not representative of the reference population in the way in which they respond to the questions (Dwyer, 1988). In an attempt to measure this type of bias, three comparisons were made of the demographic characteristics of the survey

population and the total Bourke Aboriginal population (based on 1986 census figures) — population structure, marital status, and employment and occupational status.

Many differences between the two populations are observed when population structure is compared (see Table 5.1). Among males, the survey population has a larger proportion under 30 years of age and a smaller proportion over 30 years of age than does the total population; while among females, the survey population has a similar distribution of individuals except that there are more females aged 18 to 19 years.

The comparison of marital status shows that the survey population includes more single people than does the total population (see Table 5.2). Nearly 90 per cent of the male sample and 75 per cent of the female sample are single (which compares with 64 per cent of the males and 57 per cent of the females in the total population). However, 30 per cent of the single men and 35 per cent of the single women in the sample are in *de facto* unions. These differences in the proportion married most likely reflect disparities in the age structures of the two populations.

The comparison of employment and occupational status shows that the survey population includes more persons unemployed and of those who are employed, more working in professional or semi-professional occupations than does the total population (see Tables 5.3 and 5.4). However, as with the above comparison of population structure, the sample of females appears to be much more representative of the total population than the sample of males. These differences in employment and occupational status may to some extent reflect the increasing opportunities Aborigines have had in the last five years (or since the census was taken in 1986) to be employed in semi-professional occupations. For example, several semi-professional positions were created with the establishment of the Aboriginal Health Service in 1987.

Along with problems of representativeness, or sampling bias, there are problems of selection bias in this study. This type of bias can affect the internal validity of a study

in such a way that conclusions drawn from the study are not even valid for the sample, let alone for the reference population (Dwyer, 1988). In this case, selection bias poses a problem because of the manner in which anthropometric, clinical, and locational data were obtained. Although all respondents were asked to go to the Aboriginal Health Service to get their height, weight, and blood pressure measured, only 30 per cent actually participated in this part of the survey, and only a third of these respondents (or 10 per cent of the survey population) had their serum cholesterol measured. This low participation rate was in part due to the fact that the Reflotron system for measuring serum cholesterol was on loan to the Aboriginal Health Service for only a short period of time. Additionally, although the individual questionnaire did not specifically ask in what part of town a respondent lived, some information was obtained indirectly. An informant was employed to identify whether respondents lived east or west of the general post office (GPO). This geographical classification crudely divided the respondents between those who lived on or near the mission (west of the GPO) and those who lived in town (east or northeast of the GPO). As shown in Table 5.5, this technique was rather unsuccessful because the informant only knew local Aborigines by their nicknames and not by their first or last names. Locational data was obtained for only 82 male and 53 female respondents. Not including the nicknames of individuals on the questionnaires was an error that could not have been foreseen.

In the case of anthropometric and clinical data, there tends to be a greater proportion of individuals over the age of 35 years in the two subsamples than in the total sample (the survey population). With locational data, the distribution of respondents by sex and age in the subsample does not differ significantly from the overall distribution found in the total sample (see Table 5.5).

Table 5.1 Comparison of the age and sex structure of a non-random sample of Bourke Aborigines¹ and the Bourke Aboriginal population², for persons aged 18 years and over³.

Age group	Males		Females	
	Survey	Census	Survey	Census
18-19	36 (23)	21 (9)	22 (19)	23 (10)
20-29	76 (48)	80 (35)	47 (41)	99 (42)
30-39	31 (20)	59 (26)	23 (20)	46 (19)
40-49	10 (6)	29 (13)	13 (11)	31 (13)
50 plus	4 (3)	38 (17)	9 (8)	37 (16)
All ages ⁴	157 (100)	227 (100)	114 (100)	236 (100)

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Source: Australian Bureau of Statistics 1986 census of population and housing.

3. The numbers in brackets are percentages and may not add up because of rounding.

4. Excluding two men surveyed who did not report their ages.

Table 5.2 Comparison of the marital status of a non-random sample of Bourke Aborigines¹ and the total Bourke Aboriginal population², by sex and age, for persons aged 18 years and over.

Age group	Survey			Census		
	Never Married	Married	Formerly Married	Never Married	Married	Formerly Married
Males						
18-19	33	2	0	15	0	0
20-29	71	3	0	60	4	2
30-39	24	6	0	25	17	6
40-49	6	3	0	5	12	0
50 plus	1	2	1	5	17	5
All ages	135	16	1	110	50	13
Proportion at all ages ³	89	11	0	64	29	8
Females						
18-19	21	0	1	17	0	0
20-29	40	5	0	65	10	7
30-39	14	5	1	17	14	1
40-49	3	5	1	4	13	10
50 plus	0	1	6	4	17	8
All ages	78	16	9	107	54	26
Proportion at all ages ³	76	16	9	57	29	14

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Source: Australian Bureau of Statistics 1986 census of population and housing.

3. Percentages may not add up because of rounding.

Table 5.3 Comparison of the labour force participation rates of a non-random sample of Bourke Aborigines¹ and the total Bourke Aboriginal population², by sex and age, for persons aged 18 years and over.

Age group	Survey			Census		
	Employed	Unemployed	Not in labour force	Employed	Unemployed	Not in labour force
Males						
18-19	3	27	5	7	17	20
20-29	21	50	4	25	27	9
30-39	8	19	4	24	12	7
40-49	4	5	1	8	5	5
50 plus	0	0	4	6	5	15
All ages	36	101	18	70	66	56
Proportion at all ages ³	23	65	12	36	34	29
Females						
18-19	0	14	8	3	12	30
20-29	7	7	31	18	15	40
30-39	6	1	15	8	4	20
40-49	2	2	9	6	3	15
50 plus	0	0	9	1	0	23
All ages	15	24	72	36	34	128
Proportion at all ages ³	14	22	65	18	17	65

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Source: Australian Bureau of Statistics 1986 census of population and housing.

3. Percentages may not add up because of rounding.

Table 5.4 Comparison of the occupational status of a non-random sample of Bourke Aborigines¹ and the total Bourke Aboriginal population², by sex, for persons aged 18 years and over³.

Occupation	Survey		Census	
	Males	Females	Males	Females
Managers and administrators	0 (0)	1 (7)	1 (1)	0 (0)
Professionals and para-professionals	13 (35)	6 (43)	7 (11)	5 (14)
Tradespersons	6 (16)	1 (7)	5 (8)	2 (6)
Clerks, salespersons and personal service workers	3 (8)	4 (29)	5 (8)	26 (74)
Plant and machine operators	1 (3)	1 (7)	11 (17)	0 (0)
Labourers	14 (38)	1 (7)	35 (55)	2 (6)
Total	37 (100)	14 (100)	64 (100)	35 (100)
Not stated ⁴	0	0	6	2
Total employed	37	14	70	37

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Source: Australian Bureau of Statistics 1986 census of population and housing.

3. The numbers in brackets are percentages.

4. Insufficient information or not stated.

Table 5.5 The location of residence¹⁻² for a non-random sample of Bourke Aborigines³ aged 18 years and over, by sex and age.

Age group	Males		Females	
	Mission	Town	Mission	Town
18-19	14 (33)	11 (28)	10 (36)	5 (20)
20-29	20 (48)	19 (48)	8 (29)	11 (44)
30-39	4 (10)	8 (20)	6 (21)	5 (20)
40-49	3 (7)	1 (2)	4 (14)	1 (4)
50 plus	1 (2)	1 (2)	0 (0)	3 (12)
All ages	42 (100)	40 (100)	28 (100)	25 (100)

1. Mission refers to people living on or near the mission; while town refers to people living in town.

2. The numbers in brackets are percentages.

3. Aboriginal risk factor prevalence survey conducted in April and May 1989.

5.5 Aboriginal health and health care

Before discussing the biological and behavioural risk factor prevalence levels of Aborigines in Bourke (see chapters 6 and 7), some mention needs to be made of their current health status. In this study the health status of adult Aborigines was evaluated by assessing current patterns of Aboriginal and non-Aboriginal mortality and Aboriginal adult survivorship; and by determining community levels of adult morbidity, health care services use, and familial support. Although this appraisal is rather limited, it does provide some insight into the existence of community mortality and morbidity differentials and the reliance of Aborigines on health care services and family members for medical care, support, and advice.

5.5.1 Direct estimates of Aboriginal and non-Aboriginal mortality

Although no outright comparisons have been made of regional differences in mortality, death rates for the western New South Wales population (as for the populations of other rural areas of the state) as a rule seem to be higher than those for the total New South Wales population. These differences are generally assumed to be a result of the higher proportion of Aborigines and lower proportion of ethnic-born Australians in rural areas. To test whether these assumptions are correct, potential differences in the levels of Aboriginal and non-Aboriginal mortality were assessed using information collected on all deaths and their causes in Bourke and Brewarrina from 1979 to 1988.

Differences in Aboriginal, non-Aboriginal, and total New South Wales mortality regimes were evaluated by constructing life tables and age- and cause-specific mortality rates. As in chapter 4 when comparing changes in Aboriginal mortality over time (see section 4.3.2), life expectancy was evaluated by constructing a measure of standard error around each e_x^o value (the life expectancy at age x) so as to provide reasonable estimates of the possible variability in life tables caused by small numbers and to compare Aboriginal with non-Aboriginal expectation of life. Age- and cause-specific rates were appraised by constructing relative risk ranges based on a ratio of the

Aboriginal or non-Aboriginal rate to the 1986 total New South Wales rate. Finally, to limit the effects of small numbers, age intervals were grouped and the calculation of relative risk ranges was limited to a small selection of relevant causes of death and to age- and cause-specific rates based on five or more deaths.

For the 1979-1988 period, Aboriginal life expectancy at birth in Bourke and Brewarrina was 47 years for males and 61 years for females and was notably lower than figures calculated for Aboriginal males and females in western New South Wales, 1984-1987 (see Tables A1.14 and A1.15 in Appendix 1). In fact, these figures represent a significant gap of six years for males and five years for females between community and regional life expectancies at birth. Among females, this difference is not significant after 34 years of age; while among males, it remains significant at all ages. Non-Aboriginal life expectancy at birth in these two communities was estimated to be 76 years for males and 84 years for females. However, these figures are probably fairly inaccurate because of the small number of deaths occurring at younger ages and the large number at older ages (see Table 5.7). Non-Aboriginal sex-specific life expectancies in these two communities are probably much closer to figures for the total New South Wales population in 1986 of 73 years for males and 79 years for females (Australian Bureau of Statistics, 1988c). Nevertheless, Aborigines in Bourke and Brewarrina can expect to live approximately 20 years less than their non-Aboriginal counterparts.

Substantial disparities in the levels of Aboriginal and non-Aboriginal crude and age-specific mortality also exist in these two communities. As shown in Table 5.6, when differences in the Aboriginal and total New South Wales age structures are standardized, Aborigines have a death rate for males which is six times and for females which is almost three times the total New South Wales rate in 1986. In comparison, males and females in the non-Aboriginal population of Bourke and Brewarrina have death rates which are slightly lower than or the same as the total New South Wales

population. Aborigines also have much higher age-specific death rates than non-Aborigines (see Table 5.7). Among males, Aboriginal rates were found to be higher than non-Aboriginal at all ages with the difference in relative risk of death being highest between the ages of 25 and 64 years; while among females, the most prominent differences in age-specific death rates were found for those under one year of age and 45 years of age and over.

When cause-specific death rates are compared, the most apparent difference between Aborigines and non-Aborigines in the two communities is in their rates of circulatory system diseases (see Tables 5.8 and 5.9). Among Aborigines, the rate of death from heart disease was at least five times higher for males and at least two times higher for females than the rates for their non-Aboriginal or total New South Wales counterparts. As a rule, Aboriginal cause-specific death rates tend to be much higher than non-Aboriginal or total New South Wales rates; while non-Aborigines tend to have cause-specific death rates very similar to or lower than those experienced by the total New South Wales population.

These results clearly suggest that the Aboriginal as opposed to the non-Aboriginal mortality regime is the most important factor in increasing the overall level of mortality in Bourke and Brewarrina. Aborigines have appallingly high death rates, especially during adulthood and most notably from circulatory system diseases; while non-Aborigines have age- and cause-specific death rates very similar to those found among the total New South Wales population. These non-Aboriginal rates indicate that even if there is a lower proportion of ethnic-born Australians among the non-Aboriginal population in Bourke and Brewarrina, this lower proportion has little effect on the overall level of mortality.

Table 5.6 Crude and standardized death rates¹ and standardized mortality ratios² for Aborigines and non-Aborigines living in two western New South Wales communities³, 1979-1988, and the total New South Wales population⁴, 1986, by sex.

	Males	Females
Aboriginal, 1979-1988		
Total number of deaths	116	55
Crude death rate	17.4	8.0
Standardized death rate	49.3	20.3
Non-Aboriginal, 1979-1988		
Total number of deaths	184	95
Crude death rate	6.9	4.1
Standardized death rate	7.3	5.4
New South Wales, 1986		
Total number of deaths	22,571	19,596
Crude death rate	8.2	7.1
Standardized mortality ratio		
Aboriginal standardized rate to New South Wales crude rate	603 (504 - 727)	287 (219 - 385)
Non-Aboriginal standardized rate to New South Wales crude rate	90 (78 - 105)	76 (62 - 94)

1. Rates are deaths per 1,000 population per year. Aboriginal and non-Aboriginal crude death rates were directly standardized to the New South Wales age structure, 30 June 1986.

2. A ratio of expected Aboriginal or non-Aboriginal deaths to the total number of deaths in New South Wales in 1986. Confidence intervals, at a 95 per cent level of confidence, are designated by the ratios shown in the brackets. The ratios have been multiplied by 100.

3. Based on mortality data for Bourke and Brewarrina (see section 4.2 in chapter 4).

4. Source: Australian Bureau of Statistics (1988b).

Table 5.7 Age-specific mortality rates (ASMR)¹ and relative risk² for Aborigines and non-Aborigines living in two western New South Wales communities³, 1979-1988, and the New South Wales population⁴, 1986, by sex.

Age group	Community		New South Wales		Relative risk range
	Deaths	ASMR	Deaths ⁵	ASMR	
Aboriginal Males					
0	8	42.1	436	10.3	(2.3 - 7.9)
1-24	10	2.4	843	0.8	(1.6 - 6.6)
25-44	24	14.3	1,338	1.6	(6.2 - 13.9)
45-54	20	54.1	1,428	5.0	(6.9 - 18.0)
55-64	19	135.7	3,843	14.5	(6.2 - 15.0)
65 plus	35	269.2	14,671	58.0	(3.3 - 6.7)
Non-Aboriginal Males					
0	2	4.2	436	10.3	-
1-24	10	1.0	843	0.8	(0.7 - 2.2)
25-44	10	1.2	1,338	1.6	(0.5 - 1.2)
45-54	22	7.1	1,428	5.0	(0.9 - 2.3)
55-64	39	14.4	3,843	14.5	(0.7 - 1.6)
65 plus	101	47.9	14,671	58.0	(0.6 - 1.1)
Aboriginal Females					
0	6	21.4	323	8.0	(1.5 - 5.6)
1-24	5	1.2	350	0.3	(1.9 - 7.3)
25-44	13	8.3	624	0.8	(7.0 - 18.3)
45-54	8	16.3	810	3.0	(2.3 - 16.9)
55-64	5	20.0	2,079	7.8	(1.4 - 5.6)
65 plus	18	105.9	15,408	43.6	(1.7 - 3.6)
Non-Aboriginal Females					
0	0	0.0	323	8.0	-
1-24	2	0.2	350	0.3	-
25-44	1	0.2	624	0.8	-
45-54	3	1.3	810	3.0	-
55-64	13	6.4	2,079	7.8	(0.4 - 1.8)
65 plus	76	35.5	15,408	43.6	(0.6 - 1.0)

1. Rates are deaths per 1,000 population per year.

2. A ratio of the observed Aboriginal or non-Aboriginal rate to the total New South Wales rate. The relative risk range represents confidence intervals, at a 95 per cent level of confidence, around each ratio. A relative risk range was calculated for age-specific mortality rates based on five or more deaths.

3. Based on mortality data for Bourke and Brewarrina (see section 4.2 in chapter 4).

4. Source: Australian Bureau of Statistics (1988b).

5. Excluding 'age not stated'.

Table 5.8 Male cause-specific mortality rates (CSMR)¹, relative risk², and proportion of excess risk³ for Aborigines and non-Aborigines living in two western New South Wales communities⁴, 1979-1988, as compared with the New South Wales population (NSW)⁵, 1986.

Cause of death	Community Deaths	CSMR	NSW CSMR	Relative risk range	Proportion of excess risk
Aboriginal					
All causes	116	49.3	8.2	(5.0 - 7.3)	100
Malignant neoplasms	9	5.3	2.1	(1.3 - 5.6)	8
Circulatory system	51	26.6	3.8	(5.3 - 9.5)	56
Respiratory system	12	6.5	0.6	(5.9 - 19.9)	14
Digestive system	12	3.4	0.3	(6.8 - 23.2)	8
Accidents, poisonings and violence	9	1.8	0.7	(1.4 - 5.9)	3
Other causes	23	5.7	0.8	(5.1 - 11.6)	12
Non-Aboriginal					
All causes	184	7.3	8.2	(0.8 - 1.0)	100
Malignant neoplasms	39	1.5	2.1	(0.5 - 1.0)	64
Circulatory system	85	3.4	3.8	(0.7 - 1.1)	40
Respiratory system	21	0.9	0.6	(0.9 - 2.3)	-30
Digestive system	4	0.2	0.3	-	15
Accidents, poisonings and violence	24	0.9	0.7	(0.9 - 2.1)	-26
Other causes	11	0.4	0.8	(0.3 - 1.2)	37

1. Rates are deaths per 1,000 population per year. Aboriginal and non-Aboriginal crude death rates were directly standardized to the New South Wales age structure, 30 June 1986.

2. A ratio of the directly standardized Aboriginal or non-Aboriginal rate to the total New South Wales rate. The relative risk range represents confidence intervals, at a 95 per cent level of confidence, around each ratio. A relative risk range was calculated for cause-specific mortality rates based on five or more deaths.

3. Excess risk was calculated by subtracting cause-specific death rates for the total New South Wales population from directly standardized Aboriginal and non-Aboriginal rates. The proportion of excess risk was then derived by producing a ratio of cause-specific excess risk to the excess risk for all causes.

4. Based on mortality data for Bourke and Brewarrina (see section 4.2 in chapter 4).

5. Source: Australian Bureau of Statistics (1988c).

Table 5.9 Female cause-specific mortality rates (CSMR)¹, relative risk², and proportion of excess risk³ for Aborigines and non-Aborigines living in two western New South Wales communities⁴, 1979-1988, as compared with the New South Wales population (NSW)⁵, 1986.

Cause of death	Community Deaths	CSMR	NSW CSMR	Relative risk range	Proportion of excess risk ⁵
Aboriginal					
All causes	55	20.3	7.1	(2.2 - 3.8)	100
Malignant neoplasms	6	2.5	1.6	(0.7 - 4.1)	6
Circulatory system	23	10.8	3.7	(1.9 - 4.6)	54
Respiratory system	2	1.0	0.4	-	4
Digestive system	3	1.1	0.3	-	7
Accidents, poisonings and violence	8	1.0	0.3	(1.7 - 7.8)	5
Other causes	13	3.9	0.8	(2.9 - 9.3)	23
Non-Aboriginal					
All causes	95	5.4	7.1	(0.6 - 0.9)	100
Malignant neoplasms	16	0.9	1.6	(0.3 - 0.9)	46
Circulatory system	54	3.1	3.7	(0.6 - 1.1)	36
Respiratory system	6	0.3	0.4	(0.4 - 2.4)	3
Digestive system	5	0.3	0.3	(0.5 - 3.7)	-3
Accidents, poisonings and violence	4	0.2	0.3	-	7
Other causes	10	0.6	0.8	(0.4 - 1.6)	11

1. Rates are deaths per 1,000 population per year. Aboriginal and non-Aboriginal crude death rates were directly standardized to the New South Wales age structure, 30 June 1986.

2. A ratio of the directly standardized Aboriginal or non-Aboriginal rate to the total New South Wales rate. The relative risk range represents confidence intervals, at a 95 per cent level of confidence, around each ratio. A relative risk range was calculated for cause-specific mortality rates based on five or more deaths.

3. Excess risk was calculated by subtracting cause-specific death rates for the total New South Wales population from directly standardized Aboriginal and non-Aboriginal rates. The proportion of excess risk was then derived by producing a ratio of cause-specific excess risk to the excess risk for all causes.

4. Based on mortality data for Bourke and Brewarrina (see section 4.2 in chapter 4).

5. Source: Australian Bureau of Statistics (1988c).

5.5.2 Indirect estimates of Aboriginal mortality

An indirect method of estimating adult Aboriginal mortality was used to evaluate the validity of the above direct estimates and to assess the past duration of current patterns of adult survivorship. In this case, adult sex-specific mortality rates were indirectly estimated using the Brass methods of estimating conditional adult survivorship from maternal and paternal orphanhood data (United Nations, 1983: 97-107). These methods require information on the proportion of respondents with a surviving mother or father and on the mean age of mothers or fathers at the birth of their children. Owing to the lack of data on registered births, estimates of the mean age of maternity and paternity were based on values used by Gray (1987a) for Aborigines in northern New South Wales. Adult mortality estimates were produced in the form of survivorship ratios which measure the probability that men aged 25 years and women aged 35 years (the estimated mean ages of paternity and maternity) will still be living at increasing age increments from these ages.

As already stated, the household questionnaire used in the survey collected demographic details of all persons living in a household, including their sex, age or date of birth, and whether their parents were still alive (see section 5.4.1 and household data sheet 1 in Appendix 2). In total, information on the survival of parents was obtained from a non-random sample of 229 individuals, or 32 per cent of the total Bourke Aboriginal population in 1986.

As shown in Table 5.10, the proportion of Bourke Aborigines with both mother and father living decreases quite rapidly with age; by the time individuals reach 40 years of age or more, it is very unlikely that both or even one parent will be living. For the 20-29 years age group, 71 per cent have both parents living, 18 per cent have only a mother living, 3 per cent have only a father living, and 8 per cent have neither parent living. This pattern of the survival of parents seems to closely resemble that obtained by the

Aboriginal Family Demography study for Aborigines living on the north coast of New South Wales (Gray, 1987a).

The estimation of adult survivorship from Bourke Aboriginal paternal and maternal orphanhood data was rather more successful for women than for men (see Table 5.11). Survival ratios calculated for Bourke Aboriginal females are strikingly similar to those produced for Aboriginal females living in western New South Wales, 1984-1987. For example, the survivorship of women aged from 25 to 50 years was 0.88 for Bourke and 0.90 for western New South Wales. In fact, only for women aged from 25 to 70 years is there a substantial difference in survivorship estimates. However, this rather low estimate of survivorship is most probably due to the effects of small numbers.

The close similarity of mortality rates for female Bourke and western New South Wales Aborigines is not surprising, considering that there are very few differences in female life expectancies between these two groups after the age of 25 years. As shown in Table A1.15 in Appendix 1, differences in life expectancy after age 25 in Bourke and Brewarrina, 1979-1988, and western New South Wales, 1984-1987, were generally not significant. In fact, the critical ratios which were produced suggest that only at ages 25 to 34 years were life expectancies significantly different at a 0.01 probability level.

The survivorship ratios produced here for female Bourke Aborigines are not all contemporaneous; rather, they refer to specific periods in time before the survey was conducted. For example, the time estimate for the survival of women from ages 25 to 70 years, produced on the last row and column of Table 5.11, is based on reported deaths which occurred approximately 17 years ago to mothers of persons now between the ages of 40 and 50 years. Overall, these time estimates suggest that the current high mortality rates among female Bourke Aborigines have existed for a relatively long time — at least 17 years.

Indirect estimates of male survivorship were on the whole much less satisfactory. In fact, the pattern of male mortality described here is not plausible, because male survivorship does not decrease with age. The Aboriginal Family Demography study on the north coast of New South Wales had a similar problem of estimating male survivorship. Gray (1987a: 9-13) attributed this difficulty to a variety of underlying causes including: the quality and reliability of collected data; the intrusion of high adult male mortality into the childbearing years; and the likely difference between the survivorship of married and unmarried men. These reasons almost certainly were important here as well.

Conclusions which can be drawn from this exercise are certainly not clear-cut. For women, estimates of adult survivorship suggest that the pattern of mortality in Bourke is virtually identical to that in western New South Wales from 1984 to 1987 and that this pattern has existed for at least 17 years. However, no generalizations can be drawn from the estimated male survivorship ratios. Overall, the evidence presented here at least partially supports the idea that high adult Aboriginal mortality is not a new phenomenon, but one that has existed for some time.

Table 5.10 Survival of parents of Bourke Aborigines based on a non-random sample¹ of adults aged 18 years and over.

Age group	Parents of Bourke Aborigines						North coast coast (per cent) ²
	Total	Neither living	Only father living	Only mother living	Both living	Both living (per cent)	
0-4	37	0	0	1	36	97	96
5-9	27	0	0	2	25	93	95
10-14	17	0	0	0	17	100	85
15-19	42	1	0	4	37	88	70
20-29	62	5	2	11	44	71	69
30-39	25	4	2	4	15	60	26
40 plus	19	12	1	4	2	11	6
All ages	229	22	5	26	176	77	63

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Based on the survival of both parents of 533 Aborigines on the north coast of New South Wales (Gray 1987a: 6, Table 1).

Table 5.11 Estimation of female adult Aboriginal mortality from the proportion of Aborigines with surviving mothers¹.

Age group	Proportion surviving	Survivorship ratios ²		Year to which estimates refer ⁴
		Bourke	Western NSW 1984-87 ³	
5-9	1.00	-	-	-
10-14	1.00	1.00	0.99	1984
15-19	0.98	0.99	0.96	1982
20-24	0.91	0.95	0.93	1980
25-29	0.83	0.88	0.90	1978
30-34	0.90	0.86	0.86	1977
35-39	0.67	0.81	0.79	1976
40-44	0.67	0.67	0.69	1975
45-49	0.17	0.39	0.61	1972

1. Source: Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. The probability that a woman aged 25 years will still be living at age 25+n, where n is the first age shown in column one.

3. Source: Gray and Hogg (1989: 13, Table 3).

4. Time estimates refer to specific periods of time for which the Bourke survivorship ratios applied.

5.5.3 Morbidity

Information on the general health of the Bourke Aboriginal community was obtained by asking respondents: (1) to assess their current health status and to compare it with that of other Aborigines of the same age; (2) to self-diagnose and report the presence of various disease conditions related to heart disease; and (3) to indicate if they had been in the hospital during the past year (see individual data sheets 5 and 7 to 9 in Appendix 2).

As shown in Table 5.12, respondents generally felt that they were in excellent or very good health and that they were in better health than other Aborigines the same age as themselves. Of the male respondents, 52 per cent indicated that they were in excellent or very good health, and half of these individuals (26 per cent) indicated that they were in better health than other Aborigines of the same age. On the other hand, female respondents were less likely to think of themselves as being healthy. Only 47 per cent of the female respondents implied that they were in excellent or very good health; and out of these only 18 per cent thought they were in better health than other Aborigines of the same age. Although these results seem to reflect an overall feeling of good health in the Bourke Aboriginal population, they may also reflect a bias in this non-random sample towards the opinion of younger adults.

As discussed in section 5.4.1, a series of diagnostic questions, designed by the London School of Tropical Medicine and Hygiene (Rose, 1962), were used to determine whether respondents had angina pectoris, a possible myocardial infarction, or intermittent claudication; while a series of general morbidity questions were used to determine whether respondents had ever been told by their doctor that they had any of the following conditions: high blood pressure, high blood cholesterol, diabetes, a heart attack, or a stroke.

The prevalence of these self-diagnosed and reported disease conditions tended to be underreported (see Table 5.13). Only 4 per cent of the male and 6 per cent of the

female respondents indicated that they had diabetes, when actually about 17 per cent of men and 15 per cent of women in Bourke are known to be diabetic (Cameron *et al.*, 1986). Angina seems also to have been underreported, as diagnosed levels (4 or 5 per cent) were somewhat lower than those observed for Aborigines in the west Kimberley region of Western Australia with cardiac ischaemia — inadequate blood flow to the heart caused by a constriction or blockage of the coronary arteries supplying it (Bastian, 1979; Gracey and Spargo, 1987). Likewise, intermittent claudication was most likely underreported, as no respondents identified themselves as having had this condition. There was also a noticeable difference in the proportion of male respondents who reported having a possible myocardial infarction and those who reported being told by a doctor that they had had a heart attack — 5 versus 1 per cent. Although this difference is difficult to explain, it may in part be due to the fact that in some cases individuals may not be aware that they have had a heart attack. Only self-reported high blood pressure prevalence levels appear to reflect actual levels — the proportion of respondents who reported having high blood pressure is similar to the proportion observed to be hypertensive from measured systolic and diastolic blood pressure readings (26 versus 29 per cent).

Rates of hospital admission were determined by asking respondents if they had been in hospital for any reason during the past year. For all ages, the Bourke Aboriginal self-reported hospital admission rate was 298 per 1,000, or 289 per 1,000 for males (43 cases out of 149 respondents surveyed) and 313 per 1,000 for females (31 cases out of 99 respondents surveyed). These sex-specific rates compare closely with an Aboriginal statewide rate in 1978 of 312 per 1,000 for males and 365 per 1,000 for females, but are somewhat higher than recently calculated hospital admission rates for the north coast of New South Wales of 223 per 1,000 for males and 229 per 1,000 for females (Yusuf and Hamilton, 1982; Thomson *et al.*, 1990). These apparent disparities may be due to the underlying differences in the age structure of these three populations or to the underreporting of hospital admissions, especially repeat visits, by Bourke Aborigines.

Differences in the rates of self-reported hospital admissions by sex and age group and various risk markers and factors were compared using odds ratios². In this case, odds ratios were constructed to measure the odds of reporting a hospital visit during the past year among respondents of a certain sex and age group or with a particular risk marker or factor, such as employment and marital status, education, and body mass index. Confidence intervals, at a 95 per cent level of confidence, were calculated around each odds ratio using Woolf's (1955) method. Differences between odds ratios among various categories were generally not significant (see Tables 5.15 and 5.16). The only exceptions appear to be with employment and smoking status. As a rule, respondents who were non-smokers or employed were significantly less likely to have been hospitalized than persons who were current or ex-smokers or who were unemployed. Although there are few significant differences in the odds ratios shown in these tables, there are many substantive differences. Among females, for example, the odds of reporting a hospital visit increases after 34 years of age; and for both sexes, education, location, body mass index, diastolic blood pressure, serum cholesterol, and vigorous exercise showed notable differences in the odds of reporting hospital visits.

Unquestionably this analysis of Aboriginal morbidity has highlighted several interesting associations between Aboriginal health status and the underlying economic, environmental, and social conditions in Bourke. As noted above, significant and substantive differences were observed in the reporting of hospital visits by sex and age group and various risk markers and factors. This type of finding is not new to Bourke. A recent survey noted a link between certain types of illnesses, especially those in children, and housing conditions (Harris and Davies, 1984; Harris *et al.*, 1984). For example, *Staphylococcus aureus* skin infections tend to occur at a much greater rate among individuals living in dwellings with no sewage or septic tanks, no bathing facilities, and/or no screens or glass on the windows; while the incidence of trachoma is

2. A ratio of the probability of one event to that of an alternative event.

higher among individuals living in households of six or more persons. What is new, however, is that such a wide range of factors have been implicated, and that the general perception of health in this community is so different from the actual reality.

Table 5.12 Self-reported health status and health status in comparison with others of the same age for a non-random sample of Bourke Aborigines¹ aged 18 years and over.

		In comparison with others			
Health status	n ²	Better (per cent) ³	Same (per cent) ³	Worse (per cent) ³	Total (per cent) ³
Males					
Excellent / very good	79	26	24	1	52
Good	42	14	13	1	28
Fair / poor	31	0	18	2	20
Total	152	40	56	4	100
Females					
Excellent / very good	48	18	26	3	47
Good	25	13	12	0	25
Fair / poor	29	10	17	2	28
Total	102	40	55	5	100

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Number of respondents, excluding 'not stated'.

3. Percentages may not add up because of rounding.

Table 5.13 Self-diagnosed and self-reported morbidity for a non-random sample of Bourke Aborigines¹ aged 18 years and over, by sex and age.

Condition	Males		Females	
	n ²	Per cent ³	n ²	Per cent ³
Self-diagnosed				
Angina				
18-24	80	4	55	2
25-34	46	4	27	4
35 plus	31	6	32	12
All ages	157	4	114	5
Possible myocardial infarction				
18-24	80	1	55	4
25-34	46	9	27	0
35 plus	31	10	32	6
All ages	157	5	114	4
Self-reported				
Diabetes				
18-24	77	1	55	4
25-34	46	4	27	0
35 plus	30	13	30	17
All ages	153	5	112	6
Heart attack				
18-24	77	0	55	2
25-34	43	2	22	0
35 plus	29	0	26	8
All ages	149	1	103	3
High serum cholesterol				
18-24	78	4	55	4
25-34	43	12	21	0
35 plus	28	14	27	30
All ages	149	8	103	10
High blood pressure				
18-24	80	14	55	13
25-34	46	20	27	33
35 plus	29	45	32	66
All ages	155	21	114	32

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Total number of respondents, excluding 'not stated'.

3. Proportion of respondents who self-diagnosed or reported the following conditions.

Table 5.14 The odds of relating self-reported hospitalization of a day or longer to sex and age and various risk markers for a non-random sample of Bourke Aborigines¹ aged 18 years and over.

		Period of hospitalization			
	n ²	Day patient	Overnight or longer	Odds ratio	Confidence interval ³
Males					
18-24	80	8	16	1.1	(0.5 - 2.3)
25-34	41	3	5	0.5	(0.2 - 1.2)
35 plus	28	7	4	1.8	(0.8 - 4.2)
All ages	149	18	25	-	-
Females					
18-24	52	2	13	0.8	(0.3 - 1.8)
25-34	21	2	4	0.8	(0.3 - 2.4)
35 plus	26	2	8	1.6	(0.6 - 4.0)
All ages	99	6	25	-	-
Both sexes					
Marital status					
Never had a partner	140	17	26	1.2	(0.7 - 2.1)
Married / <i>de facto</i>	99	7	20	0.9	(0.5 - 1.6)
Other	9	0	2	0.7	(0.1 - 3.4)
Employment status					
Employed	45	4	3	0.4	(0.2 - 0.9)
Unemployed	204	20	47	2.6	(1.1 - 6.3)
Secondary education⁴					
Yes	179	13	35	0.5	(0.2 - 1.0)
No	44	11	8	2.1	(1.0 - 4.1)
Location⁵					
Mission	67	8	10	0.8	(0.4 - 1.8)
Town	62	3	16	1.2	(0.6 - 2.6)

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Total number of respondents, excluding 'not stated'.

3. Confidence intervals, at a 95 per cent level of confidence, were calculated using Woolf's (1955) method.

4. Secondary education refers to people with at least some secondary level training.

5. Mission refers to people living on or near the mission; while town refers to people living in town.

Table 5.15 The odds of relating self-reported hospitalization of a day or longer to various risk factors for a non-random sample of Bourke Aborigines¹ aged 18 years and over.

		Period of hospitalization			
	n ²	Day patient	Overnight or longer	Odds ratio	Confidence interval ³
Body mass index					
Under and acceptable	23	2	7	0.8	(0.2 - 2.3)
Overweight	18	3	3	0.5	(0.2 - 1.8)
Obese	8	0	6	5.2	(0.9 - 29.1)
Diastolic blood pressure					
< 95 mm Hg	35	3	10	0.5	(0.2 - 1.8)
> 95 mm Hg	15	2	6	1.9	(0.6 - 6.6)
Serum cholesterol					
< 6.0 (mmol/litre)	14	2	2	0.3	(0.0 - 2.2)
> 6.0 (mmol/litre)	5	1	2	3.8	(0.4 - 31.6)
Alcohol					
Non-drinker	44	3	11	1.2	(0.6 - 2.3)
Drinker	205	21	38	0.9	(0.4 - 1.8)
Tobacco					
Non-smoker	90	2	10	0.2	(0.1 - 0.4)
Current smoker	118	16	30	2.3	(1.3 - 4.0)
Ex-smoker	33	6	9	2.2	(1.0 - 4.6)
Vigorous exercise					
Yes	87	5	15	0.6	(0.3 - 1.1)
No	163	19	35	1.7	(0.9 - 3.0)

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Total number of respondents, excluding 'not stated'.

3. Confidence intervals, at a 95 per cent level of confidence, were calculated using Woolf's (1955) method.

5.5.4 Health care

Information on health care in the Bourke Aboriginal community was obtained by asking respondents a series of open-ended questions, such as: what they thought of local health care practitioners and services; whether they sought medical care, support, or advice from family members or relatives; where they went for medical advice and care when they were sick; and if they were satisfied with the medical services available to them in Bourke (see individual interview data sheets 3 to 7 in Appendix 2). Respondents were reluctant to tell the interviewers what type of medical conditions they had treated in the hospital, so the question relating to this issue was dropped.

Health care practitioners were as a rule highly regarded. In times of medical need respondents sought the advice and attention of doctors (general practitioners) more often than hospital staff or Aboriginal health workers (see Table 5.16). Respondents principally saw the hospital as a facility to go to when the doctor's surgery was closed or when they had a referral from their doctor or an Aboriginal health worker. As one female respondent [ID 159] stated: 'It all depends what time it is in the day, if it's six or seven [in the evening] I would go to hospital'. The attention of an Aboriginal health worker was often sought for minor ailments or for organizing medical appointments with and transportation to the doctor or the hospital. The service given by the Aboriginal health workers was generally perceived as being more preventive in nature than treatment-oriented.

In regard to the quality of medical care in Bourke, respondents were generally happier with the advice and care given by doctors than by other services. The only perceived problem seen with the quality of care given by doctors was that there were not enough of them. As one female respondent [ID 49] stated: 'No [I am not satisfied], because only two doctors where there is a need for three'. This opinion was echoed by other respondents, many of whom thought the wait for an appointment to see a doctor was much too long. The hospital was also seen in a favourable light, even though a few

respondents remarked unfavourably about the quality of the nursing care or the waiting time before receiving medical attention. The Aboriginal Health Service had a lower approval rating than either the doctors or the hospital. Even though the majority of respondents, especially males, thought the service was very adequate and good for Aboriginal people, a small minority were dissatisfied. Perceived problems with the Health Service almost certainly reflect underlying differences in opinion between leading Aboriginal families on how the service should be operated.

Most respondents who were hospitalized were happy with the care and medical attention they received in the hospital. However, quite a large minority — 30 per cent of the male and 26 per cent of the female respondents — did not seek medical attention right away. In fact, one or two respondents waited for a week or more before going to the hospital. Once they were admitted the majority of them were satisfied with the treatment and care they received from the doctors and nurses. Additionally, most felt that the doctors and nurses kept them informed about their treatment and care. One female respondent [ID 105] said: 'Yes [the nurses and doctors did tell me everything about what was going on], but also I made it a point of knowing'.

Surprisingly, a greater proportion of men than women (48 versus 43 per cent) noted that they would seek care, support, or advice from one or more family member. However, females seem to play a more important role in giving support or care than males. In total, 41 per cent of the male and 47 per cent of the female respondents who sought the care, support, or advice of a relative chose their mother, who was by far the most important relative (see Table 5.17). Other important categories of relatives included brothers, sisters, father, cousins, aunts, and uncles.

As a rule, the health care practitioners in Bourke, especially the doctors, were perceived as being important sources of medical care and advice, which almost certainly reflects the dominant role doctors have played in Aboriginal health care and research over the last twenty to thirty years. As Coolican states (1974: 127):

In western New South Wales, the medical care of the rural Aborigines has always been the private charity of the general practitioner. Their state of health reflects the interest that the doctor and those who work with him have in the care of the poorest of the poor.

Overall, Bourke Aborigines seem happy with and make use of the wide variety of health care services and specialists available to them, yet adult Aboriginal mortality, especially from heart disease, remains excessive — at a higher level than what is experienced by almost any other Aboriginal community in western New South Wales.

Table 5.16 Use of and satisfaction with health care practitioners for a non-random sample of Bourke Aborigines¹ aged 18 years and over, by sex.

Category	Males		Females	
	n ²	Yes (per cent) ³	n ²	Yes (per cent) ³
Aboriginal health worker				
Would approach for advice and care	152	86	103	73
Satisfied with care	146	88	98	81
Doctors				
Would approach for advice and care	153	100	103	99
Satisfied with care	120	98	88	98
Hospital⁴				
Would approach for advice and care	148	94	102	89
Satisfied with care	120	92	83	96
Care while in hospital⁵				
Went right away	43	70	31	74
Satisfied with care from doctors	43	95	30	97
Satisfied with care from nurses	43	95	31	90
Kept informed about treatment or care	43	91	31	84

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Total number of respondents, excluding 'not stated'.

3. Percentages shown here are not cumulative, but instead each cell reflects the proportion of individuals with affirmative responses.

4. The care given by both doctors and nurses.

5. For persons hospitalized for a day or longer.

Table 5.17 The relative from whom advice was sought for a non-random sample of Bourke Aborigines¹ aged 18 years and over, by sex.

Category	Males (per cent) ²	Females (per cent) ²
Male relatives		
Son	1	4
Nephew	3	4
Brother ³	36	6
Partner	-	2
Father	16	12
Uncle	11	4
Grandfather	3	2
Female relatives		
Daughter	1	4
Niece	0	2
Sister ⁴	29	33
Partner	4	-
Mother	41	47
Aunt	15	12
Relatives of either sex		
Child	0	2
Cousin	20	10
Family	3	12
Other relatives	4	2
Persons surveyed⁵		
	75	49
Relatives mentioned⁶		
	137	77

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Percentages shown here are not cumulative, but instead each cell reflects the proportion of individuals who sought advice from that category of relative.

3. Including step-brother.

4. Including step-sister.

5. Total number of respondents, excluding 'not stated'.

6. Total number of relatives mentioned by respondents.

5.6 Summary

This chapter has described the manner in which the risk factor prevalence survey was conducted and has briefly outlined the health status of adult Bourke Aborigines.

Several important points have been raised.

Most importantly, the chapter has clarified how the non-random sampling and selection approaches taken in the survey are major sources of potential bias. As a rule, the male portion of the sample was less representative than the female portion, while both were more youthful than the total reference population. Additionally, the subsample who had their blood pressure, height, weight, and serum cholesterol measured tended to be older than the total sample (the survey population). These problems suggest that conclusions drawn in this study should be viewed with appropriate caution.

The limited appraisal of the health status of Bourke Aborigines has highlighted some major health concerns. In particular, this review makes note of the striking contrast between non-Aboriginal and Aboriginal death rates and the appallingly high levels of Aboriginal circulatory system disease mortality in Bourke and Brewarrina. Among Aborigines, the rate of death from heart disease was five times higher for men and two times higher for women than respective rates for either non-Aborigines in Bourke and Brewarrina or the total New South Wales population. Furthermore, parental survivorship data from a non-random sample of Bourke Aborigines suggest that the current pattern of mortality among female Aborigines has existed for some time — at least 17 years. This conclusion seems realistic considering that Kamien (1976: 38) noted that between 1967 and 1971 Bourke Aborigines aged 15 years and over had a death rate of 18 per 1,000 and that 64 per cent of these deaths were from ischaemic heart disease.

In terms of Bourke Aboriginal morbidity, associations were noted between reported hospital admissions and underlying economic, environmental, and social conditions. Respondents who were non-smokers or employed were significantly less likely to have

been hospitalized than respondents who were current or ex-smokers or who were unemployed. There were also substantive differences in the odds of reporting hospital visits by education, location, body mass index, diastolic blood pressure, serum cholesterol, and vigorous exercise.

Health care services and practitioners were as a rule highly regarded by the Bourke Aboriginal community, but in times of medical need most respondents sought the advice and attention of doctors. Aboriginal health workers had the lowest approval rating. However, this rating most likely reflects divisions within the community about the way in which the Aboriginal Health Service should be operated.

Overall, Bourke Aborigines perceive themselves as being healthy and appear to make use of and to be amply aware of the health care services available to them. Why then does the overall level of adult morbidity and mortality, especially from heart disease, remain so high? In the next two chapters I will attempt to answer this and other questions by examining the prevalence levels of various risk factors of coronary heart disease.

CHAPTER SIX

BIOLOGICAL AND BEHAVIOURAL RISK FACTORS

6.1 Introduction and overview

Although Australian Aborigines suffer from a high rate of circulatory system disease mortality (see Lincoln *et al.*, 1983; Smith *et al.*, 1983; Health Department of Western Australia, 1985; Plant, 1988; Gray and Hogg, 1989; Khalidi, 1989; Divakaran-Brown and Honari, 1990; Hicks, 1990), little is actually known about the prevalence of and association between the biological and behavioural risk factors of heart disease in this population. Over the past 15 years only three major risk factor prevalence studies¹ have been conducted among Aborigines — in Inverell (Simons *et al.*, 1981) and Wilcannia, New South Wales (Stephenson and Lenz, 1990), and in the west Kimberley region of Western Australia (Bastian, 1979). In addition, a broad range of studies relating to Aboriginal health (see Aboriginal Health Service, n.d.; Schwartz *et al.*, 1957; Casley-Smith, 1959; Charnock *et al.*, 1959; Abbie and Schroder, 1960; Kamien, 1975a, 1975b, 1976; Kamien *et al.*, 1974, 1975a; Cameron *et al.*, 1986; Sladden *et al.*, 1986; Sibthorpe, 1988; Watson *et al.*, 1988; Thomson, 1989) have gathered information on the prevalence of one or more coronary heart disease risk factors (several of these studies have been discussed in chapter 2). However, information on risk factors gathered from both of these types of research investigations remains sketchy.

This and the following chapter assess the prevalence of and association between various biological and behavioural risk factors of coronary heart disease among Bourke Aborigines aged 18 years and over. In particular, this chapter evaluates risk factors relating to body composition, blood pressure, serum cholesterol, alcohol and tobacco

1. I have been informed by Dr. Alan Gray that there are several cardiovascular risk factor studies being conducted, but the results from these have not yet been published.

consumption, and vigorous exercise, and the next chapter examines risk factors relating to diet and nutrition.

6.2 Prevalence of coronary heart disease among Bourke Aborigines

Of the Bourke Aborigines who participated in the survey, 5 per cent are suspected of having angina pectoris — a major clinical manifestation of coronary heart disease. This figure has been arrived at by analyzing the responses to a series of diagnostic questions on cardiovascular health (see section 5.4.1 in chapter 5). Males are almost twice as likely as females to be suspected of having angina — 6 versus 4 per cent. These respondents, both male and female, range in age from 18 to 44 years, with the disproportionate majority of them over the age of 29 years.

Compared to those respondents without angina, those with suspected angina tend: to be older; to be heavier; to have higher blood pressure and serum cholesterol levels; to be diabetic; to be current or ex-smokers; and to have reported being in the hospital in the year prior to the interview (see Table 6.1). Although these differences are not significant, they do suggest some notable disparities in the health and lifestyle of Aborigines with and without angina.

Bastian (1979: 287) found similar notable differences. He notes that tribal Aborigines with suspected coronary heart disease tended to be older, heavier, diabetic, and to have higher blood pressure and serum cholesterol levels than those without coronary heart disease. However, compared to Bourke Aborigines with suspected angina, tribal Aborigines with the same condition were on average significantly older (31 versus 53 years) and less likely to be current smokers. These differences seem to reflect the fact that Bastian's sample was older.

Table 6.1 Mean values of age and various biological risk factors, plus the prevalence of other criteria, for a non-random sample of Bourke Aborigines¹ aged 18 years and over with and without angina.

	No angina		Suspected angina	
	n ²	Mean ³	n ²	Mean ³
Age (years)	258	28 (1.4)	13	31 (4.2)
Biological risk factors				
Body mass index (kilogram / metre ²)	61	27 (1.4)	3	29 (6.6)
Systolic blood pressure (mm Hg)	62	129 (4.4)	3	133 (6.6)
Diastolic blood pressure (mm Hg)	62	87 (3.6)	3	90 (0.0)
Cholesterol (mmol / litre)	33	5.1 (0.4)	1	6.6 (0.0)
Other criteria				
	No angina		Suspected angina	
	n ²	Per cent ⁴	n ²	Per cent ⁴
Diabetes mellitus	253	4	13	30
Current smokers	250	48	12	58
Ex-smokers	250	14	12	25
At risk drinkers ⁵	244	44	13	38
Hospitalized in the year prior to interview	238	29	12	50

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Total number of respondents, excluding 'not stated'.

3. The numbers in brackets refer to two standard errors around the mean.

4. Proportion of respondents characterized by the following conditions or events.

5. See section 6.4.1 for a definition of drinkers at risk.

6.3 Biological risk factors

As discussed in chapter 1, biological risk factors are biological abnormalities detectable before the appearance of the first symptoms or clinical problems characteristic of the disease (see section 1.2.2.1). Possible precursors or risk factors of coronary heart disease include: genetic susceptibility, diabetes, obesity, hypertension, and elevated serum cholesterol. Only the last three factors were looked at in any detail in this study.

As mentioned in chapter 5, information on height, weight, blood pressure, and serum cholesterol levels was obtained by Aboriginal health workers from a non-random subsample of respondents who came into the Aboriginal Health Service after being interviewed (see section 5.4.1). This subsample was rather small — only 30 per cent of the total respondents had their height, weight, and blood pressure measured and only 10 per cent had their serum cholesterol measured. As a rule, the subjects in the subsample tend to be slightly older than those in the total sample (the survey population).

Estimates of the level of obesity among Bourke Aborigines were based on the height and weight measurements of 64 respondents — 36 males and 28 females. Height and weight measurements were related to body composition using Quetelet's body mass index (Bray, 1978; Bray, 1985). This index is a useful way of relating body weight to height and of assessing the magnitude of potential health risks, such as cardiovascular and digestive system diseases and cancer, associated with being overweight (Bray, 1985). It is calculated as specified below:

$$\text{Body mass index} = \frac{W}{H^2}$$

Where:

W = Weight in kilograms

H = Height in metres

As outlined by Bray (1978), body mass index can be used to describe the prevalence of four types of human body compositions — underweight, acceptable weight, overweight,

and obese. In this study, using standards established by the National Heart Foundation (1985: 31), these body compositions were defined as follows:

Body mass index (kilogram / metre ²)		
Body composition	Males	Females
Underweight	<20	<19
Acceptable weight	20-25	19-24
Overweight	26-30	25-30
Obese	>30	>30

Blood pressure readings were taken from 65 individuals in a sitting position using a sphygmomanometer. This instrument measures the diastolic (lowest pressure) and systolic (highest pressure) pressures of the blood against the walls of the brachial artery in the arm. Information on the serum cholesterol levels of 34 individuals was obtained by using the Reflotron dry-chemistry system that was made available to the Aboriginal Health Service by the New South Wales Department of Health for a three-day testing period in May of 1989.

Two types of comparisons were made using these clinical data. First, a comparison was made between the biological risk factor prevalence levels of Bourke Aborigines² and all Australians³. Although my study collected data on adults aged 18 years and over, for comparative purposes this analysis was limited to those aged 25 years and over.

Relative risk, the ratio of the Aboriginal rate to the all Australian rate, was used to make this comparison. To make the estimates of relative risk more reliable, confidence intervals, at a 95 per cent level of confidence, were constructed around each ratio

2. The term 'Bourke Aborigines' refers to those Aborigines who participated in the Aboriginal risk factor prevalence survey conducted in April and May of 1989.

3. The term 'all Australians' has been coined to refer to those adults aged 25 years and over living in capital cities throughout Australia who participated in the 1983 National Heart Foundation (1985) risk factor prevalence study and the companion 24-hour dietary recall study conducted by the Department of Health and Community Services (1987).

(Bailar and Ederer, 1964). To limit the effects of differences in population structure, Aboriginal rates were directly standardized to the all Australian sample population (National Heart Foundation, 1985: 19). To limit the effects of small numbers, sex-specific prevalence levels were combined and confidence intervals were only calculated for relative risk ratios based on a sample of five or more. The aim of this comparison was to assess whether Bourke Aboriginal biological risk factor prevalence levels are different from those exhibited by all Australians.

Second, comparisons were made between the biological risk factor prevalence levels of specific groups of Bourke Aborigines as defined by various risk markers — age, marital status, employment status, secondary education, and location. Differences in prevalence levels were evaluated by distinguishing between population means and by using the Brown-Mood non-parametric median test, which determined if a particular risk factor had the same distribution across different groups. This type of non-parametric test is quite robust because it makes no assumption about the underlying distribution of the sample. Sex-specific prevalence levels were combined to limit the effects of small numbers. However, unlike the first comparison, prevalence levels were not standardized. The goal of these comparisons was to better describe Bourke Aboriginal biological risk factor prevalence levels and to highlight, using a variety of risk markers, those Aborigines with a particularly high risk of coronary heart disease.

6.3.1 Comparison with National Heart Foundation survey

As shown in Table 6.2, there are several notable differences in Bourke Aboriginal and all Australian biological risk factor prevalence levels. To begin, Bourke Aborigines are more likely to be overweight or obese than all Australians. Most significantly, they are at least two times more likely to be obese (26 versus 8 per cent). Other researchers have noted similar high levels of obesity among Aborigines. Another recent study in Bourke noted that 36 per cent of Aborigines aged 20 years and over were obese (Cameron *et al.*, 1986). However, in this case obesity was defined as a body mass index greater than

27.5 for males and 27 for females. In Kempsey, New South Wales, Sibthorpe (1988: 283) noted that, from a non-random sample of Aboriginal subjects aged 15 years and over, 15 per cent of the male and 19 per cent of the female respondents were obese. In Wilcannia, New South Wales, a recent study observed that the prevalence of obesity among Aborigines was over two times higher than among non-Aborigines — 31 versus 14 per cent (Stephenson and Lenz, 1990). A study in South Australia noted that 26 per cent of all Aborigines surveyed (or 16 per cent of the males and 33 per cent of the females) from tribal, rural, and urban communities were obese (Aboriginal Health Organization, n.d.: 60-61). Obesity was evident throughout adulthood and was slightly more prevalent in rural than in tribal or urban communities. Thus the high level of obesity among Bourke Aborigines is by no means unique, but is indicative of a general Aboriginal pattern.

Compared with the all Australian sample, Bourke Aborigines are also at least three times more likely to have diastolic hypertension (≥ 95 mm Hg). Diastolic hypertension was observed in 39 per cent of the Aboriginal and 11 per cent of the all Australian subjects. In contrast, there was little difference in the prevalence of systolic hypertension (≥ 165 mm Hg) between these two populations.

Studies of Aboriginal blood pressure do not generally distinguish between diastolic and systolic hypertension. One study that did, however, stated that 17 per cent of Aborigines aged 21 years and over had diastolic hypertension (Simons *et al.*, 1981). Comparable levels have also been noted in studies that do not distinguish between the two types of hypertension. For example, the recent study by Stephenson and Lenz (1990) in Wilcannia categorized 26 per cent of Aborigines as being hypertensive; while the South Australian study noted that only 12 per cent of all those sampled were hypertensive (Aboriginal Health Organization, n.d.: 68). Overall, the results from both types of studies suggest that the level of hypertension among Bourke Aborigines may be overstated. This overestimate is most likely caused by the bias towards older ages in the subsample of respondents who had their blood pressure measured.

No substantial difference was observed in the proportion of Bourke Aborigines and all Australians who have high serum cholesterol, which is not surprising considering that other studies, as discussed in chapter 2 (see section 2.5.4), have found Aboriginal mean serum cholesterol levels to be generally lower than or about the same as non-Aboriginal levels (see Schwartz *et al.*, 1957; Charnock *et al.*, 1959; Simons *et al.*, 1981; Cameron *et al.*, 1986; Stephenson and Lenz, 1990). However, there are several problems with this comparison. First, Bourke Aboriginal cholesterol readings were taken from a rather small sample that was heavily weighted towards older ages (see section 5.4.3 in chapter 5). Second, all Australian cholesterol readings were taken from individuals who had fasted the night before being tested and from females who were not taking oral contraceptives; among the Aboriginal population, however, the cholesterol readings were taken from non-fasting adults (see section 5.4.1 in chapter 5). Third, the National Heart Foundation study based its readings on laboratory rather than on-site analysis of blood samples. Therefore, the similarity between Bourke Aboriginal and all Australian cholesterol levels should be viewed with some caution, as the effects of these biases are difficult to discern.

Table 6.2 Comparison of a non-random sample of Bourke Aborigines¹ and all Australians², by various biological risk factors, for persons aged 25 years and over.

	Bourke Aborigines n ³ per cent ⁴		All Australians n ³ per cent		Relative risk range ⁵
Body mass index⁶					
Overweight	20	47	2,404	32	(1.0 - 2.5)
Obese	11	26	616	8	(1.8 - 6.4)
Blood pressure⁷					
Diastolic (≥ 95 mm Hg)	16	39	837	11	(2.2 - 6.1)
Systolic (≥ 165 mm Hg)	3	7	589	8	-
Serum cholesterol⁸					
5.0-5.9 (mmol / litre)	11	39	2,316	35	(0.6 - 2.2)
≥6.0 (mmol / litre)	7	31	1,545	34	(0.4 - 2.2)

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Source: National Heart Foundation (1985).

3. Total number of respondents, excluding 'not stated'.

4. Aboriginal percentages have been directly standardized to the all Australian sample population.

5. A ratio of the observed Aboriginal rate to the all Australian rate. The relative risk range represents confidence intervals, at a 95 per cent level of confidence, around each ratio. A relative risk range was calculated for age-specific prevalence levels based on a sample of five or more.

6. Excluding those respondents who are underweight or acceptable weight.

7. Excluding those respondents who are not hypertensive.

8. Excluding those respondents who have serum cholesterol readings below 5.0 mmol / litre.

6.3.2 Comparison of Bourke Aboriginal biological risk factor prevalence levels

There are many notable differences in Bourke Aboriginal biological risk factor prevalence levels by age, marital status, employment status, secondary education, and location (see Tables 6.3 to 6.6). The most significant differences are by age and location.

The levels of all three biological risk factors examined here rise with age. Body mass index, as suggested by the Brown-Mood median test, significantly increases with age from a mean of 23.6 at ages 18 to 24 years to 28.5 at ages 35 years and over.

Furthermore, over these same age groups, the proportion who are overweight or obese more than doubles — from 29 per cent at ages 18 to 24 years to 74 per cent at ages 35 years and over. Mean diastolic and systolic blood pressure readings also increase with age, and with them so does the proportion who are hypertensive. From ages 18 to 24 years to 35 years of age and over, levels of systolic hypertension increase from 0 to 7 per cent, while levels of diastolic hypertension increase from 10 to 44 per cent. Lastly, mean total serum cholesterol levels rise from 4.5 mmol/litre at ages 18 to 24 years to 5.6 mmol/litre at ages 35 years and over.

Although sex-specific differences were not examined here because of the small sample size, the study conducted in Bourke by Cameron and colleagues (1986: 309-310) noted several sex-specific disparities in Aboriginal mean body mass index and total serum cholesterol and triglyceride readings. As a rule, females had greater mean body mass index values than males. Among males, there was little change in mean body mass index values with age; while among females, the older age groups had higher values than the younger age groups. Serum cholesterol and triglyceride readings for females also tended to rise with age. This increase was especially noticeable in the 55 to 64 years age group. However, even with these disparities, the sex-specific mean body mass index and total serum cholesterol readings for all ages noted by Cameron and colleagues (1986) were not significantly different from the sex-combined values observed in this study.

Variations in biological risk factor prevalence levels by marital status, employment status, secondary education, and location are mainly substantive. The most consistently apparent differences are by location. Bourke Aborigines living in town generally have higher mean body mass index, blood pressure, and serum cholesterol readings than those living on or near the mission. Furthermore, this group is more likely to be overweight, obese, hypertensive, and/or to have high serum cholesterol. Although these differences by location can possibly be explained by the stress involved with living in town, the true scenario is very difficult to assess.

Differences in biological risk factor prevalence levels by marital status and secondary education also exist, but they are less clear-cut. In fact, the pattern noted by these risk markers is probably mostly attributable to the effects of age. Generally, Bourke Aborigines with only a primary education are older than those with at least some secondary education; while those who have never had a partner are usually younger than those with a partner, or those who are divorced or widowed. Gender may also play a small role, especially in the case of marital status. Here the 'other' category (separated, divorced, or widowed) is based predominantly on a subsample of women who are over 50 years of age.

Table 6.3 Comparison of mean body mass index and the proportion who are overweight and obese for a non-random sample of Bourke Aborigines¹ aged 18 years and over, by age and various risk markers.

	n ²	Mean	Standard error ³	Proportion who are		Median test ⁴
				Overweight	Obese	
Age group						0.05
18-24	21	23.6	2.2	19	10	
25-34	16	28.9	3.2	43	25	
35 plus	27	28.5	2.0	48	26	
All ages	64	27.0	1.4	38	20	
Marital status						n.s.
Never had a partner	28	25.6	1.6	46	7	
Married / <i>de facto</i>	18	25.8	2.2	22	22	
Other	4	32.1	11.0	25	50	
Employment status						n.s.
Employed	16	26.7	2.0	44	12	
Unemployed	48	27.1	1.8	33	23	
Secondary education⁵						n.s.
Yes	42	26.9	1.8	33	21	
No	16	26.1	2.6	44	19	
Location⁶						n.s.
Mission	14	24.5	2.4	36	7	
Town	23	27.4	2.2	48	26	

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Total number of respondents, excluding 'not stated'.

3. Two standard errors around the mean.

4. Brown-Mood non-parametric median statistical test was used to determine if body mass index had the same distribution across different groups or categories.

5. Secondary education refers to people with at least some secondary education.

6. Mission refers to people living on or near the mission; while town refers to people living in town.

Table 6.4 Comparison of mean systolic blood pressure and the proportion with hypertension (≥ 160 mm Hg) for a non-random sample of Bourke Aborigines¹ aged 18 years and over, by age and various risk markers.

	n ²	Mean	Standard error ³	Proportion with hypertension	Median test ⁴
Age group					0.05
18-24	21	121.9	5.4	0	
25-34	17	127.1	8.0	0	
35 plus	27	135.6	6.8	7	
All ages	65	128.9	4.2	3	
Marital status					n.s.
Never had a partner	29	124.3	5.2		
Married / <i>de facto</i>	18	130.8	8.4	6	
Other	4	137.5	9.6	0	
Employment status					n.s.
Employed	16	128.8	8.0	0	
Unemployed	49	129.0	4.8	4	
Secondary education⁵					n.s.
Yes	43	127.4	4.8	2	
No	16	133.4	9.6	6	
Location⁶					n.s.
Mission	14	123.9	8.2	0	
Town	23	130.2	6.6	0	

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Total number of respondents, excluding 'not stated'.

3. Two standard errors around the mean.

4. Brown-Mood non-parametric median statistical test was used to determine if systolic blood pressure had the same distribution across different groups or categories.

5. Secondary education refers to people with at least some secondary education.

6. Mission refers to people living on or near the mission; while town refers to people living in town.

Table 6.5 Comparison of mean diastolic blood pressure and the proportion with hypertension (≥ 95 mm Hg) for a non-random sample of Bourke Aborigines¹ aged 18 years and over, by age and various risk markers.

	n ²	Mean	Standard error ³	Proportion with hypertension	Median test ⁴
Age group					n.s.
18-24	21	81.0	5.6	10	
25-34	17	85.0	5.2	24	
35 plus	27	94.1	5.6	44	
All ages	65	87.5	3.6	28	
Marital status					0.01
Never had a partner	29	84.8	4.2	14	
Married / <i>de facto</i>	18	90.8	8.8	44	
Other	4	97.5	12.6	75	
Employment status					n.s.
Employed	16	85.6	5.2	33	
Unemployed	49	88.1	4.4	33	
Secondary education⁵					n.s.
Yes	43	86.0	4.6	26	
No	16	89.1	6.4	25	
Location⁶					n.s.
Mission	14	82.1	8.4	21	
Town	23	90.7	4.6	26	

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Total number of respondents, excluding 'not stated'.

3. Two standard errors around the mean.

4. Brown-Mood non-parametric median statistical test was used to determine if diastolic blood pressure had the same distribution across different groups or categories.

5. Secondary education refers to people with at least some secondary education.

6. Mission refers to people living on or near the mission; while town refers to people living in town.

Table 6.6 Comparison of mean total serum cholesterol and the proportion with high serum cholesterol (≥ 6.0 mmol / litre) for a non-random sample of Bourke Aborigines¹ aged 18 years and over, by age and various risk markers.

	n ²	Mean	Standard error ³	Proportion with high cholesterol	Median test ⁴
Age group					n.s.
18-24	7	4.5	1.0	14	
25-34	10	4.9	0.4	0	
35 plus	17	5.6	0.4	41	
All ages	34	5.2	0.4	24	
Marital status					n.s.
Never had a partner	9	5.0	0.6	11	
Married / <i>de facto</i>	8	4.7	0.8	12	
Other	3	6.5	0.6	100	
Employment status					n.s.
Employed	11	5.0	0.6	18	
Unemployed	23	5.3	0.4	22	
Secondary education⁵					n.s.
Yes	21	5.1	0.4	19	
No	10	5.2	0.8	30	
Location⁶					n.s.
Mission	5	4.6	1.0	0	
Town	15	5.3	0.6	33	

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Total number of respondents, excluding 'not stated'.

3. Two standard errors around the mean.

4. Brown-Mood non-parametric median statistical test was used to determine if serum cholesterol had the same distribution across different groups or categories.

5. Secondary education refers to people with at least some secondary education.

6. Mission refers to people living on or near the mission; while town refers to people living in town.

6.4 Behavioural risk factors

As discussed in chapter 1, behavioural risk factors are characteristics which, as they appear before the onset of the disease, can be controlled by clinical, epidemiological, and other types of intervention (see section 1.2.2.2). Habits like heavy alcohol and tobacco consumption and physical inactivity are considered to be important behavioural risk factors of coronary heart disease. The effects of these risk factors, which depend on the intensity and duration of exposure, may influence both the initial appearance and the subsequent development of coronary heart disease (Péron and Strohmenger, 1985).

A series of questions were asked to elicit information on alcohol and tobacco consumption and physical activity (see section 5.4.1 in chapter 5). The three basic questions concerning alcohol consumption were: how many days per week did respondents drink alcohol; how much did they drink on a drinking day; and what type of beer, if any, did they drink. As already stated, no other questions were asked because of the possibility that more specific questions would be found offensive.

In regards to smoking behaviour, all respondents were asked whether they had ever regularly smoked cigarettes, cigars, or a pipe. Current and ex-smokers were then asked: the age at which they first started smoking; if they had ever quit smoking; and how much do they or did they smoke.

Exercise sessions in which respondents participated during the two-week period prior to the interview were categorized as being either: physical activities done for health fitness, recreation, or sport; or daily activities with some physical exertion.

Respondents who had participated in one or more exercise session were asked to indicate the total number of times they had participated in an activity and each activity's average duration. These exercise sessions were then classified as being either: vigorous exercise, if they involved heavy physical exertion; less vigorous exercise, if they involved moderate physical exertion; or daily activities, major daily tasks done around

home or at work, which involved at least a moderate to heavy amount of physical exertion.

As shown in Table 6.7, the most popular vigorous activities for males were weight lifting, cycling, jogging and running, and rugby; while for females jogging and running, aerobic exercise, and netball were the most popular. Also, less vigorous exercises, like fishing, bushwalking, and dancing, and daily activities, like walking, housecleaning, and gardening, were popular with both men and women.

Two types of comparisons were made with this information. First, a comparison was made between the behavioural risk factor prevalence levels of Bourke Aborigines, aged 25 years and over, and all Australians in the same age group. Once again, relative risk ranges were used to make this comparison. To provide a more reliable estimate of the differences between the two populations, confidence intervals at a 95 per cent level of confidence, were constructed around each relative risk ratio (Bailar and Ederer, 1964). However, to limit the effects of small numbers, these confidence intervals were only calculated for ratios based on a sample of five or more. As with biological risk factor prevalence levels, Aboriginal rates were directly standardized to the all Australian sample population (National Heart Foundation, 1985: 19). The purpose of this comparison was to determine whether Aboriginal behavioural risk factor prevalence levels are different from those exhibited by all Australians.

Second, comparisons were made between the behavioural risk factor prevalence levels of specific groups of Bourke Aborigines as defined by various risk markers — sex and age, marital status, employment status, secondary education, and location. In particular, differences in prevalence levels were measured using population means, a non-parametric median test, and odds ratios⁴. In this case, confidence intervals at a 95 per cent level of confidence, were calculated around each odds ratio using Woolf's (1955)

4. As defined in chapter 5, a ratio of the probability of one event to that of an alternative event.

method. As with the comparison of biological risk factors, these prevalence levels were not standardized. The general aim of these comparisons was to examine the associations between various behavioural risk factors and risk markers and to highlight particular high risk groups within the Bourke Aboriginal population.

As well as examining the patterning or frequency of alcohol consumption in this second type of comparison, an attempt was made to classify at risk behaviour. At present, although there is no internationally accepted way of defining a level of risk⁵ to a specific drinking pattern, researchers generally agree that with identical drinking patterns, women are at greater risk than men (see National Heart Foundation, 1985: 61). In fact, because of the risk associated with drinking during pregnancy, many researchers suggest that there is no safe level of drinking for women. In this study at risk drinking behaviour is defined in terms of the National Heart Foundation rating scale which classifies data on the frequency and intensity of drinking into no, low, intermediate, and high risk behaviour. However, because of the way the questions on alcohol consumption were worded, Aboriginal at risk behaviour could only be defined as no risk or at risk for men and low risk or high risk for women (see Tables A3.1 and A3.2 in Appendix 3).

As shown in Tables A3.3 and A3.4 in Appendix 3, 44 per cent of the male and 68 per cent of the female respondents were non-drinkers and no or low risk drinkers; while 56 per cent of the male and 32 per cent of the female respondents were at risk or high risk drinkers. Overall, this pattern of risk shows little variation across the ages.

5. In this case risk relates to harm not only from heart disease, but also from a wide variety of possible causes — such as cirrhosis of the liver, gallstone disease, and cancers of the mouth, throat, and oesophagus.

Table 6.7 Comparison of exercises and daily activities in which a non-random sample of Bourke Aborigines¹ aged 18 years and over participated over a two-week period, by sex.

Activity	Males (per cent) ²	Females (per cent) ²
Vigorous exercise		
Aerobic exercise	2	16
Basketball	3	4
Boxing	2	-
Cricket	3	-
Cycling or exercise bike	17	16
Hockey	1	12
Jogging and running	32	24
Netball	-	16
Rugby	28	-
Squash or tennis	2	8
Swimming ³	2	-
Touch football	2	-
Weight lifting	6	4
Total	100	100
Total individuals	74	12
Less vigorous exercise		
Bushwalking	12	25
Dancing	9	40
Fishing	55	35
Golfing	5	-
Hunting	17	-
Lawn bowling	2	-
Total	100	100
Total individuals	46	17
Daily activities		
Gardening	16	15
Heavy manual labour	16	2
Housecleaning	21	44
Light manual labour	8	-
Walking	39	39
Total	100	100
Total individuals	57	53

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Percentages may not add up because of rounding.

3. The pool was closed while the survey was being conducted.

6.4.1 Comparison with National Heart Foundation survey

Tables 6.8 and 6.9 show several notable differences in Bourke Aboriginal and all Australian behavioural risk factor prevalence levels. Generally there is little difference in the drinking habits of the two populations — that is, there are similar proportions of drinkers and non-drinkers and drinking days per week. However, Aborigines are significantly more likely to drink heavily on a drinking day. Aboriginal men are at least two and half times and Aboriginal women at least nine times more likely to drink five or more drinks on a drinking day than all Australians. This pattern of drinking is what Kamien (1975a: 295) refers to as 'the periodic binge'.

Compared to the National Heart Foundation survey results, a greater proportion of Bourke Aborigines are current smokers: Aboriginal prevalence levels are one and a half times higher for males (57 versus 35 per cent) and two times higher for females (48 versus 24 per cent). In fact, as noted by the relative risk ranges, Aboriginal men and women are significantly more likely to be current smokers. Additionally, fewer Aboriginal men are ex-smokers than their counterparts in the all Australian sample population — 15 versus 30 per cent.

Like other Australian current smokers, Bourke Aborigines smoke mainly manufactured cigarettes. Among males, there appears to be little difference in the proportion of Aborigines and all Australians who smoke 21 or more cigarettes per day — 37 and 41 per cent respectively. Among females, however, almost twice as many Bourke Aborigines smoke this amount — 39 versus 21 per cent.

In regards to tobacco consumption, a similar pattern has been noted in Wilcannia, New South Wales (Stephenson and Lenz, 1990). This study found that Aborigines are two to three times more likely to be current smokers — 71 and 76 per cent of male and female Aborigines compared with 39 and 42 per cent of male and female non-Aborigines. Also, a survey of Northern Territory Aboriginal communities observed that between 70 to 78 per cent of men and 43 to 64 per cent of women were current smokers (Watson *et*

al., 1988). These high prevalence levels for Aborigines in Wilcannia and the Northern Territory suggest that the results for Bourke Aborigines may be underestimated.

Finally, a smaller proportion of Aborigines had participated in vigorous exercise in the two-week period prior to being interviewed. This is especially so among Aboriginal women, where the proportion who had participated in vigorous exercise was only a third of that noted for the all Australian sample population (7 versus 26 per cent). The fact that Bourke Aboriginal participation rates decline more rapidly with age partially explains this difference (see section 6.4.2).

Table 6.8 Comparison of a non-random sample of male Bourke Aborigines¹ and all Australians², by various behavioural risk factors, for persons aged 25 years and over.

	Bourke Aborigines n ³ per cent ⁴⁻⁵		All Australians n ³ per cent ⁵		Relative risk range ⁶
Alcohol					
Drinking status					
Non-drinkers	8	15	446	12	(0.6 - 3.0)
Drinkers	69	85	3,294	88	(0.8 - 1.3)
Drinking days per week					
< 3 days	40	50	1,649	50	(0.7 - 1.4)
3 to 6 days	20	33	1,005	30	(0.7 - 1.8)
Every day	9	17	640	19	(0.5 - 1.9)
Drinks on a drinking day					
1 or 2 drinks	2	4	1,290	39	(0.0 - 0.8)
3 or 4 drinks	11	14	1,154	35	(0.2 - 0.8)
5 or more drinks	56	83	850	26	(2.5 - 4.2)
Smoking					
Smoking status					
Non-smokers	26	29	1,314	35	(0.6 - 1.2)
Current smokers	36	57	1,302	35	(1.2 - 2.3)
Ex-smokers	12	15	1,120	30	(0.3 - 1.0)
Cigarettes smoked per day ⁷					
1 to 20	20	63	655	59	(0.7 - 1.8)
21 or more	13	37	452	41	(0.5 - 1.7)
Exercise					
Vigorous exercise					
Yes	23	24	1,191	32	(0.5 - 1.1)
No	53	76	2,539	68	(0.9 - 1.5)

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Source: National Heart Foundation (1985).

3. Total number of respondents, excluding 'not stated'.

4. Aboriginal percentages have been directly standardized to the all Australian sample population.

5. Percentages may not add up because of rounding.

6. A ratio of the observed Aboriginal rate to the all Australian rate. The relative risk range represents confidence intervals, at a 95 per cent level of confidence, around each ratio. A relative risk range was calculated for age-specific prevalence levels based on a sample of five or more.

7. By current smokers of manufactured cigarettes.

Table 6.9 Comparison of a non-random sample of female Bourke Aborigines¹ and all Australians², by various behavioural risk factors, for persons aged 25 years and over.

	Bourke Aborigines n ³ per cent ⁴⁻⁵		All Australians n ³ per cent ⁵		Relative risk range ⁶
Alcohol					
Drinking status					
Non-drinkers	20	39	952	25	(1.0 - 2.6)
Drinkers	38	61	2,922	75	(0.6 - 1.1)
Drinking days per week					
< 3 days	31	82	2,056	70	(0.8 - 1.8)
3 to 6 days	6	17	525	18	(0.4 - 2.5)
Every day	1	1	341	12	-
Drinks on a drinking day					
1 or 2 drinks	1	4	2,083	71	
3 or 4 drinks	5	16	668	23	(0.3 - 2.2)
5 or more drinks	30	80	170	6	(9.6 - 20.3)
Smoking					
Smoking status					
Non-smokers	18	34	2,283	59	(0.4 - 1.0)
Current smokers	27	48	948	24	(1.3 - 2.9)
Ex-smokers	10	19	641	17	(0.6 - 2.3)
Cigarettes smoked per day ⁷					
1 to 20	14	61	737	79	(0.5 - 1.6)
21 or more	11	39	200	21	(1.0 - 3.6)
Exercise					
Vigorous exercise					
Yes	6	7	989	26	(0.1 - 0.8)
No	51	93	2,883	74	(0.9 - 1.7)

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Source: National Heart Foundation (1985).

3. Total number of respondents, excluding 'not stated'.

4. Aboriginal percentages have been directly standardized to the all Australian sample population.

5. Percentages may not add up because of rounding.

6. A ratio of the observed Aboriginal rate to the all Australian rate. The relative risk range represents confidence intervals, at a 95 per cent level of confidence, around each ratio. A relative risk range was calculated for age-specific prevalence levels based on a sample of five or more.

7. By current smokers of manufactured cigarettes.

6.4.2 Comparison of Bourke Aboriginal behavioural risk factor prevalence levels

As shown in Tables 6.10 to 6.15, Bourke Aboriginal behavioural risk factor prevalence levels vary considerably by age, marital status, employment status, secondary education, and location. However, the associations or relationships are less definitive than those between the biological risk factors.

Age is still an important variable. For both sexes, the proportion of at risk drinkers decreases with age, while the proportion of non-drinkers increases (see Table 6.10). This is especially so among females where the proportion of non-drinkers rises from 24 per cent at ages 18 to 24 years to 47 per cent at ages 35 years and over. Additionally, among either sex, the odds of being an at risk drinker slightly decrease with age.

Age-specific differences are less clear-cut when examining: the proportion who are current smokers; the mean age at which smokers started smoking; and the mean number of cigarettes current smokers smoke (see Tables 6.11 to 6.13). The proportion of male current smokers increases with age, while the proportion of non-smokers decreases. Among females, the proportion who are non- and current smokers is constant with age. In contrast, the mean age at which current and ex-smokers started smoking has remained stable with age for males and has dramatically decreased with age for females. The mean age at which women started smoking has declined five years in a generation — from 19 years of age for those aged 35 years and over to 15 years of age for those aged 18 to 24 years. Furthermore, the mean number of cigarettes women smoke tends to increase with age.

Results from this study suggest that the proportion of Bourke Aborigines who are current smokers and the intensity of their smoking habits have decreased somewhat from what Kamien (1976) observed in the early 1970s. At that time Kamien (1976: 40) noted that women began smoking at a mean age of 16 years and men at a mean age of 13 years. This study found a similar figure for all women aged 18 years and over but a slightly higher figure for men (mean ages of 16 and 15 years respectively — see Table

6.12). Kamien also observed that only 13 per cent of the Aboriginal men in Bourke and 29 per cent of the Aboriginal women were non-smokers, which compares with 39 per cent of the men and 33 per cent of the women in this study, and that a large proportion of smokers smoked more than 15 cigarettes per day — 60 per cent of the men and 36 per cent of the women.

Nearly half of the males (49 per cent) and over one-tenth of the females (13 per cent) who were interviewed had engaged in at least one vigorous exercise activity of 20 minutes or more during the two-week period prior to being interviewed; almost a third of these men (32 per cent) and two-thirds of these women (64 per cent) had taken part in three or more vigorous exercise activities per week. However, for both sexes these levels of vigorous exercise decline sharply with age (see Tables 6.14 and 6.15). The proportion of males who had participated in vigorous exercise drops from 66 per cent for those aged 18 to 24 years to 17 per cent for those aged 35 years and over; while among females it decreases from 16 per cent to 3 per cent between the same age groups. Also, the mean number of vigorous exercise activities completed over a two-week period by males drops from 7 to 3 between the age groups 18 to 24 years and 35 years and over; while for females it declines from 10 to 0.

Apart from age, the other most notable variable is employment status, especially with the proportion of Bourke Aborigines who currently smoke and who participate in vigorous exercise activities (see Tables 6.11, 6.13 and 6.14). The odds of being a smoker are significantly less for those employed than for those unemployed — 0.3 versus 3.3. Current smokers who are employed also tend to smoke fewer cigarettes. Additionally, the odds of participating in vigorous exercise are significantly higher for those employed than for those unemployed — 2.9 versus 0.3.

Differences in behavioural risk factor prevalence levels by marital status and secondary education are again most likely attributable to the effects of age. However, location is an important variable when looking at the proportion of current smokers and the mean

number of cigarettes smoked (see Tables 6.11 and 6.13). As a rule, respondents living on or near the mission are more likely to smoke and to smoke more heavily than those living in town.

Table 6.10 The odds of being an at risk drinker¹ for a non-random sample of Bourke Aborigines² aged 18 years and over, by sex, age, and various risk markers.

		Proportion who are			
	n ³	Non- drinkers	At risk drinkers	Odds ratio ⁴	Confidence interval ⁵
Males					
18-24	77	12	55	1.2	(0.6 - 2.3)
25-34	42	5	50	0.9	(0.4 - 1.8)
35 plus	26	23	50	0.9	(0.4 - 2.1)
All ages	145	11	56	-	-
Females					
18-24	55	24	66	1.5	(0.7 - 3.4)
25-34	25	24	66	1.6	(0.6 - 4.0)
35 plus	30	47	53	0.3	(0.1 - 1.0)
All ages	110	30	70	-	-
Both sexes					
Marital status					
Never had a partner	137	12	65	1.5	(0.9 - 2.5)
Married / <i>de facto</i>	95	22	57	0.8	(0.4 - 1.3)
Other	10	60	40	0.4	(0.1 - 1.5)
Employment status					
Employed	50	8	60	1.0	(0.5 - 1.9)
Unemployed	206	22	60	1.0	(0.5 - 1.9)
Secondary education⁶					
Yes	183	15	64	0.4	(0.2 - 1.0)
No	45	33	58	2.3	(1.0 - 5.0)
Location⁷					
Mission	69	19	65	1.4	(0.7 - 2.7)
Town	65	22	62	0.7	(0.4 - 1.5)

1. See section 6.4 for a definition of drinkers at risk.

2. Aboriginal risk factor prevalence survey conducted in April and May 1989.

3. Total number of respondents, excluding 'not stated'.

4. The odds of being an at risk drinker.

5. Confidence intervals, at a 95 per cent level of confidence, were calculated using Woolf's (1955) method.

6. Secondary education refers to people with at least some secondary education.

7. Mission refers to people living on or near the mission; while town refers to people living in town.

Table 6.11 The odds of being a current smoker for a non-random sample of Bourke Aborigines¹ aged 18 years and over, by sex, age, and various risk markers.

		Proportion who are			
	n ²	Non-smokers	Current smokers	Odds ratio ³	Confidence intervals ⁴
Males					
18-24	78	42	47	1.0	(0.5 - 1.8)
25-34	44	43	39	0.6	(0.3 - 1.2)
35 plus	30	23	63	2.2	(1.0 - 5.0)
All ages	152	39	48	-	-
Females					
18-24	53	34	53	1.2	(0.5 - 2.5)
25-34	24	29	54	1.2	(0.5 - 2.9)
35 plus	31	36	45	0.7	(0.3 - 1.7)
All ages	108	33	51	-	-
Both sexes					
Marital status					
Never had a partner	142	40	43	0.9	(0.6 - 1.5)
Married / <i>de facto</i>	96	32	52	1.2	(0.7 - 2.1)
Other	10	30	30	0.4	(0.1 - 0.7)
Employment status					
Employed	49	55	27	0.3	(0.2 - 0.6)
Unemployed	212	32	54	3.3	(1.6 - 6.5)
Secondary education⁵					
Yes	184	39	46	0.5	(0.3 - 1.0)
No	50	20	62	1.9	(1.0 - 3.7)
Location⁶					
Mission	68	35	57	2.0	(1.0 - 3.7)
Town	64	38	41	0.5	(0.2 - 1.0)

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Total number of respondents, excluding 'not stated'.

3. The odds of being a current smoker.

4. Confidence intervals, at a 95 per cent level of confidence, were calculated using Woolf's (1955) method.

5. Secondary education refers to people with at least some secondary education.

6. Mission refers to people living on or near the mission; while town refers to people living in town.

Table 6.12 Comparison of the mean age at which current and ex-smokers started smoking for a non-random sample of Bourke Aborigines¹ aged 18 years and over, by sex, age, and various risk markers.

	n ²	Mean	Standard error ³	Per cent smoking by		Median test ⁴
				age 12	age 16	
Males						n.s.
18-24	45	15.0	0.6	7	87	
25-34	27	15.7	0.8	4	74	
35 plus	23	14.8	1.4	17	83	
All ages	95	15.1	0.6	8	82	
Females						0.01
18-24	36	14.6	0.6	17	94	
25-34	15	15.0	1.6	13	73	
35 plus	16	19.4	2.4	0	19	
All ages	67	15.9	0.8	12	72	
Both sexes						
Marital status						0.05
Never had a partner	86	14.7	0.4	5	86	
Married / <i>de facto</i>	68	15.8	0.8	7	69	
Other	6	21.7	5.6	0	17	
Employment status						n.s.
Employed	21	15.9	0.8	0	71	
Unemployed	141	15.4	0.6	10	77	
Secondary education⁵						n.s.
Yes	113	15.5	0.6	7	82	
No	35	14.7	1.0	17	74	
Location⁶						n.s.
Mission	45	15.2	0.6	5	80	
Town	39	15.5	1.0	10	89	

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Total number of respondents, excluding 'not stated'.

3. Two standard errors around the mean.

4. Brown-Mood non-parametric median statistical test was used to determine if the age of starting smoking had the same distribution across different groups or categories.

5. Secondary education refers to people with at least some secondary education.

6. Mission refers to people living on or near the mission; while town refers to people living in town.

Table 6.13 Comparison of the mean number of cigarettes smoked by current smokers for a non-random sample of Bourke Aborigines¹ aged 18 years and over, by sex, age, and various risk markers.

	n ²	Mean	Standard error ³	Proportion smoking 21 or more cigarettes	Median test ⁴
Males					n.s.
18-24	36	17.7	4.8	31	
25-34	14	19.6	4.8	50	
35 plus	19	15.0	5.0	32	
All ages	69	17.3	3.0	35	
Females					0.05
18-24	27	14.0	3.8	11	
25-34	13	20.8	5.4	54	
35 plus	12	20.1	6.4	33	
All ages	52	17.1	2.8	27	
Both sexes					
Marital status					n.s.
Never had a partner	65	16.2	3.0	26	
Married / <i>de facto</i>	47	18.8	3.2	40	
Other	2	12.0	16.0	0	
Employment status					n.s.
Employed	12	14.4	4.0	17	
Unemployed	109	17.6	2.2	33	
Secondary education⁵					n.s.
Yes	81	17.4	2.8	25	
No	28	16.2	3.6	8	
Location⁶					n.s.
Mission	38	14.6	3.2	21	
Town	26	19.1	7.0	27	

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Total number of respondents, excluding 'not stated'.

3. Two standard errors around the mean.

4. Brown-Mood non-parametric median statistical test was used to determine if the number of cigarettes smoked had the same distribution across different groups or categories.

5. Secondary education refers to people with at least some secondary education.

6. Mission refers to people living on or near the mission; while town refers to people living in town.

Table 6.14 The odds of participating in vigorous exercise for a non-random sample of Bourke Aborigines¹ aged 18 years and over, by sex, age, and various risk markers.

	n ²	Proportion who participated in vigorous exercise	Odds ratio ³	Confidence interval ⁴
Males				
18-24	80	66	4.5	(2.3 - 8.9)
25-34	46	39	0.6	(0.3 - 1.2)
35 plus	30	17	0.2	(0.1 - 0.4)
All ages	156	49	-	-
Females				
18-24	55	16	1.7	(0.5 - 5.0)
25-34	27	18	1.7	(0.5 - 5.5)
35 plus	30	3	0.2	(0.0 - 1.3)
All ages	112	13	-	-
Marital status				
Never had a partner	146	40	1.8	(1.0 - 3.0)
Married / <i>de facto</i>	101	30	0.7	(0.4 - 1.2)
Other	10	0	-	-
Employment status				
Employed	51	55	2.9	(1.6 - 5.5)
Unemployed	218	29	0.3	(0.2 - 0.6)
Secondary education⁵				
Yes	194	39	3.7	(1.6 - 8.6)
No	48	15	0.3	(0.1 - 0.6)
Location⁶				
Mission	70	33	0.8	(0.4 - 1.6)
Town	64	39	1.3	(0.6 - 2.7)

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Total number of respondents, excluding 'not stated'.

3. The odds of participating in vigorous exercise.

4. Confidence intervals, at a 95 per cent level of confidence, were calculated, using Woolf's (1955) method.

5. Secondary education refers to people with at least some secondary education.

6. Mission refers to people living on or near the mission; while town refers to people living in town.

Table 6.15 Comparison of the mean number of vigorous activities over a two-week period for a non-random sample of Bourke Aborigines¹ aged 18 years and over, by sex, age, and various risk markers.

	n ²	Mean	Standard error ³	Per cent exercising ≥ 3 times per week	Median test ⁴
Males					n.s.
18-24	53	6.8	2.2	34	
25-34	16	5.2	2.4	38	
35 plus	5	2.6	1.2	0	
All ages	74	6.1	1.6	32	
Females					n.s.
18-24	9	10.1	6.4	56	
25-34	5	6.4	2.6	80	
35 plus	0	-	-	0	
All ages	14	8.8	4.2	64	
Both sexes					
Marital status					n.s.
Never had a partner	58	6.5	2.0	38	
Married / <i>de facto</i>	30	6.1	2.4	40	
Other	0	-	-	0	
Employment status					n.s.
Employed	26	7.4	3.2	42	
Unemployed	63	6.2	1.8	37	
Secondary education⁵					n.s.
Yes	72	7.2	1.8	40	
No	7	5.0	2.6	43	
Location⁶					n.s.
Mission	23	9.6	4.2	39	
Town	24	8.3	3.4	46	

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Total number of respondents, excluding 'not stated'.

3. Two standard errors around the mean.

4. Brown-Mood non-parametric median statistical test was used to determine if the number of vigorous activities had the same distribution across different groups or categories.

5. Secondary education refers to people with at least some secondary education.

6. Mission refers to people living on or near the mission; while town refers to people living in town.

6.5 Multiple risk factors

The analysis employed thus far has examined single biological and behavioural risk factors independently of each other. This section considers the prevalence of and association between multiple risk factors. This type of analysis is important because it examines the simultaneous occurrence and interaction of one or more risk factor within an individual, which provides a more realistic picture.

As above, two types of comparisons were made. First, the multiple prevalence of three coronary heart disease risk factors among Bourke Aborigines was compared with that found among all Australians. In particular, the prevalence of high blood pressure (diastolic pressure ≥ 95 Hg mm), high serum cholesterol (≥ 6.5 mmol/litre), and cigarette smoking (smoking one or more manufactured and/or hand-rolled cigarettes per day) was considered. Because the sample of Bourke Aborigines was so small, sex-combined totals of Aborigines aged 18 years and over were compared to similar totals of all Australians aged 25 years and over. Second, the associations between various risk factors found among Bourke Aborigines were investigated using odds ratios. Woolf's (1955) method was again used to calculate confidence intervals at a 95 per cent level of confidence, around each odds ratio. In particular, this comparison examines the odds of being obese, of being a drinker of alcohol, or of being a current smoker by other biological and behavioural risk factors.

Twice as many Bourke Aborigines as all Australians have two risk factors — 18 versus 9 per cent (see Table 6.16). Fewer Aborigines have one risk factor, while a similar proportion in both populations have no risk factors. Because of the small Aboriginal sample size, no major conclusions can be drawn from this comparison. However, what is suggested is that the high prevalence of coronary heart disease could be caused by a compounding effect of more than one risk factor. This issue needs to be considered in future studies.

Several notable associations have been found between obesity, drinking alcohol, or current smoking and other biological or behavioural risk factors (see Table 6.17). First, Bourke Aborigines who are obese are significantly more likely to have diastolic hypertension (≥ 95 mm Hg) and high serum cholesterol levels (≥ 6.5 mmol/litre) and significantly less likely to be current smokers or drinkers of alcohol. The first two relationships are not surprising considering that obesity has repeatedly been shown to predispose individuals to the development of hypertension and hyperlipidaemia (see section 1.2.2.1 in chapter 1). Second, those who consume alcohol are significantly more likely to be current smokers and to participate in vigorous exercise, while those who are current smokers are significantly less likely to participate in vigorous exercise. The relationship between alcohol consumption and vigorous exercise is not unexpected considering that most vigorous exercise is done by young men (18 to 34 years of age) of whom only a small minority are non-drinkers (see Tables 6.10 and 6.14). Third, the lack of association between current smoking and hypertension is also noteworthy considering that smoking has been shown to be not positively related to hypertension (see section 1.2.2.2 in chapter 1). Again, these comparisons suggest that the risk factors of coronary heart disease often occur together and therefore tend to reinforce one another or act synergistically.

Table 6.16 Comparison of a non-random sample of Bourke Aborigines¹, aged 18 years and over, and all Australians², aged 25 years and over, by various multiple risk factors³.

	Bourke Aborigines		All Australians	
	n ⁴	Per cent	n ⁴	Per cent
No risk factors	19	56	3,736	53
One risk factor	9	26	2,649	37
Two risk factors	6	18	668	9
Three risk factors	0	0	60	1
Not stated	0	0	7	0
Total	34	100	7,120	100

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Source: National Heart Foundation (1985).

3. Diastolic blood pressure ≥ 95 mm Hg, serum cholesterol ≥ 6.5 mmol / litre, current smoker (smoking one or more manufactured and/or hand-rolled cigarettes per day).

4. Total number of respondents, excluding 'not stated'.

Table 6.17 The odds of relating obesity, alcohol consumption, or cigarette smoking to various risk factors for a non-random sample of Bourke Aborigines¹ aged 18 years and over.

Risk factor	n ²	Joint cases	Odds ratio ³	Confidence interval ⁴
Obesity				
Diastolic hypertension ⁵	64	7	4.8	(1.3 - 17.4)
High serum cholesterol ⁶	34	4	25.0	(2.2 - 284.6)
Alcohol drinker	60	9	0.2	(0.1 - 0.6)
Current smoker	62	6	0.3	(0.1 - 0.8)
Vigorous exercise	61	2	0.4	(0.1 - 2.1)
Alcohol drinker				
Diastolic hypertension ⁵	65	11	0.5	(0.2 - 1.7)
High serum cholesterol ⁶	34	3	1.1	(0.2 - 7.3)
Current smoker	246	126	2.7	(1.4 - 5.2)
Vigorous exercise	261	82	2.1	(1.0 - 4.2)
Current smoker				
Diastolic hypertension ⁵	63	8	0.8	(0.3 - 2.5)
High serum cholesterol ⁶	32	1	0.6	(0.1 - 6.2)
Vigorous exercise	259	33	0.5	(0.3 - 0.8)

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Total number of respondents, excluding 'not stated'.

3. The odds of relating obesity, alcohol consumption, or cigarette smoking to various risk factors of coronary heart disease.

4. Confidence intervals, at a 95 per cent level of confidence, were calculated using Woolf's (1955) method.

5. Diastolic blood pressure ≥ 95 mm Hg.

6. Serum cholesterol reading ≥ 6.5 mmol / litre.

6.6 Summary

The purpose of this chapter has been to estimate the prevalence of and highlight the association between various biological and behavioural risk factors of coronary heart disease among Bourke Aborigines. Overall, four major points have been raised. First, substantive differences were noted between the health and lifestyle of Bourke Aborigines who are and are not suspected of having angina. In particular, respondents with suspected angina are more likely to be older, to be heavier, to be diabetic, to be current smokers, to have high blood pressure and high serum cholesterol readings, and to have reported being in the hospital in the year prior to the interview. As a rule, these disparities are similar to those noted by Bastian (1979) for tribal Aborigines with and without suspected coronary heart disease.

Second, Bourke Aboriginal biological and behavioural risk factor prevalence levels differ from all Australian levels in several ways. In terms of biological risk factors, Bourke Aborigines are more likely to be overweight or obese and to have diastolic hypertension. In terms of behavioural risk factors, Bourke Aborigines are more likely to drink heavily on a drinking day and to be current smokers, and are less likely to participate in vigorous exercise.

Third, Bourke Aboriginal risk factor prevalence levels can vary considerably by age, location, and employment status. Both biological and behavioural risk factor prevalence levels tend to vary with age. Interestingly, Aborigines living in town generally have higher mean body mass index, blood pressure, and serum cholesterol readings than those living on or near the mission; and employed respondents were less likely to smoke and more likely to participate in vigorous exercise than unemployed respondents.

Fourth, there are several notable associations found between risk factors. The most interesting of these interrelationships suggests that Bourke Aborigines who are obese

are significantly more likely to have diastolic hypertension (≥ 95 mm Hg) and high serum cholesterol levels (≥ 6.5 mmol/litre).

CHAPTER SEVEN

DIET AND NUTRITION

7.1 Introduction and overview

Although a large number of studies concerning Aboriginal nutrition have been conducted in western New South Wales over the last 20 years, few of them have paid much attention to adult nutrition. Among these studies, Edwards (1970), Edwards and Craddock (1973), and Heywood and Zed (1977) look at the nutritional status of pre-school and school age children in Walgett; Brand and colleagues look at lactose malabsorption in infants and its relation to human growth and nutrition (Mitchell *et al.*, 1977; Brand, 1978); and Kamien and associates look at infant and childhood nutrition in Bourke (Kamien *et al.*, 1974; Nobile, 1974; Kamien *et al.*, 1975a). Only Kamien and associates have additionally dealt in any great detail with aspects of adult nutrition (see Kamien *et al.*, 1974, 1975a, 1975b).

This remarkable shortage of quantitative information on the nutritional status of adult Aborigines is by no means limited to western New South Wales. In addition to the works by Kamien and associates on Bourke Aborigines, the handful of studies that have been conducted throughout Australia examine the nutritional status of adult Aborigines living in: Port Augusta, South Australia (Wise *et al.*, 1970); a country town in southwestern Australia (Hitchcock and Gracey, 1975); four Anangu Pitjantjatjara communities in South Australia (South Australia, 1987); an isolated traditional-oriented community in the Northern Territory (O'Dea *et al.*, 1988); and Kempsey, New South Wales (Sibthorpe, 1988). Overall, there is a need for more detailed analyses of the nutritional status of adult Aborigines, especially in concert with a study of their current health status.

On a small scale, this study has attempted to do just that. Here a 24-hour dietary recall survey method was used to obtain information on the dietary habits of Bourke Aborigines aged 18 years and over. The mean daily intakes of selected macro- and micro-nutrients were calculated from this recall information. The overall aim of this chapter is to assess whether the dietary habits of Bourke Aborigines can be considered to be placing them at risk of developing and dying from coronary heart disease.

7.2 Dietary measurement method

The 24-hour dietary recall method was chosen for this study because it required little formal training of the interviewers and little time or commitment from the respondents, which meant that they were more likely to answer the dietary recall questions. There are, however, two potential sources of error with this type of method. First, the ability to remember what was eaten varies from individual to individual. The error stems from the fact that respondents are more likely to forget an item or items that were consumed, rather than from items that were not actually consumed being reported. Although the size of the error is largely dependent on what is forgotten, this type of bias has been shown to affect the reporting of daily nutrient intake in the range of 10 to 35 per cent (Bingham, 1987). Studies which have compared the abilities of different groups to remember what they have eaten have demonstrated that women are generally better at this task than men, and younger adults better than older ones (Rutishauser, 1988: 106).

Respondents who participated in this study were most likely to underreport their intakes of alcohol, sugar, milk, and fat. The consumption of either beer or wine was recorded on only 4 out of 216 recalls (2 per cent). This type of underreporting was difficult to circumvent, as respondents were quite sensitive about discussing what and how much they drank. Similar types of problems with the reporting of alcohol intake have been found in other Aboriginal dietary surveys (see Wise *et al.*, 1970; Kamien *et al.*, 1975a; Sibthorpe, 1988). Additionally, respondents tended to not recall the amount of sugar and milk they put in their tea or coffee and/or to not recall what they spread on their

bread or toast — in the form of butter, margarine, honey, jam, peanut butter, syrups, or other types of spreads. To compensate for this underreporting of sugar, milk, and fat, I made assumptions, based on observations of local dietary habits, about the amount of sugar and milk respondents put in their tea or coffee and the amount of spreads they put on their bread or toast¹.

The second potential source of error with the dietary recall method is that the ability to estimate portion sizes also varies from individual to individual. Reports in the literature indicate that errors incurred by the estimation of individual food portion weights are regularly in the 20 to 50 per cent range. However, this level of error can be reduced with the use of models, life-size photographs, and/or standard household measures (Rutishauser, 1988: 105).

Recalls that were collected in this study varied greatly in terms of how particular food items were described. In an attempt to counteract this problem, ingredient lists of recipes and estimates of portion sizes were standardized². Ingredient lists, especially for recipes of curries, salads, sandwiches, and stews, were determined by: observing local dietary habits; asking informants how they made particular items; or making a note of recipes described in published sources. The portion size of a particular ingredient or food item was estimated by: observing local dietary habits; determining the average size or weight of store-bought items; or using estimates of portion sizes taken from tables of the composition of Australian foods (Department of Community Services and Health, 1989, 1990a, 1990b) or from other Aboriginal dietary studies.

1. See Table A4.2 in Appendix 4 for the recipes of tea, coffee, and toast used in this study.

2. A complete listing of the recipes and food items that were standardized can be found in Appendix 4.

7.2.1 Collection of dietary data

The dietary habits of a non-random sample of 125 male and 91 female Bourke Aborigines were assessed by examining the food items they consumed at different meals and snacks over various one-day periods during April and May of 1989 (see individual data sheets 14 to 25 in Appendix 2). As discussed in chapter 5, respondents were asked to recall what they ate and drank the day prior to the interview starting with the morning meal or snack (see section 5.4.1). Dietary recalls were quite evenly distributed over all the days of the week and collected information on 81 per cent of all the respondents who participated in the survey.

The local Aboriginal interviewers who collected the dietary data were given instructions on how to prompt respondents to recall what food items they ate and the portion sizes and on how to record these items on the data sheets provided in the questionnaire. The interviewers were also given a set of household measures³ to help respondents estimate portion sizes. However, most interviewers found these visual prompts to be inappropriate and they were not used. As noted above, this caused discrepancies in the way portion sizes were estimated.

7.2.2 Coding of dietary data

Approximately 2,500 food items from the 216 recall records were coded using guidelines set out by the Nutrition Section of the Department of Community Services and Health (see Department of Health, 1986). The macro- and micro-nutrient content of each food item was calculated using a computer program called DIET / 1 (Xyris Software, 1989a), which contains a data disk version of the tables of the composition of Australian foods — the 1989 NUTTAB data base (Xyris Software, 1989b). Along with nutrient-composition information, the data record for each food item contained information on: the food item's volume or weight; the respondent's sex, age,

3. A set of four metric measuring cups (1 cup, 1/2 cup, 1/3 cup, and 1/4 cup) and measuring spoons (1 tablespoon, 1 teaspoon, 1/2 teaspoon, and 1/3 teaspoon).

identification number, and place of residence; and whether the recall took place during a pay or off pay week. The subsequent analysis of these data records, for food items and nutrients consumed, was done using SAS, a mainframe statistical computer package.

DIET / 1 readily allows the ingredient lists of recipes and the portion sizes of food items to be standardized. For many of the foods in the NUTTAB data base, common measures have already been predefined. However, in this study these were often replaced by reference measures more appropriate for the survey population.

Additionally, the RECIPE program was used to standardize recipes and portion sizes of particular food items not found on the NUTTAB data base.

The major problem with the NUTTAB data base and the tables of the composition of Australian foods is that they do not contain any information on the nutrient content of Australian bush foods. Although a large number of other relevant tables of the composition of Australian bush foods exist (see Brand *et al.*, 1983; James, 1983; Brand and Cherikoff, 1985; Brand *et al.*, 1985; Cherikoff *et al.*, 1985), none describe the nutrient composition of emu meat or the local fish species that were eaten by Bourke Aborigines during the survey. Therefore, local species of fish were assumed to have the equivalent nutrient composition of more common varieties. However, no appropriate alternative was found for emu meat, so the four recalls containing that food item were not coded or analyzed.

7.3 Dietary habits

This section examines the foods items that were consumed by respondents at different meals or snacks during the whole recall period. The aim here is to examine the dietary habits of Bourke Aborigines⁴ in terms of the contribution of various food items to their

4. As outlined in chapter 6, the term 'Bourke Aborigines' refers to those Aborigines who participated in the Aboriginal risk factor prevalence survey conducted in April and May of 1989.

overall energy, protein, carbohydrate, and fat intakes. Because of the underreporting of alcohol intake and possibly of other nutrients, a broad-based comparison using all Australian data sources has not been made.

According to guidelines set out by the Department of Health and Community Services (Department of Health, 1986), food items consumed were grouped into the following 15 food groups: cereal and cereal products; vegetables; fruits; meat and meat products; fish, seafood, and their products; eggs; nuts and seeds; milk and milk products; fats; snack foods; sugar, jam, honey, and syrups; confectionery; beverages, non-alcoholic; beverages, alcoholic; and condiments, flavourings, and soups. Within each food group, food items were categorized into subgroups of similar types — for example, curries and stews were included within the mixed dishes subgroup. Overall, food items were classified into groups according to their similarities, their type and use, and their quantity and frequency of consumption (see Table A4.1 in Appendix 4).

7.3.1 Food items consumed

Bourke Aborigines derive much of their energy intake from meat and meat products, especially from lamb and take-aways (see Table 7.1). Lamb, which accounted for 18 per cent of male and 10 per cent of female total energy intake, was mainly eaten in the form of chops; while take-aways, which accounted for 9 per cent of male and 5 per cent of female total energy intake, were consumed as chicken, chiko rolls, hamburgers, meat pies, pizza, and sausage rolls.

Another important source of energy is cereal and cereal products. This food group's most notable contribution was made by bread, which accounted for 17 per cent of male and 22 per cent of female total energy intake. A much smaller contribution, about 3 to 4 per cent of total energy intake, was made by breakfast cereals and other cereal items, like cakes, biscuits, pasta, rice, and desserts containing cereal.

Additionally, vegetables, fats (all non-cooking uses), milk and milk products, and non-alcoholic beverages make small but notable contributions to energy intake. Although a large variety of vegetables were consumed, including beans, beetroot, broccoli, cabbage, capsicum (green and red peppers), carrots, cauliflower, corn, cucumber, leafy greens, onions, peas, and pumpkin, potatoes were the most important vegetable source of energy — they accounted for 6 per cent of both male and female total energy intake. Similar proportions of energy were also derived from milk, margarine, and non-alcoholic beverages, especially tea and coffee.

Surprisingly, Bourke Aborigines derive very little of their energy intake from fruit or locally caught fish, which is the major source of bush food. Only six respondents (3 per cent of all respondents) noted that they had consumed a piece a fruit during their recall period. This may be due in part to the fact that the survey was conducted in autumn and winter, when cheaper locally produced varieties of citrus fruit are not readily available. Locally caught fish species, including bony bream, freshwater catfish, silver perch, golden perch, Murray cod, and carp, contributed about 2 per cent of both male and female total energy intake. Even though fish and emu meat were the only bush foods that were recalled, I was informed that Aborigines in the area still eat kangaroos, wallabies, and yabbies (freshwater crayfish). However, it appears that the bush foods eaten today are only a small proportion of the plant and animal species which were eaten as part of the traditional diet of Bourke Aborigines (see Gillon and Knight, 1986).

Meat and meat products and cereal and cereal products also make a significant contribution to the total intake of protein, carbohydrates, and fat (see Table 7.2).

Approximately half of the total protein and fat intakes are derived from meat and meat products; while over a third of the total carbohydrate intake is derived from cereal and cereal products. Additionally, a notable proportion of the total protein intake is derived from cereal and cereal products and a large proportion of the total carbohydrate intake is derived from meat and meat products (mainly from take-aways and mixed dishes).

Overall, this analysis shows the underlying importance of meat and meat products and cereal and cereal products, especially lamb and bread, to the Bourke Aboriginal diet.

Table 7.1 Comparison of the contribution of different food groups to the total energy intake of a non-random sample of Bourke Aborigines¹ aged 18 years and over, by sex.

Food group and subgroup ²	Males (per cent) ³	Females (per cent) ³
Meat and meat products	45	32
Lamb	18	10
Take-away (non-fish)	9	5
Beef and veal	5	6
Poultry	2	3
Pork: ham, bacon, pork meat	2	1
All other meat items	9	8
Cereal and cereal products	20	25
Bread, all varieties	17	22
Breakfast cereals, all varieties	1	2
All other cereal items	2	2
Vegetables	9	9
Potatoes	6	6
All other vegetable items	3	4
Fats (all non-cooking uses)	7	8
Margarine, all varieties	6	8
All other fat items (including butter)	1	1
Milk and milk products	5	6
Milk, liquid	4	5
Cheeses	1	2
All other milk items	1	1
Beverages	7	8
Non-alcoholic	7	8
Alcoholic	0	0
All other food items	7	12
Total	100	100

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Source: Department of Community Services and Health (1987: 113-114, Appendix J).

3. Percentages are based on total energy intake and may not add up because of rounding.

Table 7.2 Comparison of the contribution of different food groups to the protein, carbohydrate, and fat intakes of a non-random sample of Bourke Aborigines¹ aged 18 years and over, by sex.

Food group or subgroup ²	Males (per cent) ³	Females (per cent) ³
Protein		
Meat and meat products	68	58
Cereal and cereal products	13	19
Milk and milk products	6	8
All other food items	13	15
Carbohydrates		
Cereal and cereal products	38	47
Non-alcoholic beverages	23	17
Meat and meat products	14	8
Vegetables	12	12
Sugar, jam, honey, and syrups	6	7
All other food items	7	9
Fat		
Meat and meat products	53	43
Fat (all non-cooking uses)	14	20
Vegetables	8	9
Eggs	6	5
Milk and milk products	6	9
All other food items	13	14

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Source: Department of Community Services and Health (1987: 113-114, Appendix J).

3. Percentages may not add up because of rounding.

7.3.2 Meal patterns

Meal patterns were appraised by evaluating the contribution of different meals and snacks to total energy intake. Meals and snacks were divided between those that were purchased and those that were prepared and eaten at home. As a rule, morning, midday, and evening meals tended to be prepared at home, while snacks tended to be purchased and eaten away from home. Of the three main meals, there was a greater tendency for the midday meal to be purchased, especially by men. Therefore, the contribution of purchased meals to total energy intake was greatest for the midday meal and snacks and lowest for morning and evening meals (see Table 7.3). However, only 16 per cent of male and 17 per cent of female total energy intake was derived from purchased meals. Sibthorpe (1988: 187) observed a similar pattern among adult Aborigines in Kempsey, New South Wales. She notes that respondents commonly bought food items as a snack or sometimes as a midday meal when they were away from home for one reason or another.

Meat and meat products and cereal and cereal products, especially lamb and bread, formed the basis of most meals (See Table 7.4). For both sexes, the contribution of meat and meat products to total energy intake was highest during the evening meal; while for cereal and cereal products it was highest during the morning meal. Food items consumed at the morning meal, however, made the greatest contribution to total energy intake (see Table 7.3).

Table 7.3 Comparison of the contribution of prepared and purchased meals or snacks to the total energy intake of a non-random sample of Bourke Aborigines¹ aged 18 years and over, by sex and meal type.

Meal type	Prepared meal (per cent) ²	Purchased meal (per cent) ²	Total (per cent) ²
Males			
Morning	32	2	34
Midday	22	8	30
Evening	26	2	28
Snacks	4	5	9
Total	84	16	100
Females			
Morning	32	1	33
Midday	17	10	27
Evening	28	0	28
Snacks	6	6	12
Total	83	17	100

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Percentages are based on total energy intake and may not add up because of rounding.

Table 7.4 Comparison of the contribution of meals and snacks to the total energy intake of a non-random sample of Bourke Aborigines¹ aged 18 years and over, by sex and food group.

Food group or subgroup ²	Morning (per cent) ³	Midday (per cent) ³	Evening (per cent) ³	Snacks (per cent) ³
Males				
Meat and meat products	34	38	60	31
Cereal and cereal products	30	18	10	18
Vegetables	2	13	12	10
Fats (all non-cooking uses)	10	8	3	5
Milk and milk products	8	4	2	3
Beverages	2	12	4	18
Eggs	10	1	2	1
All other food items	3	7	8	13
Total	100	100	100	100
Females				
Meat and meat products	17	34	51	15
Cereal and cereal products	38	21	15	26
Vegetables	3	12	10	14
Fats (all non-cooking uses)	13	8	5	7
Milk and milk products	12	6	2	5
Beverages	5	12	7	12
Eggs	9	1	1	1
All other food items	4	6	9	20
Total	100	100	100	100

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Source: Department of Community Services and Health (1987: 113-114, Appendix J).

3. Percentages are based on total energy intake and may not add up because of rounding.

7.4 Macro-nutrient intake

The macro-nutrients of concern in this study are total energy intake, protein, carbohydrates, fat, and dietary fibre. Energy intake is defined as the physiologically available or metabolizable energy yield of the foods actually consumed. Total energy intake, which is expressed in kilojoules⁵ (KJ), is calculated by estimating the energy derived from the total intake of various macro-nutrients⁶ in foods. The energy requirements of adults have been shown to be dependent on activity levels, body size, age, and on the climate and environment. However, the predominant factor leading to variability in energy needs is the proportion of time an individual devotes to moderate and heavy physical activities in contrast with light or sedentary activities (National Academy of Sciences, 1980: 19).

Food proteins provide amino acids for the synthesis of body proteins and many other tissue constituents. The requirement for protein is thus a requirement for amino acids, especially for the nine essential amino acids which are not synthesized by humans — histidine, isoleucine, leucine, lysine, methionine, phenylalanine, threonine, tryptophan, and valine (National Academy of Sciences, 1980: 39).

The principal carbohydrates in food are sugars and starches. The sugars include the monosaccharides and disaccharides found in refined sugars, jams, jellies, syrups, honey, fruits, soft drinks, and milk; while starches include the polysaccharides found in cereals, flour, potatoes, and other vegetables (National Academy of Sciences, 1980: 32).

5. The joule (J) is the accepted international unit of energy. Mean nutrient intakes are generally expressed in either kilojoules (1 kilojoule = 1,000 joules) or megajoules (1 megajoule = 10,000 joules). To convert total energy intake from joules to calories, divide by a factor of 4.184.

6. The calculation of these contributions, which takes into account the energy lost in human digestion and excretion processes, was made using the following nutrient conversion factors: protein 17 KJ, fat 37 KJ, carbohydrate 16 KJ, alcohol 29 KJ, and organic acids 10-15 KJ per gram (Department of Community Services and Health, 1987: 22).

Dietary fat, found in either a solid or a liquid state, is an essential nutrient because it is a concentrated source of energy, a carrier of fat-soluble vitamins, and an important source of essential fatty acids, phospholipids, and sterols, especially cholesterol. As noted in chapter 1, fat is also the most important dietary risk factor of coronary heart disease (see section 1.2.2.2). Keys (1952) was the first to suggest that the intake of fat elevates serum cholesterol levels and individuals with elevated serum cholesterol levels are at risk of developing atherosclerosis and coronary heart disease. Since then the ability to reduce concentrations of total serum cholesterol by limiting total fat and saturated fatty acid intakes has been widely demonstrated in patients who have high cholesterol readings (National Advisory Committee on Nutrition Education, 1983) and in metabolic studies (Keys and Palin, 1966).

Although no recommended levels of total fat intake exist for Australia (see National Health and Medical Research Council, 1987), various national and international governing bodies in other countries have recommended that fat intake, in populations at high risk of coronary heart disease, should be limited to between 20 and 35 per cent of total energy intake. In the United States, the National Academy of Sciences (1980: 35-37) recommends that total fat intake should not exceed 35 per cent of total energy intake, and that one-fourth to one-third (8 to 10 per cent) of total fat intake should be polyunsaturated (essential) fatty acids. In Britain, the Committee on Medical Aspects of Food Policy (Department of Health and Social Security, 1984: 5) suggests that energy derived from saturated fatty acids should be limited to 15 per cent and from total fat intake to 35 per cent of total energy intake. Finally, the World Health Organization (1982: 22) considers a moderate intake of fat (between 20 to 30 per cent of total energy intake), of which approximately a third is derived from saturated fatty acids, to be appropriate for a population with a high rate of coronary heart disease.

Fibre, another important dietary risk factor, is defined as the sum of the indigestible carbohydrate and carbohydrate-like components of food — including cellulose, lignin, hemicelluloses, pentosans, gums, and pectin (see section 1.2.2.2 in chapter 1).

Researchers have shown that the mean intake of dietary fibre is inversely related to the incidence of coronary heart disease (Morris *et al.*, 1977; Kromhout *et al.*, 1982; Kromhout, 1985). A similar relationship has also been noted with starch intake. However, the physiological significance of either of these relationships has not yet been adequately researched.

Following the analysis of macro-nutrient intake, two types of comparisons were made. First, a comparison was made between the mean daily nutrient intakes of Bourke Aborigines and all Australians. All Australian dietary information comes from a 24-hour dietary recall survey conducted by the Department of Community Services and Health (1987) in conjunction with the 1983 National Heart Foundation (1985) risk factor prevalence study. In total, recalls were taken from a random stratified sample of 3,027 males and 3,228 females aged 25 years and over living in capital cities throughout Australia. Although my survey collected similar information on the nutrient intakes of adults aged 18 years and over, for comparative purposes this analysis was limited to those aged 25 years and over. Second, comparisons were made between the mean daily nutrient intakes of specific groups of Bourke Aborigines aged 18 years and over as defined by various risk markers — age, location, and pay period. Pay period was used as a proxy measure of income variability between the week when pension benefits were paid out and the alternate week when they were not. This analysis was limited to these three risk markers because they are the most meaningful in this instance and because they have been used before in other Aboriginal dietary studies. In both types of comparisons, differences in mean daily nutrient intakes were assessed using population means and sample distributions. The second type of comparison also relied on the Brown-Mood non-parametric median test to determine whether the intake of particular nutrients had similar distributions across different groups. As noted above, the main aim here is to assess whether the mean daily macro-nutrient intake patterns of Bourke Aborigines, as determined from their dietary habits, are placing them at risk of coronary heart disease.

7.4.1 Comparison with national dietary survey

The mean daily intakes of energy, protein, carbohydrates, starch, sugar, fat, saturated fatty acids, cholesterol, and dietary fibre for Bourke Aborigines and all Australians are summarized in Tables 7.5 and 7.6. Compared with all Australians, Bourke Aborigines generally have significantly lower mean daily intakes of energy, carbohydrates, sugar, and dietary fibre and comparable mean daily intakes of total fat and saturated fatty acids. Additionally, Aboriginal men have significantly lower mean daily intakes of protein and starch than their all Australian counterparts.

The most notable difference between the two populations is in their mean daily intake of sugar. Aboriginal mean daily intake levels are half of those observed for all Australians. Although these observed levels seem rather low (50 grams per day for both men and women), Sibthorpe (1988: 230) has noted a similar mean daily intake level for Kempsey Aboriginal women of 65 grams per day and a higher but not statistically different level for men of 86 grams per day. This low intake of sugar is most likely due to the fact that at the time of the survey Bourke Aborigines consumed little fruit and milk and milk products, which are two important sources of sugar in the diets of all Australians (Department of Community Services and Health, 1987: 31).

Another notable difference is in the mean daily intake of dietary fibre. Again Aboriginal mean daily intake levels are only half of those observed for all Australians. This low intake of fibre is most likely due to the fact that unlike all Australians, Bourke Aborigines eat little wholegrain bread or cereal products and fruit. Aboriginal mean daily intake of starch is also low. In absolute terms, however, Bourke Aborigines derive a greater proportion of their mean daily intake of carbohydrates from starch than do all Australians — approximately 64 and 63 per cent for male and female Aborigines versus 55 and 52 per cent for males and females in the all Australian population.

The most notable similarities between the two populations are in the mean daily intakes of total fat, saturated fatty acids, and cholesterol. Among males Aborigines, the mean

daily intakes of total fat and saturated fatty acids are slightly lower; while among female Aborigines, they are identical to the levels observed for their all Australian counterparts. Additionally, Aborigines have slightly lower or higher but not significantly different mean daily cholesterol intake levels. However, male Aborigines derive a much greater proportion of their total fat intake from saturated fatty acids. The ratio of polyunsaturated to saturated fat intake among Bourke Aborigines is 0.29/1 for males and 0.35/1 for females; while among all Australians it is 0.36/1 for both sexes. This difference is probably due to the fact that Bourke Aborigines, especially men, derive about half of their fat intake from meat and meat products, compared to a third for all Australians (Department of Community Services and Health, 1987: 33).

In addition to comparing mean macro-nutrient intake levels, the contribution of various macro-nutrients to total energy intake was assessed in both populations using Atwater's conversion factors (see footnote 6). The estimated contribution of alcohol intake to total energy intake for Bourke Aborigines was indirectly determined using the lifestyle questions in the survey on the frequency and intensity of drinking habits (see Table A3.5 in Appendix 3). These questions were used to determine the average number of drinks consumed by Aboriginal men and women, which were then converted into sex-specific estimates of alcohol intake⁷. Although these estimates are only proxy measures, they give some idea of the proportion of total energy intake derived from alcohol. Compared to all Australians, Bourke Aborigines derive a greater proportion of their total energy intake from fat, alcohol, and protein, and less of it from carbohydrates (see Figure 7.1). The proportion of total energy intake derived from fat (45 per cent for males and 42 per cent for females) is well over the recommended levels of between 20 and 35 per cent for populations with a high rate of coronary heart disease (see section 7.4). By these standards all Australians derive too much of their total energy intake from dietary fat as well. Overall, the comparatively high intake of fat and low intake of

7. Based on the assumption that a drink of alcohol referred to one 250 millilitre glass of beer with a 3 per cent alcohol content.

dietary fibre could be contributing to the high rate of coronary heart disease among Bourke Aborigines.

Table 7.5 Comparison of the mean daily intakes of energy and selected macro-nutrients for a non-random sample of male Bourke Aborigines¹ and all Australians², for persons aged 25 years and over.

	Mean	Standard error ³	Percentiles		
			25	50	75
Bourke Aborigines					
Energy (KJ)	7,737	694	6,578	7,816	8,895
Protein (g)	90	12	55	90	118
Carbohydrates (g)	141	15	104	143	178
Starch (g)	91	11	68	88	110
Sugar (g)	50	8	26	52	69
Total fat (g)	102	11	70	100	129
Saturated fatty acids (g)	42	5	29	41	53
Cholesterol (mg)	432	91	179	351	604
Dietary fibre (g)	9.6	1.2	6.0	9.0	12.0
All Australians					
Energy (KJ)	11,010	90	8,330	10,600	13,140
Protein (g)	106	1	76	100	129
Carbohydrates (g)	274	3	193	260	340
Starch (g)	151	2	103	142	186
Sugar (g)	123	2	75	111	159
Total fat (g)	110	1	75	105	137
Saturated fatty acids (g) ⁴	44	1	29	42	56
Cholesterol (mg)	417	6	225	362	543
Dietary fibre (g)	23.2	0.3	14.6	20.6	29.1

1. Aboriginal risk factor prevalence survey conducted in April and May 1989. Non-random sample of 55 males aged 25 years and over.

2. Source: Department of Community Services and Health (1987: 42-77, Tables 8.6.1 to 8.8.1).
Random sample of 3,027 males aged 25 years and over.

3. Two standard errors around the mean.

4. Source: Department of Community Services and Health (1988: 19A, Table 7.4.1).

Table 7.6 Comparison of the mean daily intakes of energy and selected macro-nutrients for a non-random sample of female Bourke Aborigines¹ and all Australians², for persons aged 25 years and over.

	Mean	Standard error ³	Percentiles		
			25	50	75
Bourke Aborigines					
Energy (KJ).	6,245	893	4,151	5,555	7,511
Protein (g)	65	11	42	55	83
Carbohydrates (g)	139	20	88	124	178
Starch (g)	88	12	62	86	103
Sugar (g)	50	13	25	44	60
Total fat (g)	75	13	46	62	90
Saturated fatty acids (g)	31	6	18	25	39
Cholesterol (mg)	258	61	90	213	416
Dietary fibre (g)	10.0	1.8	6.0	9.0	13.0
All Australians					
Energy (KJ)	7,410	70	5,420	7,090	9,050
Protein (g)	74	1	52	70	92
Carbohydrates (g)	192	2	135	180	236
Starch (g)	100	1	67	93	127
Sugar (g)	92	1	55	82	117
Total fat (g)	76	1	50	70	96
Saturated fatty acids (g) ⁴	31	0	19	28	39
Cholesterol (mg)	311	8	157	253	399
Dietary fibre (g)	18.7	0.3	11.9	17.1	23.6

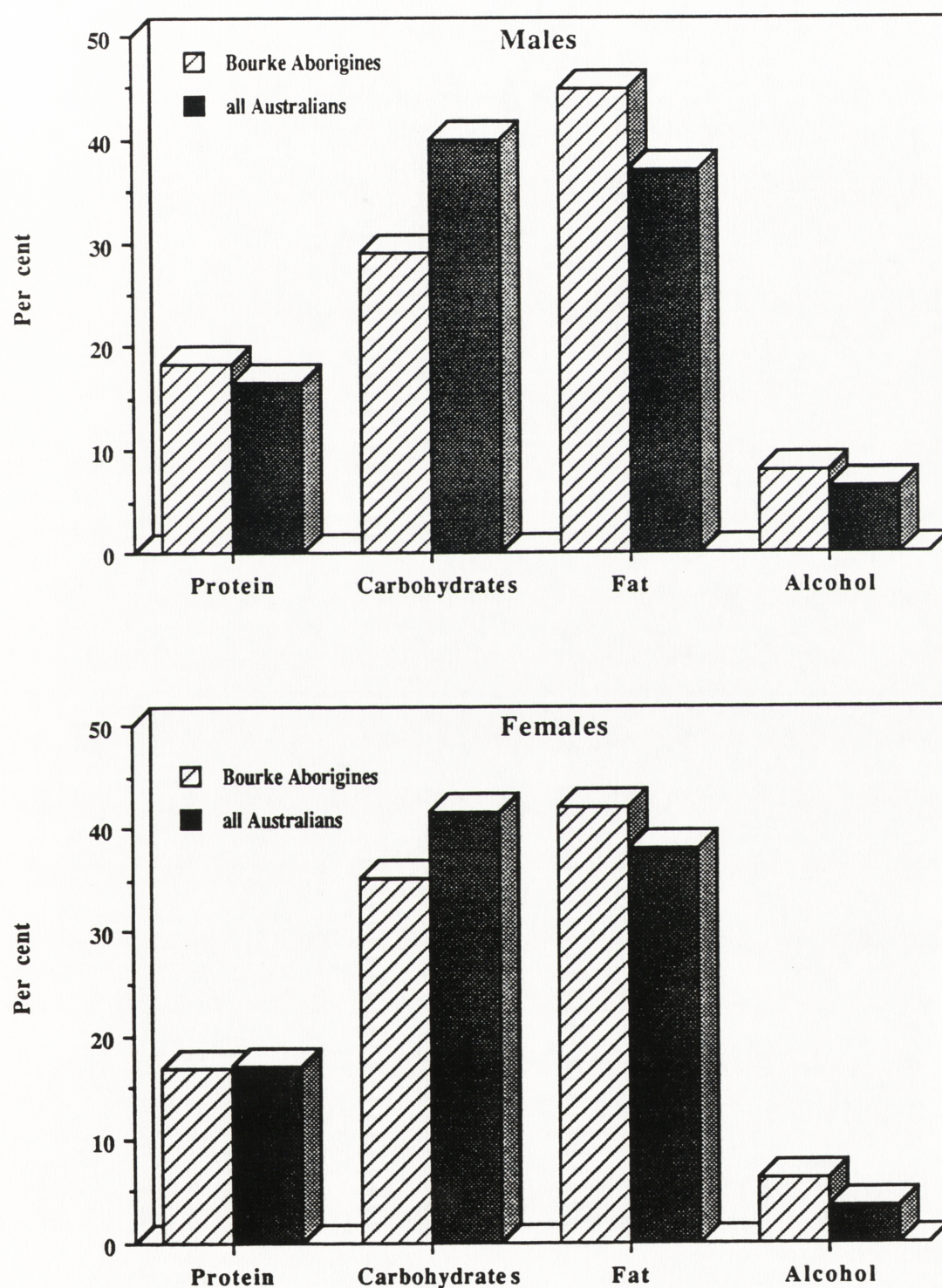
1. Aboriginal risk factor prevalence survey conducted in April and May 1989. Non-random sample of 41 females aged 25 years and over.

2. Source: Department of Community Services and Health (1987: 42-77, Tables 8.6.1 to 8.8.1).
Random sample of 3,228 females aged 25 years and over.

3. Two standard errors around the mean.

4. Source: Department of Community Services and Health (1988: 19A, Table 7.4.1).

Figure 7.1 Comparison of the proportion of total energy derived from various macronutrients for a non-random sample of Bourke Aborigines¹ and all Australians², by sex, for persons aged 25 years and over.



1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Source: Department of Community Services and Health (1987: xiv).

3. Based on the energy derived from the total intake of protein (17 KJ per gram), starch (16 KJ per gram), and fat (37 KJ per gram) and the estimated intake of alcohol (29 KJ per gram).

7.4.2 Comparison of Bourke Aboriginal mean daily macro-nutrient intake levels

Among Bourke Aborigines, there are many notable differences in the mean daily intakes of energy, protein, starch, sugar, fat, saturated fatty acids, cholesterol, and dietary fibre by age, location, and pay period (see Tables 7.7 to 7.14).

The most consistent and discernible variation in the mean daily intakes of these selected macro-nutrients is by pay period. As a rule, the mean daily intakes recorded during pay week are higher than those recorded during off pay week. The only exception appears to be with the mean daily intake of cholesterol among males which is practically identical by pay period (see Table 7.13). Sibthorpe (1988: 262) has noted a similar phenomenon among Kempsey Aborigines, although apparent fluctuations in nutrient intake did not always go in the expected direction. Among males, there was a rise in the mean daily intakes of energy, protein, fat, magnesium, iron, zinc, vitamin A, and niacin and a fall in the mean daily intakes of carbohydrates, calcium, and vitamin C during off pay week; while among females, during the same week there was a fall in the mean daily intakes of all nutrients with the exception of starch, cholesterol, and zinc, which rose marginally. Although Sibthorpe's data does not demonstrate as consistent a pattern as the Bourke Aboriginal data, both comparisons suggest that weekly variations in available income put considerable constraints on the foods that Aborigines purchase and consume.

Additionally, mean macro-nutrient intakes vary considerably by location. This analysis shows that Bourke Aboriginal men and women living on or near the mission tend to have higher mean daily intakes of all macro-nutrients than those living in town. The most significant difference by location is for male mean daily intakes of total fat and saturated fatty acids, which for those living on or near the mission are approximately 25 per cent higher (see Tables 7.11 and 7.12). The only exception to this trend appears to be with the mean daily intakes of starch and sugar among women, where there is little difference by location (see Tables 7.9 and 7.10). However, Kamien and associates (1975a) in Bourke and Hitchcock and Gracey (1975) in a community in southwestern

Australia have described a rather different picture — they suggest that mean daily intakes are higher for those Aborigines living in town. The most likely reason why this survey shows respondents living in town to have lower mean macro-nutrient intakes is that a greater proportion of them, compared to those living on or near the mission, are overweight and obese (see section 6.3.2 in chapter 6). As noted in chapter 1, there is a strong inverse relationship between total energy intake (and therefore between other macro-nutrients) and being overweight or obese (see section 1.2.2.1).

Although mean macro-nutrient intakes generally tend to decrease with age, there appear to be many exceptions to this rule. Among males, the mean daily intake of protein increases 9 per cent between the age groups 18 to 24 years and 35 years and over — from 88 to 96 grams (see Table 7.8). Furthermore, the mean daily intakes of total fat and saturated fatty acids among males are relatively stable with age — around 111 grams of total fat and 43 grams of saturated fatty acids (see Tables 7.11 and 7.12). Additionally, the male mean daily intake of cholesterol tends to fluctuate around 450 milligrams (see Table 7.13). Among females, the mean daily intake of saturated fatty acids remains relatively stable with age as well — only dropping 4 grams between the age groups 18 to 24 years and 35 years and over (see Table 7.12). Overall, this suggests that among Bourke Aborigines fat intake remains relatively stable with age, while the intake of other macro-nutrients generally decreases.

Table 7.7 Energy intake (KJ) for a non-random sample of Bourke Aborigines¹ aged 18 years and over, by sex, age, location, and pay period.

	n ²	Mean ³	Standard error ⁴	Percentiles			Median test ⁵
				25	50	75	
Males							
Age group							n.s.
18-24	70	8,573	894	6,099	8,172	9,859	
25-34	34	7,650	761	6,422	7,396	8,436	
35 plus	21	7,877	682	5,813	8,552	10,077	
All ages	125	8,205	589	6,239	7,926	9,622	
Location ⁶							n.s.
Mission	39	9,069	1,006	6,949	8,485	10,141	
Town	32	7,520	1,331	4,770	6,739	8,768	
Pay period							n.s.
Off pay week	48	7,643	822	5,684	7,311	9,369	
Pay week	77	8,555	801	6,506	8,345	10,070	
Females							
Age group							0.01
18-24	50	7,236	955	5,210	7,239	9,266	
25-34	18	6,409	1,054	4,811	5,979	7,236	
35 plus	23	6,116	1,379	3,873	4,972	9,598	
All ages	91	6,789	665	4,537	6,085	8,739	
Location ⁶							n.s.
Mission	20	7,395	1,375	5,492	6,444	10,006	
Town	23	6,906	1,223	4,537	6,418	9,259	
Pay period							n.s.
Off pay week	51	6,371	782	4,539	5,939	8,035	
Pay week	40	7,322	1,129	4,247	6,923	9,521	

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Total number of respondents, excluding 'not stated'.

3. Mean energy intake (KJ).

4. Two standard errors around the mean.

5. Brown-Mood non-parametric median statistical test was used to determine if the intake of energy had the same distribution across different groups or categories.

6. Mission refers to people living on or near the mission; while town refers to people living in town.

Table 7.8 Protein intake (g) for a non-random sample of Bourke Aborigines¹ aged 18 years and over, by sex, age, location, and pay period.

	n ²	Mean ³	Standard error ⁴	Percentiles			Median test ⁵
				25	50	75	
Males							
Age group							n.s.
18-24	70	88	11	53	90	116	
25-34	34	87	14	55	80	111	
35 plus	21	96	20	63	97	132	
All ages	125	89	8	55	90	117	
Location ⁶							n.s.
Mission	39	95	12	64	95	124	
Town	32	81	16	40	70	102	
Pay period							n.s.
Off pay week	48	85	14	46	74	112	
Pay week	77	92	10	62	92	118	
Females							
Age group							n.s.
18-24	50	71	11	42	66	93	
25-34	18	66	14	48	56	83	
35 plus	23	63	16	25	54	100	
All ages	91	68	8	42	61	93	
Location ⁶							n.s.
Mission	20	77	17	44	71	106	
Town	23	73	16	47	73	91	
Pay period							n.s.
Off pay week	51	64	9	42	59	82	
Pay week	40	73	12	44	64	102	

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Total number of respondents, excluding 'not stated'.

3. Mean protein intake (g).

4. Two standard errors around the mean.

5. Brown-Mood non-parametric median statistical test was used to determine if the intake of protein had the same distribution across different groups or categories.

6. Mission refers to people living on or near the mission; while town refers to people living in town.

Table 7.9 Starch intake (g) for a non-random sample of Bourke Aborigines¹ aged 18 years and over, by sex, age, location, and pay period.

	n ²	Mean ³	Standard error ⁴	Percentiles			Median test ⁵
				25	50	75	
Males							
Age group							n.s.
18-24	70	108	12	74	100	137	
25-34	34	96	14	69	89	117	
35 plus	21	83	18	49	80	109	
All ages	125	101	8	70	94	130	
Location ⁶							n.s.
Mission	39	107	14	80	100	137	
Town	32	102	18	67	92	138	
Pay period							n.s.
Off pay week	48	97	11	74	92	117	
Pay week	77	103	12	66	95	137	
Females							
Age group							n.s.
18-24	50	104	15	67	95	143	
25-34	18	90	15	65	88	104	
35 plus	23	87	20	49	75	102	
All ages	91	97	10	63	87	121	
Location ⁶							n.s.
Mission	20	98	18	68	88	117	
Town	23	99	11	45	104	148	
Pay period							n.s.
Off pay week	51	90	12	62	86	117	
Pay week	40	106	18	66	94	149	

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Total number of respondents, excluding 'not stated'.

3. Mean starch intake (g).

4. Two standard errors around the mean.

5. Brown-Mood non-parametric median statistical test was used to determine if the intake of starch had the same distribution across different groups or categories.

6. Mission refers to people living on or near the mission; while town refers to people living in town.

Table 7.10 Sugar intake (g) for a non-random sample of Bourke Aborigines¹ aged 18 years and over, by sex, age, location, and pay period.

	n ²	Mean ³	Standard error ⁴	Percentiles			Median test ⁵
				25	50	75	
Males							
Age group							n.s.
18-24	70	65	15	31	53	66	
25-34	34	54	9	34	56	74	
35 plus	21	42	14	19	30	61	
All ages	125	58	9	26	52	72	
Location ⁶							n.s.
Mission	39	68	25	28	50	70	
Town	32	52	12	25	54	75	
Pay period							n.s.
Off pay week	48	49	10	23	45	67	
Pay week	77	64	14	30	55	76	
Females							
Age group							n.s.
18-24	50	62	12	34	52	88	
25-34	18	45	13	29	38	56	
35 plus	23	54	19	21	49	69	
All ages	91	56	9	28	48	71	
Location ⁶							n.s.
Mission	20	54	13	34	41	74	
Town	23	56	19	22	46	70	
Pay period							n.s.
Off pay week	51	51	12	22	38	65	
Pay week	40	63	13	35	55	73	

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Total number of respondents, excluding 'not stated'.

3. Mean sugar intake (g).

4. Two standard errors around the mean.

5. Brown-Mood non-parametric median statistical test was used to determine if the intake of sugar had the same distribution across different groups or categories.

6. Mission refers to people living on or near the mission; while town refers to people living in town.

Table 7.11 Total fat intake (g) for a non-random sample of Bourke Aborigines¹ aged 18 years and over, by sex, age, location, and pay period.

				Percentiles			
	n ²	Mean ³	Standard error ⁴	25	50	75	Median test ⁵
Males							
Age group							n.s.
18-24	70	111	13	77	101	128	
25-34	34	97	13	69	92	112	
35 plus	21	111	22	76	126	144	
All ages	125	107	9	75	100	129	
Location ⁶							0.05
Mission	39	121	14	86	115	141	
Town	32	93	18	51	91	103	
Pay period							n.s.
Off pay week	48	100	13	68	94	125	
Pay week	77	112	12	80	105	131	
Females							
Age group							n.s.
18-24	50	86	14	55	79	114	
25-34	18	80	18	47	78	90	
35 plus	23	71	20	36	55	82	
All ages	91	81	10	47	69	105	
Location ⁶							n.s.
Mission	20	93	22	59	80	137	
Town	23	82	16	56	78	96	
Pay period							n.s.
Off pay week	51	78	12	47	68	103	
Pay week	40	86	16	46	76	119	

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Total number of respondents, excluding 'not stated'.

3. Mean total fat intake (g).

4. Two standard errors around the mean.

5. Brown-Mood non-parametric median statistical test was used to determine if the intake of total fat had the same distribution across different groups or categories.

6. Mission refers to people living on or near the mission; while town refers to people living in town.

Table 7.12 Saturated fatty acid intake (g) for a non-random sample of Bourke Aborigines¹ aged 18 years and over, by sex, age, location, and pay period.

	n ²	Mean ³	Standard error ⁴	Percentiles			Median test ⁵
				25	50	75	
Males							
Age group							n.s.
18-24	70	43	5	28	40	56	
25-34	34	40	6	29	38	48	
35 plus	21	46	10	30	50	66	
All ages	125	43	4	28	40	54	
Location ⁶							0.05
Mission	39	49	6	35	45	62	
Town	32	38	8	21	34	42	
Pay period							n.s.
Off pay week	48	39	6	25	36	48	
Pay week	77	46	5	30	42	58	
Females							
Age group							n.s.
18-24	50	34	6	22	31	42	
25-34	18	33	8	21	30	41	
35 plus	23	30	9	13	22	36	
All ages	91	33	4	18	28	42	
Location ⁶							n.s.
Mission	20	39	9	23	31	59	
Town	23	33	7	22	34	41	
Pay period							n.s.
Off pay week	51	31	5	18	27	40	
Pay week	40	35	7	18	31	43	

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Total number of respondents, excluding 'not stated'.

3. Mean saturated fatty acid intake (g).

4. Two standard errors around the mean.

5. Brown-Mood non-parametric median statistical test was used to determine if the intake of saturated fatty acids had the same distribution across different groups or categories.

6. Mission refers to people living on or near the mission; while town refers to people living in town.

Table 7.13 Cholesterol intake (mg) for a non-random sample of Bourke Aborigines¹ aged 18 years and over, by sex, age, location, and pay period.

	n ²	Mean ³	Standard error ⁴	Percentiles			Median test ⁵
				25	50	75	
Males							
Age group							n.s.
18-24	70	462	111	175	348	554	
25-34	34	383	98	155	301	592	
35 plus	21	511	176	245	403	626	
All ages	125	449	74	180	349	570	
Location ⁶							n.s.
Mission	39	469	92	191	487	711	
Town	32	439	200	115	310	572	
Pay period							n.s.
Off pay week	48	448	154	128	337	539	
Pay week	77	449	74	196	352	595	
Females							
Age group							n.s.
18-24	50	312	71	99	232	524	
25-34	18	281	101	103	250	460	
35 plus	23	239	75	77	213	372	
All ages	91	287	47	91	218	448	
Location ⁶							n.s.
Mission	20	356	107	200	290	534	
Town	23	302	92	123	265	435	
Pay period							n.s.
Off pay week	51	285	70	87	202	501	
Pay week	40	290	64	110	268	433	

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Total number of respondents, excluding 'not stated'.

3. Mean cholesterol intake (mg).

4. Two standards errors around the mean.

5. Brown-Mood non-parametric median statistical test was used to determine if the intake of cholesterol had the same distribution across different groups or categories.

6. Mission refers to people living on or near the mission; while town refers to people living in town.

Table 7.14 Dietary fibre intake (g) for a non-random sample of Bourke Aborigines¹ aged 18 years and over, by sex, age, location, and pay period.

	n ²	Mean ³	Standard error ⁴	Percentiles			Median test ⁵
				25	50	75	
Males							
Age group							n.s.
18-24	70	11.8	1.4	7.0	10.0	16.0	
25-34	34	9.9	1.6	6.0	9.5	12.2	
35 plus	21	9.0	1.8	5.5	9.0	12.5	
All ages	125	10.8	1.0	7.0	10.0	14.0	
Location ⁶							n.s.
Mission	39	10.7	1.8	7.0	10.0	13.0	
Town	32	11.6	2.0	8.2	10.5	13.8	
Pay period							n.s.
Off pay week	48	10.3	1.4	7.0	10.0	12.8	
Pay week	77	11.1	1.4	7.0	10.0	15.0	
Females							
Age group							n.s.
18-24	50	11.9	2.2	6.8	11.0	15.0	
25-34	18	10.0	1.6	6.8	8.5	11.3	
35 plus	23	10.0	1.4	5.0	9.0	13.0	
All ages	91	11.0	1.4	6.0	9.0	14.0	
Location ⁶							n.s.
Mission	20	11.7	1.6	7.2	10.0	15.8	
Town	23	11.1	1.8	7.0	10.0	14.0	
Pay period							0.05
Off pay week	51	10.0	1.4	7.0	9.0	13.0	
Pay week	40	12.4	1.6	5.2	11.0	16.8	

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Total number of respondents, excluding 'not stated'.

3. Mean dietary fibre intake (g).

4. Two standards errors around the mean.

5. Brown-Mood non-parametric median statistical test was used to determine if the intake of dietary fibre had the same distribution across different groups or categories.

6. Mission refers to people living on or near the mission; while town refers to people living in town.

7.5 Micro-nutrient intake

As well as assessing macro-nutrient intake levels, an appraisal was made of the contribution of various vitamins and minerals to the Bourke Aboriginal diet. The micro-nutrients of concern are retinol equivalents (vitamin A), thiamin (vitamin B), riboflavin (vitamin B₂), niacin equivalents (vitamin B₃), vitamin C, iron, calcium, zinc, magnesium, and sodium.

Vitamin A, expressed as retinol equivalents, is present in two forms — retinol and beta-carotene. Retinol, the active form of vitamin A, occurs preformed in foods of animal origin, especially milk products, egg yolk, and liver; and beta-carotene, which is converted into retinol in the human body, is present in animal foods as well as in fruit and vegetables. Vitamin A is essential for normal growth and reproduction, vision in dim light, and the maintenance of the differentiated functions of soft mucus-secreting tissues.

Vitamin B complex includes any one of a group of water-soluble vitamins that have varied metabolic functions as well as coenzymes and growth hormones (National Academy of Sciences, 1980). This group of vitamins is often found together in foods like yeast, seed germs, eggs, liver, and some vegetables. Thiamin (vitamin B₁) is essential to normal metabolism and nerve function. Riboflavin (Vitamin B₂), which functions as a growth hormone, occurs both in free form (as in milk) and combined (as in liver). Vitamin B₃ (nicotinic acid or nicotinamide), expressed as niacin equivalents, can be formed in the body in small amounts from the essential amino acid tryptophan. Good sources of niacin are meat, yeast extract, and some cereals.

Ascorbic acid (vitamin C) is a water-soluble vitamin which is essential to maintaining healthy connective tissues in the body; it is also a powerful anti-oxidant and can play a role in maintaining iron in a more bio-available state. Rich sources of vitamin C are citrus fruits and vegetables.

Iron is an essential element in the human body. Especially important are its roles in respiration, as an oxygen carrier in haemoglobin, and in the electron transport chain (National Academy of Sciences, 1980: 137). Animal sources of iron (heme-iron) are better absorbed than inorganic iron (such as the iron found in leafy green vegetables).

Calcium, zinc, and magnesium also play important roles in metabolic processes (Davidson *et al.*, 1979; National Academy of Sciences, 1980). Calcium is a metallic element that is an important constituent of bones, teeth, and blood and is essential for many metabolic processes, including nerve function, muscle contraction, and blood clotting. Zinc is a cofactor for more than 90 enzymes and is therefore involved in a variety of processes including the metabolism of alcohol and wound healing.

Magnesium is necessary for the proper functioning of muscle and nervous tissue, and functions as a cofactor for several of the enzyme systems responsible for cellular respiration and for the formation and transfer of high-energy phosphate groups.

Finally, sodium is an electrolyte which is involved primarily with the maintenance of osmotic equilibrium and extracellular fluid volume. Virtually all sodium in the diet is added to foods as salt (NaCl), either through discretionary use or in commercial or convenience foods (National Academy of Sciences, 1980: 170). Natural foods, especially unprocessed vegetables, contain relatively little sodium, but large amounts may be added during cooking, either as table salt (NaCl) or as sodium bicarbonate. Salt is also added in most forms of food processing and preserving (Davidson *et al.*, 1979: 84).

Two of these micro-nutrients have been shown to play a role in the development of hypertension. As noted in chapter 1, studies have shown that sodium intake is positively related to blood pressure, while calcium intake seems to be inversely related (see section 1.2.2.2).

In this study, intakes of the above micro-nutrients were used to make two comparisons. First, a comparison was made between the mean micro-nutrient intake levels of Bourke

Aborigines, aged 25 years and over, and all Australians in the same age group. Second, a comparison was made between mean sodium intake (from table salt) of specific groups of male and female Bourke Aborigines aged 18 years and over as defined by various risk markers — age, location, and pay period. In both comparisons differences in mean daily micro-nutrient intakes were assessed using population means and sample distributions. The second comparison also relied on the Brown-Mood non-parametric median test to determine whether sodium intake had a similar distributions across different groups. Again, the main aim here is to assess whether the mean daily micro-nutrient intake patterns of Bourke Aborigines, as determined from their dietary habits, are placing them at risk of coronary heart disease.

7.5.1 Comparison with national dietary survey

The mean daily intakes of various micro-nutrients for Bourke Aborigines and all Australians are summarized in Tables 7.15 and 7.16. As a rule, Bourke Aboriginal mean daily intakes were significantly lower than those for all Australians — the only difference that was not statistically significant was in the mean daily intake of retinol equivalents. However, this difference is due in part to the large individual variation in measured retinol intake, especially from foods like liver that have a high concentration of retinol.

The most substantial difference between the two populations is in the mean daily intakes of vitamin C and calcium. Among males, Bourke Aborigines have an mean daily intake of vitamin C which is 25 per cent of what is observed for all Australians; while among females, it is approximately 40 per cent. For Aborigines of both sexes, mean daily calcium intakes are about 50 per cent of those exhibited by their all Australian counterparts. These low mean daily intakes of both vitamin C and calcium are probably due to the fact that the Bourke Aboriginal diet is low in milk and milk products (which contain calcium) and fruit and vegetables (which contain vitamin C). Low mean daily calcium intakes could possibly be predisposing respondents to

hypertension, although this claim would be very difficult to substantiate until further research has been conducted in this area.

The mean daily intakes of these nine micro-nutrients for Bourke Aborigines aged 25 years and over were also assessed against Australian recommended dietary intakes (see Tables A4.3 to A4.4 in Appendix 4). As a rule, the mean daily intakes of thiamin, riboflavin, calcium, zinc, and magnesium were substantially lower than the recommended levels.

Table 7.15 Comparison of the mean daily intakes of selected micro-nutrients for a non-random sample of male Bourke Aborigines¹ and all Australians², for persons aged 25 years and over.

	Mean	Standard error ³	Percentiles		
			25	50	75
Bourke Aborigines					
Retinol equivalents (µg)	2,402	3,211	502	840	1,032
Thiamin (mg)	0.7	0.0	0.5	0.6	0.9
Riboflavin (mg)	1.5	0.4	0.8	1.2	1.6
Niacin equivalents (mg)	31.4	4.0	21.7	30.5	36.8
Vitamin C (mg)	32	9	13	24	39
Iron (mg)	11.6	1.4	8.2	11.0	13.7
Calcium (mg)	396	74	205	315	501
Zinc (mg)	11.6	1.6	6.5	10.9	16.1
Magnesium (mg)	164	15.8	127	157	197
All Australians					
Retinol equivalents (µg)	1,420	100	540	920	1,470
Thiamin (mg)	1.4	0.0	0.9	1.3	1.7
Riboflavin (mg)	2.3	0.0	1.5	2.1	2.8
Niacin equivalents (mg)	41.2	0.4	29.8	38.8	49.7
Vitamin C (mg)	127	3	37	79	171
Iron (mg)	15.9	0.2	11.2	15.0	19.5
Calcium (mg)	876	11	531	790	1,118
Zinc (mg)	14.7	0.2	9.4	13.4	18.1
Magnesium (mg)	276	3	198	259	331

1 Aboriginal risk factor prevalence survey conducted in April and May of 1989. Non-random sample of 55 males aged 25 years and over.

2. Source: Department of Community Services and Health (1987: 42-77, Tables 8.6.1 to 8.8.1). Random sample of 3,027 males aged 25 years and over.

3. Two standard errors around the mean.

Table 7.16 Comparison of the mean daily intakes of selected micro-nutrients for a non-random sample of female Bourke Aborigines¹ and all Australians², for persons aged 25 years and over.

	Mean	Standard error ³	Percentiles		
			25	50	75
Bourke Aborigines					
Retinol equivalents (µg)	1,456	1,731	278	435	905
Thiamin (mg)	0.7	0.2	0.4	0.6	0.8
Riboflavin (mg)	1.2	0.2	0.6	1.0	1.6
Niacin equivalents (mg)	23.1	3.8	13.3	20.0	34.5
Vitamin C (mg)	45	36	6	20	36
Iron (mg)	8.5	1.4	4.9	7.5	10.3
Calcium (mg)	345	56	242	311	439
Zinc (mg)	8.5	1.8	4.0	7.5	10.5
Magnesium (mg)	148	18	108	136	184
All Australians					
Retinol equivalents (µg)	1,820	350	440	750	1,230
Thiamin (mg)	1.0	0.0	0.7	1.0	1.3
Riboflavin (mg)	1.8	0.1	1.1	1.5	2.1
Niacin equivalents (mg)	28.7	0.4	20.0	26.9	34.8
Vitamin C (mg)	112	3	37	77	159
Iron (mg)	11.6	0.2	7.8	10.5	14.0
Calcium (mg)	707	12	424	630	888
Zinc (mg)	10.6	0.2	6.5	9.2	13.0
Magnesium (mg)	276	3	198	259	331

1. Aboriginal risk factor prevalence study conducted in April and May 1989. Non-random sample of 41 females aged 25 years and over.

2. Source: Department of Community Services and Health (1987: 42-77, Tables 8.6.1 to 8.8.1). Random sample of 3,228 females aged 25 years and over.

3. Two standard errors around the mean.

7.5.2 Comparison of Bourke Aboriginal sodium intake

As discussed in chapter 5 (see section, 5.4.1), sodium intake was assessed using a food frequency questionnaire (Millar and Beard, 1988). This checklist was designed to measure sodium intake from discretionary use (salt added in cooking or at the table) and from manufactured foods salted before purchase. The 21 items listed were chosen to represent a comprehensive register of the sources of added sodium found in a Western diet (see individual data sheets 12 and 13 in Appendix 2). A respondent's sodium intake score was obtained by adding the scores of all 21 items for a three day retrospective recall period — the higher the total score the higher the intake of dietary sodium.

This technique was chosen because it was inexpensive and required little time or commitment from the respondents. Furthermore, unlike other questionnaires (see Dahl and Love, 1954) or the 24-hour dietary recall method, this checklist is a more accurate measure of sodium intake because it is moderately, but significantly, correlated with the potassium:sodium ratio and sodium excretion levels measured from 24-hour urinary samples (Millar and Beard, 1988).

As shown in Table 7.17, the mean sodium intake score for Aboriginal males was 45 and for females was 39. For both sexes these totals were based on relatively high mean scores for the intake of sodium from discretionary use and from cured meats, bread, butter or margarine, yeast extracts, and dressings, sauces and pickles; and on relatively low mean scores for the intake of sodium from cheese, canned goods, and pharmaceutical products. The most appreciable difference in these sodium intake scores was by location for males and by pay period for both sexes. On average, men living in town had higher scores than those living on or near the mission; while men and women surveyed during pay week had higher mean scores than those surveyed during off pay week. Overall, this pattern may partially explain why the prevalence of diastolic hypertension is slightly higher in town and possibly suggests a relationship between sodium intake and income.

Bourke Aborigines also had significantly higher mean sodium intake scores than a sample of Australian tertiary students (see Millar and Beard, 1988). A mean sodium intake score of 28 for the student population was approximately 30 to 40 per cent less than that found for Bourke Aborigines. This comparison is stated here for interest's sake only, although it may give some idea of the possible differences in sodium intake levels that exist between Aboriginal and other Australian populations.

Table 7.17 Sodium intake score for a non-random sample of Bourke Aborigines¹ aged 18 years and over, by sex, age, location, and pay period.

	n ²	Mean	Standard error ³	Percentiles			Median test ⁴
				25	50	75	
Males							
Age group							n.s.
18-24	78	48	5	31	49	64	
25-34	42	45	8	20	40	66	
35 plus	28	37	7	21	28	54	
All ages	148	45	4	24	44	62	
Location ⁵							0.05
Mission	42	37	7	20	30	49	
Town	37	49	8	32	45	62	
Pay period							n.s.
Off pay week	48	41	6	22	40	55	
Pay week	74	49	6	26	52	67	
Females							
Age group							n.s.
18-24	55	38	6	24	32	53	
25-34	21	44	12	21	36	70	
35 plus	24	35	8	20	28	51	
All ages	100	39	4	23	32	55	
Location ⁵							n.s.
Mission	25	37	9	16	38	50	
Town	24	36	8	22	30	47	
Pay period							n.s.
Off pay week	51	36	6	18	31	48	
Pay week	37	43	7	24	42	60	

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Total number of respondents, excluding 'not stated'.

3. Two standard errors around the mean.

4. Brown-Mood non-parametric median statistical test was used to determine if the salt intake score had the same distribution across different groups or categories.

5. Mission refers to people living on or near the mission; while town refers to people living in town.

7.6 Summary

This chapter has highlighted three ways in which the dietary habits of Bourke Aborigines could possibly be putting them at risk, or at least at a greater risk than all Australians, of developing and dying from coronary heart disease. First, although the intakes of both total fat and saturated fatty acids by Bourke Aborigines and all Australians are comparable, Bourke Aborigines have lower total energy intakes and therefore derive a greater proportion of their total energy intake from fat. Furthermore, this proportion is well over the levels recommended by the World Health Organization, the National Academy of Sciences in the United States, and the Committee on Medical Aspects of Food Policy in Britain for populations with a high rate of coronary heart disease.

Second, Bourke Aborigines have mean daily intake levels of dietary fibre which are half of those observed for all Australians. As already stated, the intake of dietary fibre has been shown to be inversely related to the incidence of coronary heart disease (see section 7.4).

Third, while calcium intake seems to be inversely related to blood pressure, the mean daily intake of calcium by Bourke Aborigines is half of what is observed for all Australians. However, because of the inherent problems associated with the dietary recall method used in this study, all observations of mean daily intake levels should be viewed with appropriate caution, especially those relating to observed differences between Bourke Aborigines and all Australians as opposed to proportional differences.

An additional finding noted for interest's sake only is that while a positive relationship seems to exist between sodium intake and blood pressure, Bourke Aborigines had significantly higher mean sodium intake scores than another Australian population.

This chapter has also highlighted notable differences in dietary risk factors by location and pay period. Respondents surveyed during pay week as well as those living on or

near the mission tended to have higher mean daily intakes of fat and dietary fibre.

Additionally, mean daily macro-nutrient intakes tended to decrease with age.

CHAPTER EIGHT

CONCLUSION

'Health' to Aboriginal peoples is a matter of determining all aspects of their life, including control over their physical environment, of dignity, of community self-esteem, and of justice. It is not merely a matter of the provision of doctors, hospitals, medicines or the absence of disease and incapacity.

National Aboriginal Health Strategy Working Party (1989: ix)

8.1 Introduction

This thesis has attempted to evaluate whether Australian Aboriginal mortality patterns are different from those exhibited by indigenous groups in other countries or by the general Australian population; and whether the high rate of coronary heart disease among Australian Aborigines is attributable to the underlying prevalence of specific lifestyle (risk) factors. At a macro-level, I have compared the contemporary mortality patterns of and risk factor prevalence levels among Australian Aborigines and other indigenous groups. At a micro-level, I have made the same comparisons between Aborigines living in western New South Wales and other Australians; more specifically, I have explored the interrelationships between several behavioural and biological risk factors of coronary heart disease — including alcohol and tobacco consumption, blood pressure, body composition, serum cholesterol, diet, and physical activity — and the influence of demographic, environmental, and social-cultural conditions (risk markers) on these risk factors. Although in some respects I have compared "apples and oranges" (because of the incompatibility of data in so many cases), this has still been a worthwhile task because so little is known about the differences in the mortality patterns and risk factor prevalence levels between Aborigines and other Australian and indigenous populations.

8.1.1 Review of the evidence

This study has made four important points regarding the Australian Aboriginal mortality regime and the Bourke Aboriginal risk factor prevalence levels. The first point is that although Australian Aborigines and other indigenous groups have similar patterns of high adult mortality, the relative importance of some more notable causes of death is strikingly different. The rates of death from circulatory and respiratory system diseases are noticeably higher among Australian Aborigines than Canadian Indians or New Zealand Maoris; while the death rates from external causes are higher among Australian Aborigines and Canadian Indians than among New Zealand Maoris. For example, Aboriginal circulatory system disease death rates are approximately two to three times higher than those exhibited by Canadian Indians and one and a quarter to two times higher than those exhibited by New Zealand Maoris. Although a direct comparison cannot be made, American Indians probably have much lower circulatory system disease death rates than Australian Aborigines and Nauruans are likely to have similar rates.

What is surprising is that existing but rather incomplete information on risk factor prevalence levels does not highlight why Aborigines as compared to other indigenous groups have such high circulatory system disease death rates. If anything this information suggests that the levels of non-insulin dependent diabetes, hypertension, obesity, high cholesterol, and cigarette smoking are quite similar in all these populations; and that their dietary habits have undergone a corresponding shift away from traditional bush foods to prepackaged store-bought foods which are higher in salt, fat, and refined carbohydrates and lower in dietary fibre. However, chapter 2 does emphasize that certain risk factors, such as obesity, are more important than others, such as high cholesterol; and that other previously unrecognized risk factors, such as hyperinsulinaemia (a high insulin response), may be significant.

The second point is that Aborigines have much higher total and age-specific death rates than other Australians, especially during adulthood and most notably from circulatory

system diseases. As noted in chapter 4, the relative risk of death for Aboriginal males and females in western New South Wales is at least two times higher than that of their counterparts in the total New South Wales population, and up to three times higher in middle adulthood. Deaths from circulatory system diseases, especially from ischaemic heart disease and cerebrovascular disease, accounted for the greatest proportion of the difference — approximately 40 per cent of the excess risk. A similar pattern of mortality has also been noted for Bourke and Brewarrina Aborigines and non-Aborigines. In this case, Aborigines as compared with the total New South Wales population have exceptionally high death rates, while non-Aborigines have age- and cause-specific death rates very similar to those found among the total New South Wales population. This pattern of high adult Aboriginal mortality is by no means unique to western New South Wales, but has been observed again and again throughout Australia.

The third point is that Bourke Aborigines as compared with all Australians have much higher risk factor prevalence levels for a number of coronary heart disease risk factors. In terms of biological risk factors, they are more likely to be overweight or obese and to have diastolic hypertension. In terms of behavioural risk factors, they are more likely to drink heavily on a drinking day and to be current smokers, and are less likely to participate in vigorous exercise. In terms of dietary risk factors, they are more likely to derive a greater proportion of their total energy intake from fat and to have lower mean daily intakes of dietary fibre.

The fourth and final point is that Bourke Aboriginal risk factor prevalence levels tend to vary quite considerably by several risk markers — age, location, employment status, and pay period. All biological and behavioural risk factors vary with age. The most notable difference by age is in the proportion of respondents who are overweight or obese, which more than doubles between the age groups 18 to 24 years and 35 years and over — from 29 per cent to 74 per cent. With employment status, there is a tendency for respondents who are employed to smoke less and to participate in vigorous exercise more often than those who are unemployed. Additionally, there are several

notable variations in most behavioural risk factor prevalence levels by location (whether the respondent lives in town or on or near the mission) and pay period (a measure of income variability between the week when pension benefits were paid out and the alternate week when they were not).

Overall, these findings suggest that the mortality pattern of Australian Aborigines is experienced by no other population; and that lifestyle factors, which are influenced by often uncontrollable conditions, can at least partially account for the observed high rate of coronary heart disease.

8.1.2 Theoretical significance of the findings

As noted in chapter 1, the term lifestyle is defined here as a combination of behaviours, or ways of life, that each individual, rich or poor, acquires voluntarily or involuntarily. However, the lifestyle concept is not being used as a blanket explanation in which the individual is always to blame; rather, it is a means of stressing how great an impact demographic, environmental, and social-cultural conditions (risk markers) have on individual well-being and health-related behaviour.

In regard to demographic variables, this thesis, like other recently conducted Aboriginal mortality studies, has highlighted the fact that Aborigines as compared with the general Australian population are not only more at risk of dying from circulatory system diseases, but also they are dying from these diseases at earlier ages. This difference in relative risk is particularly apparent during middle adulthood — between the ages of 25 and 64 years.

Environmental and social-cultural variables are also important because they demonstrate that Aborigines as a group are not homogeneous. As noted in chapter 4, there are notable differences in Aboriginal mortality levels in western New South Wales by community. Community mortality levels can be categorized as very high, moderately high, and low and can be partly explained by a number of social-cultural

conditions. Most notably, low mortality communities have higher levels of Aboriginal employment, a smaller proportion of persons of Aboriginal descent, and a greater proportion of Aboriginal households with less than five persons. As already stated, a number of variables, or risk markers (including age, location, employment status, and pay period), have been shown to influence Bourke Aboriginal biological and behavioural risk factor prevalence levels.

Risk markers are unlikely to be the only source of variation in the prevalence of biological and behavioural risk factors, and therefore in mortality levels. Psycho-social factors, such as the coping behaviour of individuals, may also contribute to the variation (see World Health Organization, Health Education Unit, 1986). To a large extent, an individual's ability to take responsibility for his or her own life is constrained by environmental and social-cultural conditions which lead the individual to think that there is little he or she can do that will affect or improve the social situation. In order to cope with these situational constraints, individuals can try to: alter their perception of the problem; control the stress to which the problem gives rise; and/or change the situation that has created the problem. The approach an individual takes very much depends on his or her situation and on the available social and economic resources — for example, in the case of Aborigines, whether they are employed, live in a particular part of town, have a secondary education, or live in a household that is not overcrowded. Health risk behaviour, putting oneself at risk of ill health, may constitute a way in which an individual can deal with conflicts that arise in everyday life that cannot be controlled and a means of regaining the physical and psychological ability to cope with his or her situation. As the World Health Organization, Health Education Unit, (1986: 120) notes:

Often such behaviour patterns are highly acceptable in our culture and are promoted by mass media, advertising and tradition: 'You need a drink', 'Take a pill', 'Have a cigarette', etc. are phrases suggesting that escape from conflict situations by risk behaviour is a matter of conformity and therefore socially acceptable.

On the other hand, positive health behaviour may be an outcome of an individual's ability take control and responsibility for his or her own health and situation.

8.1.3 Wider theoretical implications

This type of research is important not only for its implications regarding Aboriginal lifestyle and health, but also for its possible significance in current theoretical perspectives about epidemiological transition. Formulated by Omran (1971), the epidemiological transition theory posits that over time disease patterns shift in such a way that infectious and parasitic diseases are gradually, but not totally, displaced by degenerative diseases and accidental and violent events as the primary causes of death. This theory identifies three major successive stages of mortality decline: high and fluctuating mortality; progressive declines in mortality predominantly at lower ages; and stable mortality at a lower level (see Table 8.1). With recent mortality declines in the United States, Crimmins (1981) has proposed a fourth stage of transition which is characterized by mortality declines in middle and older age groups as well as among infants, by significant declines in death rates from degenerative diseases, and by decreases in existing sex differentials in mortality. A fourth stage has also been proposed by Rogers and Hackenburg (1988), called the hybristic stage, which is increasingly influenced by individual behaviours and lifestyles. The hybristic stage accounts for recent declines in mortality from degenerative diseases as well as rapid increases in some societies in deaths from social pathologies — accidents, violence, alcoholism, and cirrhosis of the liver.

Demographers and epidemiologists generally assume that with improvements in health care service and delivery and changes in lifestyle, the mortality pattern of a population shifts from one extensively based on infectious diseases to one predominantly based on degenerative diseases and social pathologies. With this shift come extensive improvements in life expectancy and age-specific mortality at all ages. However, this thesis along with many other studies has shown that the mortality transition of

Australian Aborigines has stalled — life expectancy has remained low in the face of an ever increasing proportion of deaths from degenerative diseases, especially from circulatory system diseases. As already stated, no other population, with the possible exception of some Pacific Island populations like the Nauruans, has such low life expectancies and high mortality rates, especially from circulatory system diseases. Although Kunitz (1990a) suggests that this pattern remains a puzzle to be solved, I would argue that lifestyle factors and the rather unique environmental and social-cultural situation of Aborigines play an important role in determining the present mortality regime.

Table 8.1 The epidemiological transition: an expanded model¹.

Stage	Indicative mortality levels			Mortality differentials		Main causes of death	Major Initiatives to reduce mortality
	IMR	E ₀	E ₅₀	Sex	Socio-economic		
1. High and fluctuating mortality	200	40	10	Low	High	Infectious diseases associated with epidemics, famine, and pestilence	Famine relief, improved nutrition, sanitation, and water control
2. Progressive declines in mortality predominantly in younger ages	100	50	24	Increasing	High	Infectious diseases, but epidemics less frequent and less catastrophic	Public health and sanitation, improved nutrition, and hospital development
3. Stable mortality at a low level	12	70	25	High	Decreasing	Degenerative diseases, such as cancer and heart disease, and accidents	Therapy developments and medical breakthroughs
4. Further declines in mortality especially at older ages	9	75	30	Decreasing	Decreasing	Degenerative diseases and accidents	Lifestyle changes (diet, exercise, etc.) and medical breakthroughs

Note: IMR = infant mortality rate (infant deaths per 1,000 births).

E₀ = life expectancy at birth.

E₅₀ = life expectancy at age 50.

1. Sources: Hugo (1986: 23, Table 2.2) and Omran (1971: Table 4).

8.2 Limitations to the study and areas for future research

This study was hampered by three major limitations. First, comparisons between the mortality patterns of Aboriginal and other indigenous populations were constrained by the quality of available sources of mortality data. As noted in chapter 2, the data that were available reflect the idiosyncratic manner in which each country classifies deaths and persons at risk of dying. Often, the way in which deaths are reported is different from the way the population at risk is defined. Furthermore, in some countries mortality data only refer to a particular subgroup and not to the entire indigenous population. Although the effects of these peculiarities are impossible to measure, they are bound to play a role in distorting the reported differences in mortality patterns.

Second, the non-random sampling approach taken in the risk factor prevalence survey is a major source of potential bias, as the background characteristics and responses of those who participated may not be truly representative of the total Bourke Aboriginal population. This is especially so for biological risk factors which were based on a small subsample of respondents.

Third, there were several problems associated with the collection of dietary recall data. In particular, the collection of data was hampered by the inability of respondents to remember what they ate and to estimate their portion sizes. Although both potential sources of bias were dealt with in the study, they still may have some unknown effect on the described dietary habits and nutrient intake patterns of Bourke Aborigines.

In terms of future research, this study has highlighted the need for more Aboriginal mortality data. In particular, there is need for more regional studies, like the one conducted in western New South Wales (Gray and Hogg, 1989), and more longitudinal studies, like the one conducted by Khalidi (1989) in central Australia. These types of studies are crucial to a better understanding of Aboriginal health because they will be important aids in assessing regional and community health inequalities and in examining changes in mortality trends over time.

8.3 Practical and policy implications

Since policy-making is primarily an exercise in the selection of values to guide future behaviour, it follows in the case of Aboriginal health that Aborigines must be active participants in developing practical solutions to current problems of ill health. To a large extent the National Aboriginal Health Strategy Working Party (1989) has done just that by establishing a number of relevant long and short term policy strategies for improving Aboriginal health and life expectancy. They believe that if these policies are going to succeed, community controlled health services, such as the Aboriginal Health Service in Bourke, must be given the overall responsibility for implementing and developing preventative health programs according to local priorities.

In regards to circulatory system disease, the National Aboriginal Health Strategy Working Party (1989: 132-137) recommends that the difference in Australian and Aboriginal mortality rates from this cause of death should be reduced by 50 per cent within 10 years and by 100 per cent within 25 years¹. As noted below, this reduction in the mortality rate will be achieved by developing and implementing a number of community-level preventive health care programs concerning:

Hypertension control

Objective:

To detect all those whose blood pressure is elevated and to reduce and maintain their blood pressure at normal levels.

Strategies:

Provide the opportunity for and encourage all Aboriginal adults to have their blood pressure measured at least once a year;

provide, for those whose blood pressure is elevated, advice, education, and counselling on the health risks and mortality associated with hypertension;

1. These 10 and 25 year estimates are based on achievable targets once a full range of community services have been provided to improve the quality and availability of primary health care. The Working Party is concerned that any attempt to set targets before a full range of services is provided will inevitably lead to limited success in the achievement of these targets.

provide advice, support, and encouragement to reduce salt, saturated fat, refined sugar, and alcohol intake, to achieve weight reduction, to have regular exercise and to stop smoking;

provide appropriate medical treatment to reduce blood pressure levels to normal; and

provide facilities for regular monitoring and life-long follow up to ensure that hypertension is controlled.

Smoking prevention and reduction

Objectives:

To encourage non-smokers, young people in particular, to remain non-smokers; and to encourage, motivate, and help those who smoke, to stop.

Strategies:

Educate children about the benefits of not smoking, using accepted Aboriginal role models;

provide for those who are smoking, specific advice, support, and encouragement to stop (including self-help groups), and develop culturally appropriate programs;

encourage smoke free zones in Aboriginal services; and

promote anti-smoking campaigns amongst Aboriginal people.

Exercise promotion

Objective:

To encourage people to take sensible regular exercise as a normal part of life.

Strategies:

Encourage regular exercise as a habit in conjunction with weight reduction programs. An exercise program should ideally start in early childhood in the school;

ensure that an adequate range of sporting and recreational activities is available in all communities; and

encourage and promote traditional food gathering and hunting activities, walks to traditional sites, and ceremonial dancing.

Healthy nutrition

Objectives:

To promote bush foods; to encourage fitness and discourage obesity; to promote a diet which is low in fat (especially saturated fat), refined sugars and salt, but which is high in fibre; and to enable better control of diabetes when it is present.

Strategies:

Provide facilities for the recognition and assessment of obesity to all Aboriginal adults at least every five years from the age of 20 onwards;

provide culturally appropriate, realistic, and ongoing advice about affordable, acceptable, and appropriate nutrition, and counselling and assistance with weight reduction to all persons found to be obese;

encourage and support the use of traditional foods by Aboriginal people;

encourage an understanding amongst non-Aboriginal people of the benefits of traditional foods and hunting and gathering activities;

investigate and take action to protect traditional foodstuff/sources from non-Aboriginal exploitation;

research ways of increasing traditional foodstuffs — for example, through farming and land management techniques;

promote legislation which will enable Aboriginal people to hunt traditional food in any area without prosecution;

ensure that an adequate range of fresh foods which are low in fat (especially saturated fats), refined sugars and salt, but high in fibre is available to all Aboriginal people at prices comparable to those in metropolitan areas; and

provide stress management programs.

Given that circulatory system disease is seen as a long-term health problem that can only be improved through community-implemented programs which try to reduce risk factor prevalence levels as well as improve community social, emotional, and cultural well-being, these policy statements are realistic. As Ian Anderson (1988: 139) states:

... [I]mproved health will result from Aboriginal people shaping their future. ... No-one outside the Aboriginal community can do this for them. Others can undermine, frustrate and ignore problems — but they cannot make Aboriginal people healthy. That will be fundamentally up to the Aboriginal community and in this there is hope.

Overall, Aborigines believe that health and well-being depends very much on their ability to independently chart their future, without outside intervention or interference.

As Reid and Kerr (1983: 350) state:

To some, such statements, which dominate the debate in Aboriginal health today, may seem like radical rhetoric. But in reality, Aborigines are asking for no more than the wider Australian population takes for granted — economic security, effective political representation, freedom from domination by people of another country, colour, or culture, and the dignity generated by influence and respect.

APPENDIX ONE
ADDITIONAL TABLES FOR CHAPTERS TWO AND FOUR

Table A1.1 Male cause-specific mortality rates (CSMR)¹, relative risk², and proportion of excess risk³ for Aborigines from the Northern Territory⁴, 1979-1983, as compared with the Australian population⁵, 1986.

Cause of death	Aborigines Deaths	Aborigines CSMR	Australian CSMR	Relative risk range	Proportion of excess risk
All causes	951	28.4	7.8	(3.4 - 3.9)	100
Infectious and parasitic	50	1.1	0.0	(20.0 - 35.6)	5
Malignant neoplasms	62	2.2	2.0	(0.9 - 1.5)	1
Endocrine, nutritional	22	0.7	0.2	(3.1 - 7.5)	3
Blood and blood-forming organs	4	0.1	0.0	-	1
Mental disorders	11	0.3	0.1	(1.7 - 6.1)	1
Nervous system	24	0.5	0.1	(2.9 - 6.6)	2
Circulatory system	248	8.9	3.5	(2.3 - 2.9)	26
Respiratory system	183	6.2	0.6	(8.5 - 11.5)	27
Digestive system	28	0.8	0.3	(2.1 - 4.5)	2
Genito-urinary system	27	1.0	0.1	(7.0 - 15.6)	4
Skin and subcutaneous tissue	1	0.0	0.0	-	0
Musculoskeletal system	4	0.1	0.0	-	1
Congenital anomalies	21	0.1	0.1	(0.8 - 1.9)	0
Perinatal causes	43	0.0	0.1	(0.3 - 0.6)	0
Signs, symptoms and ill-defined conditions	58	2.1	0.1	(28.8 - 49.1)	10
Accidents, poisonings and violence	165	4.3	0.68	(5.4 - 10.6)	17

1. Rates are deaths per 1,000 population per year. Cause-specific rates were directly standardized to the 1986 Australian age structure.

2. A ratio of the directly standardized Aboriginal rate to the Australian rate. The relative risk range represents confidence intervals, at a 95 percent level of confidence, around each ratio. A relative risk range was calculated for cause-specific mortality rates based on five or more deaths.

3. Excess risk was calculated by subtracting cause-specific death rates for the Australian population from directly standardized Aboriginal rates. The proportion of excess risk was then derived by producing a ratio of the Aboriginal cause-specific excess risk to the Aboriginal excess risk for all causes. Percentages may not add up because of rounding.

4. Source: Plant (1988).

5. Source: Australian Bureau of Statistics (1988e).

Table A1.2 Male cause-specific mortality rates (CSMR)¹, relative risk², and proportion of excess risk³ for Aborigines from Western Australia⁴, 1983, as compared with the Australian population⁵, 1986.

Cause of death	Aborigines Deaths	Aborigines CSMR	Australian CSMR	Relative risk range	Proportion of excess risk
All causes	200	21.0	7.8	(2.4 - 3.1)	100
Infectious and parasitic	6	0.3	0.0	(3.3 - 19.8)	2
Malignant neoplasms	15	1.9	2.0	(0.6 - 1.7)	-1
Endocrine, nutritional	2	0.3	0.2	-	1
Blood and blood-forming organs	0	0.0	0.0	-	0
Mental disorders	4	0.6	0.1	-	4
Nervous system	11	1.2	0.1	(5.8 - 20.7)	8
Circulatory system	56	6.7	3.5	(1.5 - 2.5)	24
Respiratory system	30	3.7	0.6	(4.1 - 8.7)	23
Digestive system	9	1.0	0.3	(1.9 - 8.1)	5
Genito-urinary system	12	1.4	0.1	(8.4 - 28.5)	10
Skin and subcutaneous tissue	0	0.0	0.0	-	0
Musculoskeletal system	2	0.3	0.0	-	2
Congenital anomalies	0	0.0	0.1	-	0
Perinatal causes	16	0.0	0.1	(0.3 - 0.8)	0
Signs, symptoms and ill-defined conditions	3	0.4	0.1	(2.6 - 36.7)	3
Accidents, poisonings and violence	34	3.3	0.68	(3.5 - 7.1)	20

1. Rates are deaths per 1,000 population per year. Cause-specific rates were directly standardized to the 1986 Australian age structure.

2. A ratio of the directly standardized Aboriginal rate to the Australian rate. The relative risk range represents confidence intervals, at a 95 percent level of confidence, around each ratio. A relative risk range was calculated for cause-specific mortality rates based on five or more deaths.

3. Excess risk was calculated by subtracting cause-specific death rates for the Australian population from directly standardized Aboriginal rates. The proportion of excess risk was then derived by producing a ratio of the Aboriginal cause-specific excess risk to the Aboriginal excess risk for all causes. Percentages may not add up because of rounding.

4. Source: Health Department of Western Australia (1985).

5. Source: Australian Bureau of Statistics (1988e).

Table A1.3 Male cause-specific mortality rates (CSMR)¹, relative risk², and proportion of excess risk³ for Aborigines from western New South Wales⁴, 1984-1987, as compared with the Australian population⁵, 1986.

Cause of death	Aborigines Deaths	Aborigines CSMR	Australian CSMR	Relative risk range	Proportion of excess risk
All causes	205	23.3	7.8	(2.6 - 3.5)	100
Infectious and parasitic	4	0.2	0.0	-	1
Malignant neoplasms	21	3.0	2.0	(1.0 - 2.5)	7
Endocrine, nutritional	2	0.2	0.2	-	1
Blood and blood-forming organs	0	0.0	0.0	-	0
Mental disorders	3	0.3	0.1	-	2
Nervous system	2	0.2	0.1	-	0
Circulatory system	84	12.6	3.5	(2.9 - 4.5)	58
Respiratory system	10	1.3	0.6	(1.1 - 4.3)	4
Digestive system	17	1.7	0.3	(4.2 - 11.5)	10
Genito-urinary system	3	0.5	0.1	-	3
Skin and subcutaneous tissue	0	0.0	0.0	-	0
Musculoskeletal system	0	0.0	0.0	-	0
Congenital anomalies	5	0.2	0.1	(1.1 - 8.2)	1
Perinatal causes	6	0.2	0.1	(1.2 - 7.4)	1
Signs, symptoms and ill-defined conditions	18	1.0	0.1	(10.9 - 29.0)	6
Accidents, poisonings and violence	30	2.0	0.68	(2.1 - 4.4)	9

1. Rates are deaths per 1,000 population per year. Cause-specific rates were directly standardized to the 1986 Australian age structure.

2. A ratio of the directly standardized Aboriginal rate to the Australian rate. The relative risk range represents confidence intervals, at a 95 percent level of confidence, around each ratio. A relative risk range was calculated for cause-specific mortality rates based on five or more deaths.

3. Excess risk was calculated by subtracting cause-specific death rates for the Australian population from directly standardized Aboriginal rates. The proportion of excess risk was then derived by producing a ratio of the Aboriginal cause-specific excess risk to the Aboriginal excess risk for all causes. Percentages may not add up because of rounding.

4. Source: Gray and Hogg (1989).

5. Source: Australian Bureau of Statistics (1988e).

Table A1.4 Male cause-specific mortality rates (CSMR)¹, relative risk², and proportion of excess risk³ for Canadian Indians⁴, 1980-1986, as compared with the Australian population⁵, 1986.

Cause of death	Canadian Indian Deaths	Canadian Indian CSMR	Australian CSMR	Relative risk range	Proportion of excess risk
All causes	6,926	11.2	7.8	(1.4 - 1.5)	100
Infectious and parasitic	112	0.2	0.0	(3.8 - 5.6)	4
Malignant neoplasms	549	1.2	2.0	(0.5 - 0.6)	-24
Endocrine, nutritional	103	0.2	0.2	(1.0 - 1.5)	1
Blood and blood-forming organs	20	0.0	0.0	(1.0 - 2.5)	0
Mental disorders	88	0.1	0.1	(0.9 - 1.2)	1
Nervous system	84	0.1	0.1	(0.9 - 1.3)	0
Circulatory system	1,656	3.6	3.5	(1.0 - 1.1)	2
Respiratory system	525	1.0	0.6	(1.5 - 1.8)	12
Digestive system	287	0.5	0.3	(1.8 - 2.3)	8
Genito-urinary system	80	0.2	0.1	(1.4 - 2.2)	2
Skin and subcutaneous tissue	2	0.0	0.0	-	0
Musculoskeletal system	11	0.0	0.0	(0.5 - 1.9)	0
Congenital anomalies	121	0.1	0.1	(1.8 - 2.6)	2
Perinatal causes	180	0.2	0.1	(2.5 - 3.4)	4
Signs, symptoms and ill-defined conditions	281	0.4	0.1	(6.4 - 8.1)	10
Accidents, poisonings and violence	2,827	3.3	0.68	(4.6 - 5.0)	77

1. Rates are deaths per 1,000 population per year. Canadian Indian cause-specific rates were directly standardized to the 1986 Australian age structure.

2. A ratio of the directly standardized Canadian Indian rate to the Australian rate. The relative risk range represents confidence intervals, at a 95 percent level of confidence, around each ratio. A relative risk range was calculated for cause-specific mortality rates based on five or more deaths.

3. Excess risk was calculated by subtracting cause-specific death rates for the Australian population from directly standardized Canadian Indian rates. The proportion of excess risk was then derived by producing a ratio of the Canadian Indian cause-specific excess risk to the Canadian Indian excess risk for all causes. Percentages may not add up because of rounding.

4. Source: unpublished data from Health and Welfare Canada.

5. Source: Australian Bureau of Statistics (1988e).

Table A1.5 Male cause-specific mortality rates (CSMR)¹, relative risk², and proportion of excess risk³ for New Zealand Maoris, 1980-1986⁴, as compared with the Australian population⁵, 1986.

Cause of death	Deaths	Maori CSMR	Australian CSMR	Relative risk range	Proportion of excess risk
All causes	5,502	12.3	7.8	(1.5 - 1.6)	100
Infectious and parasitic	82	0.2	0.0	(2.9 - 4.6)	2
Malignant neoplasms	1,242	3.2	2.0	(1.5 - 1.7)	26
Endocrine, nutritional	232	0.6	0.2	(3.2 - 4.2)	9
Blood and blood-forming organs	9	0.0	0.0	(0.4 - 1.5)	0
Mental disorders	27	0.1	0.1	(0.3 - 0.7)	-1
Nervous system	48	0.1	0.1	(0.4 - 0.8)	-1
Circulatory system	1,926	5.1	3.5	(1.2 - 1.7)	35
Respiratory system	567	1.5	0.6	(2.1 - 2.5)	18
Digestive system	134	0.3	0.3	(1.1 - 1.5)	2
Genito-urinary system	71	0.2	0.1	(1.6 - 2.7)	2
Skin and subcutaneous tissue	5	0.0	0.0	-	0
Musculoskeletal system	10	0.0	0.0	(0.7 - 2.7)	0
Congenital anomalies	78	0.1	0.1	(0.8 - 1.3)	0
Perinatal causes	89	0.1	0.1	(0.7 - 1.1)	0
Signs, symptoms and ill-defined conditions	184	0.1	0.1	(2.1 - 2.9)	2
Accidents, poisonings and violence	798	0.9	0.7	(1.3 - 1.5)	6

1. Rates are deaths per 1,000 population per year. New Zealand Maori cause-specific rates were directly standardized to the 1986 Australian age structure.

2. A ratio of the directly standardized New Zealand Maori rate to the Australian rate. The relative risk range represents confidence intervals, at a 95 percent level of confidence, around each ratio. A relative risk range was calculated for cause-specific mortality rates based on five or more deaths.

3. Excess risk was calculated by subtracting cause-specific death rates for the Australian population from directly standardized New Zealand Maori rates. The proportion of excess risk was then derived by producing a ratio of the New Zealand Maori cause-specific excess risk to the New Zealand Maori excess risk for all causes. Percentages may not add up because of rounding.

4. Source: unpublished data from National Health Statistics Centre.

5. Source: Australian Bureau of Statistics (1988e).

Table A1.6 Female cause-specific mortality rates (CSMR)¹, relative risk², and proportion of excess risk³ for Aborigines from the Northern Territory⁴, 1979-1983, as compared with the Australian population⁵, 1986.

Cause of death	Deaths	Aborigines CSMR	Australian CSMR	Relative risk range	Proportion of excess risk
All causes	691	22.4	6.6	(3.2 - 3.7)	100
Infectious and parasitic	51	1.2	0.0	(28.9 - 51.1)	8
Malignant neoplasms	55	2.0	1.5	(1.0 - 1.7)	3
Endocrine, nutritional	36	1.4	0.2	(6.1 - 12.2)	8
Blood and blood-forming organs	14	0.5	0.0	(10.1 - 31.0)	3
Mental disorders	6	0.2	0.1	(1.0 - 5.9)	1
Nervous system	18	0.3	0.1	(1.8 - 4.9)	1
Circulatory system	165	6.4	3.4	(1.6 - 2.2)	19
Respiratory system	135	5.0	0.4	(11.7 - 16.4)	29
Digestive system	23	0.8	0.2	(2.1 - 5.1)	3
Genito-urinary system	29	1.1	0.1	(6.1 - 13.0)	6
Pregnancy, childbirth	7	0.1	0.0	(32.8 - 168.3)	1
Skin and subcutaneous tissue	0	0.1	0.0	-	1
Musculoskeletal system	3	0.1	0.0	-	0
Congenital anomalies	19	0.2	0.1	(3.0 - 7.9)	1
Perinatal causes	17	0.2	0.0	(2.2 - 6.2)	1
Signs, symptoms and ill-defined conditions	40	1.4	0.0	(24.6 - 47.0)	9
Accidents, poisonings and violence	73	1.5	0.3	(4.1 - 6.6)	8

1. Rates are deaths per 1,000 population per year. Cause-specific rates were directly standardized to the 1986 Australian age structure.

2. A ratio of the directly standardized Aboriginal rate to the Australian rate. The relative risk range represents confidence intervals, at a 95 percent level of confidence, around each ratio. A relative risk range was calculated for cause-specific mortality rates based on five or more deaths.

3. Excess risk was calculated by subtracting cause-specific death rates for the Australian population from directly standardized Aboriginal rates. The proportion of excess risk was then derived by producing a ratio of the Aboriginal cause-specific excess risk to the Aboriginal excess risk for all causes. Percentages may not add up because of rounding.

4. Source: Plant (1988).

5. Source: Australian Bureau of Statistics (1988e).

Table A1.7 Female cause-specific mortality rates (CSMR)¹, relative risk², and proportion of excess risk³ for Aborigines from Western Australia⁴, 1983, as compared with the Australian population⁵, 1986.

Cause of death	Aborigines Deaths	Aborigines CSMR	Australian CSMR	Relative risk range	Proportion of excess risk
All causes	123	15.3	6.6	(1.9 - 2.8)	100
Infectious and parasitic	4	0.2	0.0	-	2
Malignant neoplasms	15	2.3	1.5	(0.9 - 2.7)	9
Endocrine, nutritional	10	1.5	0.2	(5.0 - 19.1)	15
Blood and blood-forming organs	0	0.0	0.0	-	0
Mental disorders	3	0.4	0.1	-	3
Nervous system	1	0.0	0.1	-	-1
Circulatory system	37	5.5	3.4	(1.2 - 2.3)	24
Respiratory system	11	1.6	0.4	(2.4 - 8.8)	14
Digestive system	4	0.4	0.2	-	2
Genito-urinary system	9	1.5	0.1	(6.5 - 26.8)	16
Pregnancy, childbirth	0	0.0	0.0	-	0
Skin and subcutaneous tissue	0	0.0	0.0	-	0
Musculoskeletal system	0	0.0	0.0	-	-1
Congenital anomalies	6	0.2	0.1	(1.7 - 9.9)	2
Perinatal causes	0	0.0	0.0	-	-1
Signs, symptoms and ill-defined conditions	3	0.4	0.0	-	4
Accidents, poisonings and violence	20	1.4	0.3	(3.0 - 7.5)	12

1. Rates are deaths per 1,000 population per year. Cause-specific rates were directly standardized to the 1986 Australian age structure.

2. A ratio of the directly standardized Aboriginal rate to the Australian rate. The relative risk range represents confidence intervals, at a 95 percent level of confidence, around each ratio. A relative risk range was calculated for cause-specific mortality rates based on five or more deaths.

3. Excess risk was calculated by subtracting cause-specific death rates for the Australian population from directly standardized Aboriginal rates. The proportion of excess risk was then derived by producing a ratio of the Aboriginal cause-specific excess risk to the Aboriginal excess risk for all causes. Percentages may not add up because of rounding.

4. Source: Health Department of Western Australia (1985).

5. Source: Australian Bureau of Statistics (1988e).

Table A1.8 Female cause-specific mortality rates (CSMR)¹, relative risk², and proportion of excess risk³ for Aborigines from western New South Wales⁴, 1984-1987, as compared with the Australian population⁵, 1986.

Cause of death	Aborigines Deaths	Aborigines CSMR	Australian CSMR	Relative risk range	Proportion of excess risk
All causes	110	14.6	6.6	(1.8 - 2.7)	100
Infectious and parasitic	6	0.2	0.0	(3.2 - 18.8)	2
Malignant neoplasms	10	1.8	1.5	(0.6 - 2.4)	3
Endocrine, nutritional	8	1.4	0.2	(4.2 - 19.2)	15
Blood and blood-forming organs	0	0.0	0.0	-	0
Mental disorders	2	0.1	0.1	-	0
Nervous system	3	0.1	0.1	-	0
Circulatory system	41	7.2	3.4	(1.6 - 2.9)	47
Respiratory system	9	1.5	0.4	(2.2 - 9.0)	14
Digestive system	4	0.5	0.2	-	3
Genito-urinary system	5	0.6	0.1	(2.0 - 14.3)	6
Pregnancy, childbirth	0	0.0	0.0	-	0
Skin and subcutaneous tissue	0	0.0	0.0	-	0
Musculoskeletal system	0	0.0	0.0	-	-1
Congenital anomalies	2	0.0	0.1	-	0
Perinatal causes	2	0.0	0.0	-	0
Signs, symptoms and ill-defined conditions	3	0.2	0.0	-	2
Accidents, poisonings and violence	15	1.0	0.3	(2.0 - 6.0)	9

1. Rates are deaths per 1,000 population per year. Cause-specific rates were directly standardized to the 1986 Australian age structure.

2. A ratio of the directly standardized Aboriginal rate to the Australian rate. The relative risk range represents confidence intervals, at a 95 percent level of confidence, around each ratio. A relative risk range was calculated for cause-specific mortality rates based on five or more deaths.

3. Excess risk was calculated by subtracting cause-specific death rates for the Australian population from directly standardized Aboriginal rates. The proportion of excess risk was then derived by producing a ratio of the Aboriginal cause-specific excess risk to the Aboriginal excess risk for all causes. Percentages may not add up because of rounding.

4. Source: Gray and Hogg (1989).

5. Source: Australian Bureau of Statistics (1988e).

Table A1.9 Female cause-specific mortality rates (CSMR)¹, relative risk², and proportion of excess risk³ for Canadian Indians⁴, 1980-1986, as compared with the Australian population⁵, 1986.

Cause of death	Canadian Indian Deaths	Canadian Indian CSMR	Australian CSMR	Relative risk range	Proportion of excess risk
All causes	4,211	8.8	6.6	(1.3 - 1.4)	100
Infectious and parasitic	102	0.2	0.0	(5.3 - 8.0)	8
Malignant neoplasms	534	1.4	1.5	(0.8 - 1.0)	-9
Endocrine, nutritional	122	0.3	0.2	(1.6 - 2.3)	6
Blood and blood-forming organs	11	0.0	0.0	(0.5 - 1.8)	0
Mental disorders	60	0.1	0.1	(0.8 - 1.4)	0
Nervous system	47	0.1	0.1	(0.5 - 0.9)	-2
Circulatory system	1,103	3.1	3.4	(0.9 - 1.0)	-14
Respiratory system	360	0.9	0.4	(2.3 - 2.7)	24
Digestive system	280	0.6	0.2	(2.2 - 3.5)	16
Genito-urinary system	84	0.2	0.1	(1.5 - 2.3)	5
Pregnancy, childbirth	3	0.0	0.0	-	0
Skin and subcutaneous tissue	7	0.0	0.0	(0.7 - 3.4)	0
Musculoskeletal system	16	0.0	0.0	(0.5 - 1.4)	0
Congenital anomalies	90	0.1	0.1	(1.4 - 2.1)	2
Perinatal causes	113	0.1	0.0	(2.1 - 3.1)	3
Signs, symptoms and ill-defined conditions	216	0.4	0.0	(7.6 - 13.1)	15
Accidents, poisonings and violence	1,063	1.3	0.3	(4.1 - 4.6)	45

1. Rates are deaths per 1,000 population per year. Canadian Indian cause-specific rates were directly standardized to the 1986 Australian age structure.

2. A ratio of the directly standardized Canadian Indian rate to the Australian rate. The relative risk range represents confidence intervals, at a 95 percent level of confidence, around each ratio. A relative risk range was calculated for cause-specific mortality rates based on five or more deaths.

3. Excess risk was calculated by subtracting cause-specific death rates for the Australian population from directly standardized Canadian Indian rates. The proportion of excess risk was then derived by producing a ratio of the Canadian Indian cause-specific excess risk to the Canadian Indian excess risk for all causes. Percentages may not add up because of rounding.

4. Source: unpublished data from Health and Welfare Canada.

5. Source: Australian Bureau of Statistics (1988e).

Table A1.10 Female cause-specific mortality rates (CSMR)¹, relative risk², and proportion of excess risk³ for New Zealand Maoris⁴, 1980-1986, as compared with the Australian population⁵, 1986.

Cause of death	Deaths	Maori CSMR	Australian CSMR	Relative risk range	Proportion of excess risk
All causes	4,045	10.3	6.6	(1.5 - 1.6)	100
Infectious and parasitic	59	0.2	0.0	(4.0 - 6.7)	4
Malignant neoplasms	1,083	2.7	1.5	(1.7 - 1.9)	31
Endocrine, nutritional	205	0.6	0.2	(3.1 - 4.1)	11
Blood and blood-forming organs	4	0.0	0.0	-	0
Mental disorders	15	0.0	0.1	(0.3 - 0.8)	-2
Nervous system	49	0.1	0.1	(0.5 - 0.9)	-1
Circulatory system	1,465	4.6	3.4	(1.3 - 1.4)	31
Respiratory system	482	1.3	0.4	(3.3 - 3.9)	25
Digestive system	54	0.2	0.2	(0.5 - 0.9)	-2
Genito-urinary system	66	0.2	0.1	(1.2 - 2.1)	2
Pregnancy, childbirth	5	0.0	0.0	-	0
Skin and subcutaneous tissue	5	0.0	0.0	-	0
Musculoskeletal system	8	0.0	0.0	(0.2 - 1.0)	-1
Congenital anomalies	61	0.0	0.1	(0.7 - 1.1)	0
Perinatal causes	85	0.1	0.0	(1.0 - 1.6)	0
Signs, symptoms and ill-defined conditions	141	0.1	0.0	(1.9 - 2.7)	1
Accidents, poisonings and violence	258	0.3	0.3	(0.9 - 1.1)	0

1. Rates are deaths per 1,000 population per year. New Zealand Maori cause-specific rates were directly standardized to the 1986 Australian age structure.

2. A ratio of the directly standardized New Zealand Maori rate to the Australian rate. The relative risk range represents confidence intervals, at a 95 percent level of confidence, around each ratio. A relative risk range was calculated for cause-specific mortality rates based on five or more deaths.

3. Excess risk was calculated by subtracting cause-specific death rates for the Australian population from directly standardized New Zealand Maori rates. The proportion of excess risk was then derived by producing a ratio of the New Zealand Maori cause-specific excess risk to the New Zealand Maori excess risk for all causes. Percentages may not add up because of rounding.

4. Source: unpublished data from National Health Statistics Centre.

5. Source: Australian Bureau of Statistics (1988e).

Table A1.11 Sex-specific abridged life tables for Aborigines living in nine western New South Wales communities¹, 1980-1983.

x	n	P_x	D_x	${}_nM_x$	${}_nq_x$	l_x	e^o_x	S.E.
Males								
0	1	94	12	0.0320	0.0318	1.000	58	1.4
1	4	389	0	0.0000	0.0000	0.968	59	1.4
5	10	865	2	0.0006	0.0058	0.968	55	1.4
15	10	728	7	0.0024	0.0238	0.963	45	1.3
25	10	436	12	0.0069	0.0665	0.940	36	1.3
35	10	300	14	0.0117	0.1102	0.877	28	1.3
45	10	244	19	0.0195	0.1774	0.780	21	1.1
55	10	112	22	0.0491	0.3942	0.642	15	1.1
65	+	96	34	0.0885	1.0000	0.389	11	-
Females								
0	1	123	10	0.0203	0.0203	1.000	67	1.5
1	4	336	2	0.0015	0.0059	0.980	67	1.5
5	10	843	1	0.0003	0.0030	0.974	64	1.4
15	10	775	7	0.0023	0.0223	0.971	54	1.4
25	10	469	6	0.0032	0.0315	0.949	45	1.4
35	10	305	13	0.0107	0.1012	0.919	36	1.4
45	10	223	5	0.0056	0.0545	0.826	30	1.1
55	10	127	9	0.0177	0.1628	0.781	21	1.0
65	+	100	28	0.0700	1.0000	0.654	14	-

1. Based on mortality data for nine western New South Wales communities (see section 4.2 in chapter 4). For each age interval x this table yields information on: the duration of the interval, n ; the population at risk, P_x ; the number of deaths, D_x ; the age-specific death rate, ${}_nM_x$; the probability of dying, ${}_nq_x$; the proportion of people who survived, l_x ; the life expectancy at age x , e^o_x ; and the standard error around each e^o_x value for a closed age interval, S.E.

Table A1.12 Sex-specific abridged life tables for Aborigines living in nine western New South Wales communities¹, 1984-1987.

x	n	P_x	D_x	${}_nM_x$	${}_nq_x$	l_x	e_x^o	S.E.
Males								
0	1	91	11	0.0302	0.0301	1.000	54	1.3
1	4	387	3	0.0019	0.0077	0.970	55	1.3
5	10	862	2	0.0006	0.0058	0.962	51	1.2
15	10	719	10	0.0035	0.0342	0.957	42	1.2
25	10	424	15	0.0088	0.0847	0.924	32	1.2
35	10	282	13	0.0115	0.1090	0.846	26	1.1
45	10	212	20	0.0236	0.2110	0.754	18	1.0
55	10	84	22	0.0655	0.4933	0.595	12	1.0
65	+	78	40	0.1282	1.0000	0.301	8	-
Females								
0	1	121	9	0.0186	0.0186	1.000	68	1.6
1	4	334	1	0.0008	0.0030	0.981	68	1.5
5	10	840	1	0.0003	0.0030	0.979	64	1.5
15	10	765	5	0.0016	0.0162	0.976	54	1.5
25	10	462	6	0.0032	0.0320	0.960	45	1.5
35	10	290	7	0.0060	0.0586	0.929	37	1.5
45	10	216	11	0.0127	0.1197	0.875	29	1.3
55	10	119	10	0.0210	0.1901	0.770	22	1.1
65	+	75	19	0.0633	1.0000	0.624	16	-

1. Based on mortality data for nine western New South Wales communities (see section 4.2 in chapter 4). For each age interval x this table yields information on: the duration of the interval, n ; the population at risk, P_x ; the number of deaths, D_x ; the age-specific death rate, ${}_nM_x$; the probability of dying, ${}_nq_x$; the proportion of people who survived, l_x ; the life expectancy at age x , e_x^o ; and the standard error around each e_x^o value for a closed age interval, S.E.

Table A1.13 Comparison of expectation of life (e_x^o) for Aborigines living in nine western New South Wales communities¹, 1980-1983 and 1984-1987, by sex.

x	n	Aboriginal 1984-1987		Aboriginal 1980-1983		Critical ratio ³
		e ^o _x	S.E. ²	e ^o _x	S.E. ²	
Males						
0	1	54	1.3	58	1.4	1.97
1	4	55	1.3	59	1.4	1.85
5	10	51	1.2	55	1.4	1.91
15	10	42	1.2	45	1.3	1.97
25	10	33	1.2	36	1.3	1.84
35	10	26	1.1	28	1.3	1.72
45	10	18	1.0	21	1.1	2.15
55	10	12	1.0	15	1.1	2.37
Females						
0	1	68	1.6	67	1.5	-0.46
1	4	68	1.5	67	1.5	-0.42
5	10	64	1.5	64	1.4	-0.33
15	10	54	1.5	54	1.4	-0.33
25	10	45	1.5	45	1.4	-0.19
35	10	37	1.5	36	1.4	-0.25
45	10	29	1.3	30	1.1	0.63
55	10	22	1.1	21	1.0	-0.47

1. Based on mortality data for nine western New South Wales communities (see section 4.2 in chapter 4).

2. Standard error of e_x^o .

3. If the critical value exceeds 2.33 then the differences between life expectancies are significant at a 0.01 level of probability.

Table A1.14 Sex-specific abridged life tables for Aborigines living in two western New South Wales communities¹, 1979-1988.

x	n	P_x	D_x	${}_nM_x$	${}_nq_x$	l_x	e_x^o	S.E.
Males								
0	1	19	8	0.0421	0.0420	1.000	47	1.4
1	4	96	0	0.0000	0.0000	0.958	48	1.3
5	10	151	1	0.0007	0.0066	0.958	44	1.3
15	10	167	9	0.0054	0.0525	0.952	34	1.2
25	10	106	11	0.0104	0.0986	0.902	26	1.2
35	10	62	13	0.0210	0.1898	0.812	18	1.1
45	10	37	20	0.0540	0.4255	0.658	12	0.9
55	10	14	19	0.1357	0.8085	0.378	7	0.7
65	+	13	35	0.2692	1.0000	0.072	4	-
Females								
0	1	28	6	0.0214	0.0214	1.000	61	1.6
1	4	72	0	0.0000	0.0000	0.979	61	1.6
5	10	156	0	0.0000	0.0000	0.979	57	1.6
15	10	182	5	0.0028	0.0271	0.979	47	1.6
25	10	103	6	0.0058	0.0566	0.952	38	1.6
35	10	54	7	0.0130	0.1217	0.898	30	1.6
45	10	49	8	0.0163	0.1509	0.789	23	1.2
55	10	25	5	0.0200	0.1818	0.670	17	0.1
65	+	17	18	0.1059	1.0000	0.548	9	-

1. Based on mortality data for Bourke and Brewarrina (see section 4.2 in chapter 4). For each age interval x this table yields information on: the duration of the interval, n ; the population at risk, P_x ; the number of deaths, D_x ; the age-specific death rate, ${}_nM_x$; the probability of dying, ${}_nq_x$; the proportion of people who survived, l_x ; the life expectancy at age x , e_x^o ; and the standard error around each e_x^o value for a closed age interval, S.E.

Table A1.15 Comparison of expectation of life (e_x°) for Aborigines living in two western New South Wales communities¹, 1979-1988, and western New South Wales², 1984-1987, by sex.

x	n	Aboriginal 1979-1988		Aboriginal 1984-1987		Critical ratio ⁴
		e ^o _x	S.E. ³	e ^o _x	S.E. ³	
Males						
0	1	47	1.4	53	1.1	3.39
1	4	48	1.3	55	1.1	3.82
5	10	44	1.3	51	1.0	4.15
15	10	35	1.2	41	1.0	4.13
25	10	26	1.2	33	1.0	3.15
35	10	18	1.1	25	1.0	4.60
45	10	12	0.9	17	0.9	4.68
55	10	7	0.7	12	0.9	4.55
Females						
0	1	61	1.6	66	1.2	2.59
1	4	61	1.6	66	1.2	2.43
5	10	57	1.6	62	1.2	2.53
15	10	47	1.6	52	1.2	2.68
25	10	38	1.6	43	1.2	2.54
35	10	30	1.6	34	1.1	2.27
45	10	24	1.2	26	1.0	1.67
55	10	17	0.1	19	0.9	1.89

1. Based on mortality data for Bourke and Brewarrina (see section 4.2 in chapter 4).

2. Based on mortality data for nine western New South Wales communities (see section 4.2 in chapter 4).

3. If the critical value exceeds 2.33 then the differences between life expectancies are significant at a 0.01 level of probability.

4. Standard error of e_x° .

APPENDIX TWO
QUESTIONNAIRES USED IN THIS STUDY

DEMOGRAPHIC SURVEY - DEATH RECORD

Names: _____
 First and Second Names Surname

Aboriginal or Torres Strait Islander?
 Yes ... 1 No ... 2 Maybe ... 3

Recording of Aboriginality by Registrar/ ABS?
 Yes ... 1 No ... 2

Notification source:
 Registry ... 1 AHU ... 2 ABS ... 3
 Other: (list: _____)

Notification centre: _____

Death registration number: _____
 Yr ID Number

Place of residence: _____

Did he or she live:
 In town? ... 1 On a mission? ... 2

Place of Death: _____

Did the death occur in a hospital?
 Yes ... 1 No ... 2

Date of death: _____
 Day Month Year

Date of birth: ____/____/19____ or Age: ____
 Day Month Year

Age type: Minute ... 1 Hour ... 2 Day ... 3
 Week ... 4 Month ... 5 Year ... 6

Sex: Male 1
 Female ... 2

Surviving relatives (Blacken those not relevant)
 Mother Yes ... 1 Father Yes ... 1
 No 2 No 2

Partner Yes ... 1 Child(ren) Living: ____
 No 2 Dead: ____

Questions about deceased person 15 years and over

Marital status: Never had a partner 1
 Married or with a partner . 2
 Separated or divorced 3
 Widow or widower 4

As recorded in registration: _____

Occupation (in registration): _____

Employed in the last twelve months?
 Yes ... 1 No ... 2 At school ... 3

Lived in own house before final illness?
 Yes 1 No 2
 15-30 yrs, with parents, no partner .. 3

--	--	--	--

IDNUM

☐

ABOR

☐

NOTIFAB

☐

NOTSOUR

--	--

REGNO

--	--

RESCODE

☐

TOWN

--	--

PLACE

☐

HOSPITAL

DATEDETH

--	--	--

AGE

☐

AGETYPE

☐

SEX

☐

MOTH

☐

FATH

☐

PART

--	--

CHL
CHD
☐

MSTATUS

☐

MREG

☐

OCCUPAT

☐

EMPLOY

☐

HOME

DEMOGRAPHIC SURVEY - DEATH RECORD

Cause of death: _____

Inquest?

Yes ... 1 No ... 2

CAUSE1

CAUSE2

CAUSE3

ICD9

INQUEST

Questions about deceased person 15 years and over

Heart disease?

Yes ... 1 No ... 2

Hypertensive and other circulatory system
disease?

Yes ... 1 No ... 2

Liver disease?

Yes ... 1 No ... 2

Kidney disease?

Yes ... 1 No ... 2

Diabetes?

Yes ... 1 No ... 2

Other pancreatic disease?

Yes ... 1 No ... 2

Chronic alcoholism?

Yes ... 1 No ... 2

Chronic obstructive airways disease?

Yes ... 1 No ... 2

Other airways disease?

Yes ... 1 No ... 2

Cancer?

Yes ... 1 No ... 2

Accidental or violent death?

Yes ... 1 No ... 2

Infectious (viral/ bacterial) diseases?

Yes ... 1 No ... 2

Epilepsy?

Yes ... 1 No ... 2

Comments:

CA

CB

CC

CD

CED

CEO

CF

CG

CH

CI

CJ

CM

CN

ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY

HOUSEHOLD INTERVIEW

FOR ALL PERSONS IN THE HOUSEHOLD

CONFIDENTIAL

The Aboriginal Health Service
Bourke NSW

Research School of Social Sciences
The Australian National University
Canberra ACT

**ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
NAME AND ADDRESS SHEET****THIS SHEET WILL NOT BE KEPT**

Household number: _____

Household address:

People in household on / / 1989
 day month

Person number	Mr/Mrs Miss/Ms	Name
1		
2		
3		
4		
5		
6		
7		
8		
9		
10		
11		
12		
13		
14		
15		

**ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
HOUSEHOLD DATA SHEET 1**

Demographic details of people in the
household on / / 1989
 day month

Person number	Sex (M/F)	Date of birth or Age				Both parents still alive?	
		day	mnth	yr	age	Mum (yes/no)	Dad (yes/no)
1		:	:				
2		:	:				
3		:	:				
4		:	:				
5		:	:				
6		:	:				
7		:	:				
8		:	:				
9		:	:				
10		:	:				
11		:	:				
12		:	:				
13		:	:				
14		:	:				
15		:	:				

**ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
HOUSEHOLD DATA SHEET 2**

Relationships in household on / / 1989
 day month

DO NOT USE NAMES TO INDICATE RELATIONSHIPS

ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
HOUSEHOLD DATA SHEET 3

- 1 Are there any people who usually live in the household who are not here today?
YES / NO

[If "yes" record the details of these people on the name and address sheet and household data sheets in **red pen**.]

- 2 Description of dwelling (ask question if necessary):

Detached house	1
Semi detached house or townhouse	2
Flat or unit	3
Garage converted to dwelling	4
Caravan	5
Other (explain: _____)	6

☐
HSTYPE

- 3 Material of outer walls (ask question if necessary):

Brick or brick veneer	1
Concrete block	2
Weatherboard	3
Fibro cement	4
Other (explain: _____)	5

☐
MATERL

- 4a How many bedrooms does this dwelling have?
Number: _____

BEDRMS

- 4b How many other rooms (excluding toilet, laundry, bathroom)?
Number: _____

OTHRMS

- 5 Does this dwelling have:

Running water?	Yes	1
	No	2

☐
WATER

Electricity?	Yes	1
	No	2

☐
ELECT

- 6 Is this dwelling rented, or mortgaged, or is it owned by someone living here?

Renting	1
Buying	2
Owned	3
Not rented, owned or being bought	4
Other (explain: _____)	

☐
HSEOWN

ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY

INDIVIDUAL INTERVIEW

QUESTIONS TO BE ASKED OF PERSONS 18 YEARS AND OVER

CONFIDENTIAL

The Aboriginal Health Service
Bourke NSW

Research School of Social Sciences
The Australian National University
Canberra ACT

ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY

INDIVIDUAL INTERVIEW

QUESTIONS TO BE ASKED OF PERSONS 18 YEARS AND OVER

Name of respondent: _____

The Aboriginal Health Service
Bourke NSW

The Research School of Social Sciences
The Australian National University
Canberra ACT

THIS SHEET WILL NOT BE KEPT

**ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
CLINICAL DATA SHEET 1**

- 1 **ENTER** the identification numbers of the respondent.

Household number:

Person number:

HSHLDN

PERSONN

- 2 Height (whole cm): _____ cm.

HEIGHTCM

- 3 Weight (0.5 Kg): _____ . _____ Kg.

WEIGHTKG

- 4 Has your weight changed by more than half stone (3 kilograms) in the past year?

Increased 1

Decreased 2

No change 3

☐

WTCHANGE

- 5a Blood pressure (sitting):

Systolic: _____ mm Hg.

Diastolic: _____ mm Hg.

BPSYSTOL

BPDIASTO

- 5b Would you like the results of your blood pressure reading sent to your doctor?

If **"yes"** what is the address?

Name: _____

Address: _____

The next set of questions are to be asked only of women of childbearing age (18 to 50 years).

- 6a Are you now pregnant?

Yes 1

No 2 (go to 7)

☐

PREGNANT

- 6b If **"yes"** how many months have you been pregnant?

Months: _____

PRGMNTHS

- 7 Are you now breastfeeding?

Yes 1

No 2

☐

BRSTFEED

**ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
INDIVIDUAL INTERVIEW DATA SHEET 1**

Questions 1 to 4 to be completed before starting.

- 1 **ENTER** the identification numbers of the respondent.
Household number:

Person number:
- 2 **ENTER** sex of respondent.
Male 1
Female 2
- 3 **ENTER** age or date of birth of respondent.
Age: _____
- 4 **ENTER** interview date and interviewer.
Interview date: / /1989
 day month year

Interviewer: _____

HSHLDN

PERSONN

☐
SEX

AGE

Read this statement when starting.

Thank you for your participation. I am going to ask you some questions about your health, lifestyle and diet. The interview is voluntary and all information provided will be held in strict confidence. If there are any questions you do not want to answer, just let me know and we will go to the next question. If you have any questions please ask me. The interview will take 25 to 30 minutes to complete and will start now.

Note: If any questions are not answered please put a line through the question to show missing information.

First, I would like to ask you some questions about yourself.

- 5 How long have you lived in this town?
Less than 1 year 1
More than 1 year, but less than 5 .. 2
More than 5 years 3
Have always lived here 4

☐
RESSTAT

**ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
INDIVIDUAL INTERVIEW DATA SHEET 2**

- 6 What is your present marital status?
- | | |
|--------------------------------------|---|
| Single | 1 |
| Married and living with spouse | 2 |
| Living in a de facto relationship .. | 3 |
| Divorced or permanently separated .. | 4 |
| Widowed | 5 |
| Other (explain: _____) | |

☐

MARSTAT

- 7a Are you currently a full time or part time student?

Yes 1
No 2 (go to 7c)

☐

STUDENT

- 7b If "yes" are you currently enrolled in...
- | | |
|-------------------------------------|---|
| Year 10 | 1 |
| Year 11 or 12 | 2 |
| Trade/ apprenticeship program | 3 |
| University, CAE, or other | 4 |
| Tertiary institution | 5 |
| Other (explain: _____) | |

☐

CURSCHL

(From 7b go to 8a)

- 7c If "no" which of the following BEST DESCRIBES your highest level of education?
- | | |
|---|---|
| Never attended school | 1 |
| Completed primary school | 2 |
| Completed year 10 or equivalent certificate | 3 |
| Completed year 12 | 4 |
| Trade/ apprenticeship | 5 |
| Completed a University, CAE or other tertiary degree or diploma ... | 6 |
| Other (explain: _____) | |

☐

PSTSCHL

- 8a Do you have a job?

Yes 1
No 2 (go to 8d)

☐

JOB

- 8b If "yes" which of the following BEST DESCRIBES your current employment status?

Full time 1
Part time (less than 35 hours/ week). 2

☐

EMPLOY1

- 8c What is your current occupation?

Occupation: _____ (go to 9a)

☐

OCCUPAT

ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
INDIVIDUAL INTERVIEW DATA SHEET 3

8d If "no" which of the following BEST DESCRIBES
your current employment status?

- Looking for work 1
- Full time or part time student 2
- Home duties and not seeking work .. 3
- Permanently unable to work 4
- Retired 5
- Other (explain: _____)

☐

EMPLOY2

9a Do you receive a pension, benefit or
allowance?

- Yes 1
- No 2 (go to 10)

☐

PENSION

9b If "yes" what type of pension, benefit or
allowance do you receive?

- Unemployment/ JOB SEARCH 1
- Sickness benefit 2
- Age pension or widow's pension 3
- Invalid pension 4
- Supporting parents benefit 5
- Disability pension 6
- Service or defence pension/ war
widow's pension 7
- ABSTUDY/ AUSTUDY/ student allowance.. 8
- Other (explain: _____)

☐

PENTYPE

Now, I would like to ask you some questions
about your health.

10 When you feel sick or in need of medical
advice where do you go for help?

Prompts:

Would you go to the hospital?

**ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
INDIVIDUAL INTERVIEW DATA SHEET 4**

Would you seek help and advice from a doctor?

Would you seek help and advice from an
Aboriginal Health worker?

Would you seek help and advice from your
own relations? [If "Yes", Ask Who?]

- 11 Are you satisfied with the medical services
which are available to you in this town?

Prompts:

What about the services available from the
Aboriginal Health Unit or Health Service?

ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
INDIVIDUAL INTERVIEW DATA SHEET 5

What about the services available from the doctor?

What about the services available from the hospital?

- 12a Within the last 12 months were you admitted as an in-patient to any hospital?
Yes, as a day patient only ... 1
Yes, for overnight or longer . 2
No 3 (go to 13)

☐ HSPITAL

12b Can you tell me what medical condition you had when you were admitted to hospital?

12c What was the name of the hospital that you were admitted to?

ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
INDIVIDUAL INTERVIEW DATA SHEET 6

12d Did you put off going to hospital right away?

12e Were you pleased with the attention and care you received in hospital?

Prompts:

Were you pleased with the attention and care you received from the doctors?

Were you pleased with the attention and care you received from the nurses?

Did the nurses and doctors tell you everything about what was going on?

**ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
INDIVIDUAL INTERVIEW DATA SHEET 7**

Do you feel you would have had better treatment and care if you had been admitted to a different hospital?

13 Have **you** ever been told by a doctor that you had:

High blood pressure?	Yes	1
	No	2
High blood cholesterol?	Yes	1
	No	2
Diabetes?	Yes	1
	No	2
Heart attack?	Yes	1
	No	2
Stroke?	Yes	1
	No	2

☐

BPRESS

☐

CHOLEST

☐

DIABET

☐

HATTACK

☐

STROKE

14a How would you rate your health now? Is it...

Excellent	1
Very good	2
Good	3
Fair	4
Poor	5

☐

HEALTH

14b How would you rate your health compared to most Koori people your age?

Better	1
About the same	2
Worse	3
Other (explain: _____)	

☐

HLTHRL

**ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
INDIVIDUAL INTERVIEW DATA SHEET 8**

15a Have you ever had any pain or discomfort in your chest including pains like bad indigestion?

Yes 1
No 2 (go to 17a)
Maybe ... 3

☐
CHPAIN1

15b Do you ever get chest pains when you walk fast or run?

Yes 1
No 2 (go to 16)
Never hurries ... 3

☐
CHPAIN2

15c Do you ever get chest pains when you walk at an ordinary pace?

Yes 1
No 2 (go to 16)

☐
CHPAIN3

15d If you get chest pains while you are walking do you...

Stop or slow down 1
Carry on 2 (go to 16)

☐
CHPAIN4

Record "Stop or slow down" if subject carries on after taking angina medicine.

15e If you stand still, are the pains...

Relieved 1
Not relieved 2 (go to 16)

☐
CHPAIN5

15f How soon?

10 minutes or less 1
More than 10 minutes .. 2 (go to 16)

☐
CHPAIN6

15g Will you show me where the pains were?

Sternum (upper or middle) Yes ... 1
No 2

☐
UPSTERN

Sternum (lower) Yes ... 1
No 2

☐
LOSTERN

Left anterior chest Yes ... 1
No 2

☐
LANTCHS

Left arm Yes ... 1
No 2

☐
LARM

Other Yes ... 1
No 2

☐
OTHPAIN

Record all areas mentioned: _____

**ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
INDIVIDUAL INTERVIEW DATA SHEET 9**

15h Do you feel the pains anywhere else?

Yes 1
No 2

If "yes", record information above in 15g.

☐
CHPAIN7

16 Have you ever had a severe pain across the front of your chest lasting half an hour or more?

Yes 1
No 2

☐
POSSINF

17a Do you get pain in either leg when you are walking?

Yes 1
No 2 (go to 18)

☐
CLAUD1

17b Does pain ever begin when you are standing still or sitting?

Yes 1 (go to 18)
No 2

☐
CLAUD2

17c In what part of your leg do you feel the pain?

Pain includes calf/ calves 1
Pain does not include calf/ calves 2

☐
CLAUD3

If calves not mentioned, ask: Anywhere else?

☐
CLDOTH

17d Do you get the pain when you walk fast or run?

Yes 1
No 2 (go to 18)
Never hurries ... 3

☐
CLAUD4

17e Do you get the pain if you walk at an ordinary pace?

Yes 1
No 2 (go to 18)

☐
CLAUD5

17f Does the pain ever disappear while you are walking?

Yes 1 (go to 18)
No 2

☐
CLAUD6

17g If you get the pain when you are walking do you...

Stop or slow down 1
Carry on 2 (go to 18)

☐
CLAUD7

17h If you stand still is the pain...

Relieved 1
Not relieved 2

☐
CLAUD8

17i How soon?

10 minutes or less 1
More than 10 minutes .. 2

☐
CLAUD9

ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
INDIVIDUAL INTERVIEW DATA SHEET 10

18 Now I want to find out about the exercise you had during the **PAST 2 WEEKS**. Firstly, exercise for health fitness, recreation, or sport and secondly, exercise as part of your daily tasks.

18a In the PAST 2 WEEKS have you participated in any type of physical exercise for health fitness, recreation, or sport?

Yes 1

No 2 (go to 19a)

☐

EXER

18b If "yes" in which of the following exercise activities have you participated in the **PAST 2 WEEKS**? Please indicate the total number of times you participated in an activity and the average duration of each exercise session.

☐☐

VEXERN

☐☐☐

VEXERT

EXERCISE

TOTAL
NUMBER

AVERAGE
DURATION

HEALTH FITNESS

Aerobic exercise
class

☐☐

LVEXEN

Jogging/ running

☐☐☐

LVEXET

Weight lifting

RECREATION

Bush walking

Cycling

Dancing

Fishing

Gathering bush foods

Horseback riding

Hunting

Swimming

ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
INDIVIDUAL INTERVIEW DATA SHEET 11

SPORTS	TOTAL NUMBER	AVERAGE DURATION
Basketball	_____	_____
Boxing	_____	_____
Golf	_____	_____
Hockey	_____	_____
Lawn bowling	_____	_____
Netball	_____	_____
Rugby/ Football	_____	_____
Tennis	_____	_____
OTHER (explain)	_____	_____
_____	_____	_____
_____	_____	_____

- 19a In the **PAST 2 WEEKS**, in the course of your daily activities either at work or around home, were you involved in **moderate to heavy physical exercise** which made you breathe harder or sweat?

Yes 1
No 2 (go to 20)

☐
DLYACT1

- 19b If "**yes**" in which of the following exercise activities have you participated in the **PAST 2 WEEKS**? Please indicate the total number of times you participated in an activity and the average duration of each exercise session.

DLYACTH

EXERCISE	TOTAL NUMBER	AVERAGE DURATION
DAILY ACTIVITIES		
Gardening	_____	_____
Hard manual labour	_____	_____
Housework/ cleaning	_____	_____
Light manual labour	_____	_____
Walking	_____	_____

DLYACTW

ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
INDIVIDUAL INTERVIEW DATA SHEET 12

OTHER (explain)	TOTAL NUMBER	AVERAGE DURATION
20 Now, I would like to ask you some questions about your diet. The first set of questions will be concerned with the amount of salt you use and the second with your eating habits.		
Please indicate to me the number of times that you have eaten the following foods in the PAST THREE DAYS , not counting today. It is important that you answer the questions as accurately as possible, indicating every time you have eaten any of the foods mentioned. We count the occasions, not the amounts (for example, 3 slices of bread score 1, if eaten at one occasion).		
20a Food with salt added in cooking. 0 1 2 3 4 5 6 7 8 or more		 SALT1
20b Food with salt added at the table. 0 1 2 3 4 5 6 7 8 or more		 SALT2
20c Meats, such as ham, bacon, sausages, or luncheon meats. 0 1 2 3 4 5 6 7 8 or more		 SALT3
20d Corned/ canned meats, salami, meat-pastes. 0 1 2 3 4 5 6 7 8 or more		 SALT4
20e Pies, pasties, or sausage-rolls. 0 1 2 3 4 5 6 7 8 or more		 SALT5
20f Smoked or canned fish, fish-pastes (salted). 0 1 2 3 4 5 6 7 8 or more		 SALT6
20g Mature cheese, cheese spreads. 0 1 2 3 4 5 6 7 8 or more		 SALT7
20h Processed cheese (other than unsalted or low salt). 0 1 2 3 4 5 6 7 8 or more		 SALT8
20i Yeast vegetable extract such as Vegemite, Promite, and Marmite. 0 1 2 3 4 5 6 7 8 or more		 SALT9

**ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
INDIVIDUAL INTERVIEW DATA SHEET 13**

20j Olives, salted nuts, crackers, potato crisps. 0 1 2 3 4 5 6 7 8 or more		SALT10
20k Canned vegetables, canned soups (other than unsalted). 0 1 2 3 4 5 6 7 8 or more		SALT11
20l Packet soups, beef/chicken cubes. 0 1 2 3 4 5 6 7 8 or more		SALT12
20m Dressings, sauces, pickles (other than unsalted). 0 1 2 3 4 5 6 7 8 or more		SALT13
20n Bread (salted). 0 1 2 3 4 5 6 7 8 or more		SALT14
20o Breakfast cereals (salted). 0 1 2 3 4 5 6 7 8 or more		SALT15
20p Cakes, pastries, biscuits (other than low sodium). 0 1 2 3 4 5 6 7 8 or more		SALT16
20q Butter or margarine (salted). 0 1 2 3 4 5 6 7 8 or more		SALT17
20r Chocolate or confectionery (lollies). 0 1 2 3 4 5 6 7 8 or more		SALT18
20s More than 1 cup (half a pint) of milk per serving. 0 1 2 3 4 5 6 7 8 or more		SALT19
20t Pharmaceutical goods containing sodium, for example sodium pain killers (not including Disprin, Solcode, and Solprin), effervescent vitamins and minerals, some health drinks, indigestion remedies, and laxatives. 0 1 2 3 4 5 6 7 8 or more		SALT20
20u Any salt-containing food that has not been mentioned. 0 1 2 3 4 5 6 7 8 or more		SALT21
20v Compared to your normal diet over the past month, the amount of salted food that you have eaten in the PAST THREE DAYS has been: Much more 1 A little more 2 About the same 3 A little less 4 Much less 5		SALT22

ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
INDIVIDUAL INTERVIEW DATA SHEET 14

Now, I would like you to recall what you ate and drank yesterday - starting from the morning.

MORNING MEAL

Recall 1: What did you have to eat and drink for your morning meal?

21a Was morning meal eaten at home?
Yes 1 (go to 21c)
No 2

☐

HMEAT1

21b If "no" where was it eaten then?
Location: _____

☐

LOCAT1

21c Was it:
Prepared at home? 1
Purchased? 2
Other (explain: _____)

☐

MLPRP1

21d What did you eat?

Description of food and drink	Cooking method	Portion size

ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
INDIVIDUAL INTERVIEW DATA SHEET 15

MORNING SNACK 1

Recall 2: What did you have to eat and drink
in the morning between your morning
and midday meals?

22a Was your first snack in the morning eaten at
home?

Yes 1 (go to 22c)
No 2

☐

HMEAT2

22b If "no" where was it eaten then?

Location: _____

☐

LOCAT2

22c Was it:

Prepared at home? 1
Purchased? 2
Other (explain: _____)
_____)

☐

MLPRP2

22d What did you eat?

Description of food and drink	Cooking method	Portion size

ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
INDIVIDUAL INTERVIEW DATA SHEET 16

MORNING SNACK 2

Recall 3: What did you have to eat and drink
in the morning between your morning
and midday meals?

23a Was your second snack in the morning eaten at
home?

Yes 1 (go to 23c)
No 2

☐ HMEAT3

23b If "no" where was it eaten then?

Location: _____

☐ LOCAT3

23c Was it:

Prepared at home? 1
Purchased? 2
Other (explain: _____)

☐ MLPRP3

23d What did you eat?

Description of food and drink	Cooking method	Portion size

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ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
INDIVIDUAL INTERVIEW DATA SHEET 17

MIDDAY MEAL

Recall 4: What did you have to eat and drink
for your midday meal?

24a Was your midday meal eaten at home?
Yes 1 (go to 24c)
No 2

☐
HMEAT4

24b If "no" where was it eaten then?
Location: _____

☐☐
LOCAT4

24c Was it:
Prepared at home? 1
Purchased? 2
Other (explain: _____)
_____)

☐
MLPRP4

24d What did you eat?

Description of food and drink	Cooking method	Portion size

ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
INDIVIDUAL INTERVIEW DATA SHEET 18

AFTERNOON SNACK 1

Recall 5: What did you have to eat and drink
in the afternoon between your
midday and evening meals?

25a Was your first snack in the afternoon eaten
at home?

Yes 1 (go to 25c)
No 2

☐

HMEAT5

25b If "no" where was it eaten then?

Location: _____

☐

LOCAT5

25c Was it:

Prepared at home? 1
Purchased? 2
Other (explain: _____)
_____)

☐

MLPRP5

25d What did you eat?

Description of food and drink	Cooking method	Portion size

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ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
INDIVIDUAL INTERVIEW DATA SHEET 19

AFTERNOON SNACK 2

Recall 6: What did you have to eat and drink
in the afternoon between your
midday and evening meals?

26a Was your second snack in the afternoon eaten
at home?

Yes 1 (go to 26c)
No 2

☐
HMEAT6

26b If "no" where was it eaten then?

Location: _____

☐☐
LOCAT6

26c Was it:

Prepared at home? 1
Purchased? 2
Other (explain: _____)
_____)

☐
MLPRP6

26d What did you eat?

Description of food and drink	Cooking method	Portion size

ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
INDIVIDUAL INTERVIEW DATA SHEET 20

EVENING MEAL

Recall 7: What did you have to eat and drink
for your evening meal?

27a Was your evening meal eaten at home?

Yes 1 (go to 27c)

No 2

☐

HMEAT7

27b If "no" where was it eaten then?

Location: _____

☐

LOCAT7

27c Was it:

Prepared at home? 1

Purchased? 2

Other (explain: _____)

☐

MLPRP7

27d What did you eat?

Description of food and drink	Cooking method	Portion size

Recall 8: What did you have to eat and drink in the evening after your evening meal?

Yes 1 (go to 28c)
No 2

11

Location: _____

--	--

Prepared at home? 1
Purchased? 2
Other (explain:

--	--

28d What did you eat?

Description of food and drink	Cooking method	Portion size

ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
INDIVIDUAL INTERVIEW DATA SHEET 22

EVENING SNACK 2:

Recall 9: What did you have to eat and drink
in the evening after your evening
meal?

29a Was your second snack in the evening eaten at
home?

Yes 1 (go to 29c)
No 2

☐

HMEAT9

29b If "no" where was it eaten then?

Location: _____

☐

LOCAT9

29c Was it:

Prepared at home? 1
Purchased? 2
Other (explain: _____)

☐

MLPRP9

29d What did you eat?

Description of food and drink	Cooking method	Portion size

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ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
INDIVIDUAL INTERVIEW DATA SHEET 23

MEAL/ SNACK: _____

Recall 10: What other meals and snacks did you have yesterday?

30a Did you eat this meal or snack at home?
Yes 1 (go to 30c)
No 2

☐
HMEAT10

30b If "no" where was it eaten then?
Location: _____

☐☐
LOCAT10

30c Was it:
Prepared at home? 1
Purchased? 2
Other (explain: _____)

☐
MLPRP10

30d What did you eat?

Description of food and drink	Cooking method	Portion size

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ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
INDIVIDUAL INTERVIEW DATA SHEET 24

MEAL/ SNACK: _____

Recall 11: What other meals and snacks did you have yesterday?

31a Did you eat this meal or snack at home?
Yes 1 (go to 31c)
No 2

☐
HMEAT11

31b If "no" where was it eaten then?
Location: _____

☐☐
LOCAT11

31c Was it:
Prepared at home? 1
Purchased? 2
Other (explain: _____)

☐
MLPRP11

31d What did you eat?

Description of food and drink	Cooking method	Portion size

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ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
INDIVIDUAL INTERVIEW DATA SHEET 25

MEAL/ SNACK: _____

Recall 12: What other meals and snacks did you have yesterday?

32a Did you eat this meal or snack at home?
Yes 1 (go to 32c)
No 2

☐
HMEAT12

32b If "no" where was it eaten then?
Location: _____

☐☐
LOCAT12

32c Was it:
Prepared at home? 1
Purchased? 2
Other (explain: _____)

☐
MLPRP12

32d What did you eat?

Description of food and drink	Cooking method	Portion size

**ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
INDIVIDUAL INTERVIEW DATA SHEET 26**

Now I would like to ask you some questions about your health and lifestyle.

33a Have you ever smoked tobacco - cigarettes, cigars, or a pipe - regularly?
Yes 1
No 2 (go to 34a)

☐
SMOKER

33b At what age did you start smoking regularly?
Age in years: _____

SMYEARS

33c Have you given up smoking?
Yes 1
No 2 (go to 33e)

☐
GIVENUP

33d If "yes" how many years ago was the last time you gave it up?
Number of years: _____

GVUPYRS

33e How much do or did you smoke?
How many manufactured cigarettes a day. _____ ■

CIGS

How many grams of "hand rolled" cigarette tobacco* per week. _____

ROLLED

How many cigars per week. _____

CIGAR

How many grams of pipe tobacco* per week. _____

PIPE

***Note: A 1.75 ounce pouch of cigarette tobacco equals 50 grams.**

34a How often do you usually drink alcohol like wine, spirits or beer?
I don't drink alcohol 1 (go to 35)
Less than once a week 2
On 1 or 2 days a week 3
On 3 or 4 days a week 4
On 5 or 6 days a week 5
Every day 6

☐
ALCOHOL

34b On a day when you drink alcohol, how many drinks do you usually have?
1 or 2 drinks 1
3 or 4 drinks 2
5 to 10 drinks 3
More than 10 drinks 4

☐
DRINKS

ADULT ABORIGINAL HEALTH, LIFESTYLE, AND NUTRITION SURVEY
INDIVIDUAL INTERVIEW DATA SHEET 27

34c If you normally drink beer is that:

- | | |
|------------------------|---|
| Don't drink beer | 1 |
| Regular beer | 2 |
| Low alcohol beer | 3 |

☐

BEER

35 Thank you very much for your time and cooperation. In regard to the information you have given to us on your cardiovascular fitness would you like those results to be passed on to your doctor?

If "yes", what is your doctor's name and address?

Name: _____

Address: _____

INTERVIEW TERMINATED

INTERVIEW DETAILS:

36 Was the interview completed?

- | | |
|---|---|
| Yes, with little or no missing information | 1 |
| Yes, but with considerable missing information .. | 2 |
| No, terminated | 3 |

37 Please write any further information you think is relevant to the interview.

APPENDIX THREE
ALCOHOL INDEX FOR ASSESSING PERSONS AT RISK

Table A3.1 National Heart Foundation alcohol index for assessing persons at risk¹.

Drinking days per week	Drinks of alcohol per day					
	1 or 2	3 or 4	5 to 8	9 to 12	13 to 20	21 plus
< 1	B	B	B	C	D	E
1 or 2	B	B	B	C	D	E
3 or 4	B	B	C	D	E	F
5 or 6	B	B	D	E	F	F
Every day	B	C	D	E	F	F

1. Source: National Heart Foundation (1985: 62).

Scale:

A Non-drinkers

B No risk male drinkers
Low risk female drinkers
(Average daily intake of less than 3 drinks)

C Low risk male drinkers
Intermediate risk female drinkers
(Average daily intake of 4 drinks or 9 to 12 drinks in any day)

D Intermediate risk male drinkers
High risk female drinkers
(Average daily intake of 5 to 8 drinks or occasional excess)

E High risk male drinkers
Very high risk female drinkers
(Average daily intake of 9 to 12 drinks or frequent or great occasional excessive intake)

F Very high risk male drinkers
Very high risk female drinkers
(Average daily intake of over 12 drinks)

Table A3.2 The alcohol index used in this study to assess persons at risk.

Drinking days per week	Drinks of alcohol per day			
	1 or 2	3 or 4	5 to 10	11 plus
< 1	B	B	B	C
1 or 2	B	B	B	C
3 or 4	B	B	C	C
5 or 6	B	B	C	C
Every day	B	C	C	C

Scale:

A Non-drinkers

B No risk male drinkers
Low risk female drinkers
(Average daily intake of less than 3 drinks)

C At risk male drinkers
High risk female drinkers
(Average daily intake of more than 3 drinks)

Table A3.3 Comparison of weekly alcohol consumption for a non-random sample¹ of male Bourke Aborigines aged 18 years and over, by the number of drinks per drinking day².

Drinking days per week	Drinks of alcohol per day				Total
	1 or 2	3 or 4	5 to 10	11 plus	
< 1	1 (50)	13 (50)	9 (25)	23 (30)	46 (32)
1 or 2	1 (50)	11 (42)	17 (47)	24 (31)	53 (37)
3 or 4	0	1 (4)	9 (25)	17 (22)	27 (19)
5 or 6	0	0	1 (3)	3 (4)	4 (3)
Every day	0	1 (4)	0	11 (14)	12 (8)
Total	2 (100)	26 (100)	36 (100)	78 (100)	142 (100)

Scale ³	Number	Per cent
A Non-drinkers	17	11
B No risk drinkers	53	33
C At risk drinkers	89	56
Total	159	100

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. The numbers in brackets refer to the proportion of individuals who drink x number of drinks per drinking day.

3. Based on the scale in Table A3.2.

Table A3.4 Comparison of weekly alcohol consumption for a non-random sample¹ of female Bourke Aborigines aged 18 years and over, by the number of drinks per drinking day².

Drinking days per week	Drinks of alcohol per day				Total
	1 or 2	3 or 4	5 to 10	11 plus	
< 1	5 (100)	6 (46)	14 (52)	13 (39)	38 (49)
1 or 2	0	7 (54)	10 (37)	12 (36)	29 (37)
3 or 4	0	0	2 (7)	4 (12)	6 (8)
5 or 6	0	0	1 (4)	3 (9)	4 (5)
Every day	0	0	0	1 (3)	1 (1)
Total	5 (100)	13 (100)	27 (100)	33 (100)	78 (100)

Scale ³	Number	Per cent
A Non-drinkers	33	30
B Low risk drinkers	42	38
C High risk drinkers	36	32
Total	111	100

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. The numbers in brackets refer to the proportion of individuals who drink x number of drinks per drinking day.

3. Based on the scale in Table A3.2.

Table A3.5 Estimates of the number of drinks consumed¹ for a non-random sample Bourke Aborigines² aged 18 years and over, by sex, drinking days per week, and the number of drinks per drinking day.

Drinking days per week	Drinks of alcohol per day				Total
	1 or 2	3 or 4	5 to 10	11 plus	
	Males				
< 1	0.1	3.3	4.8	18.1	26.3
1 or 2	0.2	5.5	18.2	37.7	61.6
3 or 4	0.0	1.8	33.8	93.5	129.0
5 or 6	0.0	0.0	5.4	23.6	28.9
Every day	0.0	3.5	0.0	121.0	124.5
Total	0.3	14.0	62.1	293.8	370.3

Estimated number of drinks: 370 drinks / 142 drinkers³ = 2.6 drinks per male per day.

Females					
< 1	0.5	1.5	7.5	10.2	19.8
1 or 2	0.0	3.5	10.7	18.9	33.1
3 or 4	0.0	0.0	7.5	22.0	29.5
5 or 6	0.0	0.0	5.4	23.6	28.9
Every day	0.0	0.0	0.0	11.0	11.0
Total	0.5	5.0	31.1	85.6	122.2

Estimated number of drinks: 122 drinks / 78 drinkers⁴ = 1.6 drinks per female per day.

1. The number of drinks consumed was determined by multiplying the number of respondents in a particular category by the average number of drinks per day they drank and then dividing this sum by the average number of days they drank.

The number of drinks per day were respectively based on weights of 1.5, 3.5, 7.5, and 11, while the number of drinking days were respectively based on average weights of 1/14, 1/7, 1/2, 5/7 and 1. Estimates of the number of drinks per person per day were then determined by dividing the total number of drinks by the total number of drinkers.

2. Aboriginal risk factor prevalence survey conducted in April and May 1989.

3. Total number of male respondents in Table A3.3 who are drinkers.

4. Total number of female respondents in Table A3.4 who are drinkers.

APPENDIX FOUR
ADDITIONAL TABLES FOR CHAPTER SEVEN

Table A4.1 Foods consumed by a non-random sample of Bourke Aborigines¹ aged 18 years and over, by food group².

Food group and subgroup ³	Portion ⁴	Weight ⁵
Cereal and cereal products		
Bread		
Bread roll, white	1 average	60
Bread, white	1 slice	33
Bread, wholemeal	1 slice	27
Other yeast and bread-type products		
*Damper, fried (Johnny cake)	1 scone	55
*Damper, homemade	1 scone	55
Flour		
Flour, white, plain	1 cup	150
Rice and pasta		
*Curried rice	1 portion	170
Noodles, boiled	1 cup	150
Rice, white, boiled	1 cup	130
Breakfast cereals		
Breakfast cereal, all bran	1 bowl	66
Breakfast cereal, corn / rice	1 bowl	30
Breakfast cereal, cornflakes	1 bowl	27
Breakfast cereal, protein fortified	1 bowl	30
Breakfast cereal, sweetened	1 bowl	30
Breakfast cereal, wholewheat	1 bowl	17
Oatmeal / porridge, boiled	1 bowl	234
Cake and cake-type pudding		
Cake, plain, homemade	1 portion	100
Pudding, sponge, steamed, homemade	1 portion	60
Biscuits — savory and sweet		
Biscuit, cracker, high fat	1 biscuit	3
Biscuit, sweet, plain	1 biscuit	10
Desserts containing cereal		
Fruit crumble	1 portion	135

Table A4.1 — Continued

Food group and subgroup ³	Portion ⁴	Weight ⁵
Vegetables		
Potatoes		
Potato, chips, commercial, deep-fried	12 pieces	100
Potato, chips, homemade, deep-fried	17 pieces	90
Potato, chips, oven grilled, fried	12 pieces	100
Potato, new, canned, drained	1 portion	90
Potato, pale skin, baked, flesh	1 medium	90
Potato, pale skin, peeled, boiled / mashed	1 medium	90
Peas and beans		
Baked beans, canned in sauce	0.5 cup	120
Bean, haricot / cannelloni, boiled	0.3 cup	55
Pea, green, boiled	0.3 cup	51
Pea, green, canned, drained	0.3 cup	60
Tomatoes and tomato products		
Tomato, canned	0.5 cup	120
Tomato, fried	1 medium	150
Tomato, raw	1 slice	20
All other vegetables		
Beetroot, peeled, boiled	1 slice	15
Broccoli, boiled	0.3 cup	50
Cabbage, common, unsweetened, boiled	0.3 cup	56
Carrot, peeled, boiled	0.3 cup	55
Capsicum, boiled	1 shell	60
Cauliflower, boiled	0.5 cup	100
Celery, boiled / raw	0.5 cup	60
Cucumber, common, raw, peeled	1 slice	10
Lettuce, common, raw	1 leaf	15
Mushrooms, canned in sauce	0.5 cup	100
Onions, mature, peeled, boiled / raw	0.5 cup	100
Onion, fried	0.5 cup	100
Pumpkin, peeled, boiled	0.5 cup	60
Sweet corn, frozen, boiled	0.3 cup	41
Fruits		
All fruit		
Apple, raw	1 medium	100
Banana, raw	1 medium	100
Orange, raw	1 medium	150
Pineapple	1 slice	80

Table A4.1 — Continued

Food group and subgroup ³	Portion ⁴	Weight ⁵
Meat and meat products		
Beef and veal		
Beef, burger, fried	1 patty	75
Beef, chuck, simmered, lean and fat	1 portion	120
Beef, mince	1 portion	120
Beef, silverside, baked, lean and fat	1 slice	35
Beef, skirt steak, simmered	1 portion	120
Beef, steak, fried, lean and fat	1 portion	120
Veal, crumbed	1 portion	120
Lamb		
Lamb, chop, grilled, lean and fat	1 portion	120
Lamb, heart, baked	1 average	90
Lamb, leg, baked, lean and fat	1 portion	170
Lamb, neck chop, lean and fat	1 portion	120
Poultry		
Chicken, boneless, baked	1 slice	120
Pork: ham, bacon, pork meat		
Bacon, breakfast, pressed grill	1 rasher	20
Ham, lean and fat	1 slice	30
Pork, forequarter chops, grilled	1 portion	120
Meat offals		
Lamb, brain	1 set	140
Lamb, liver, fried	1 portion	120
Lamb, tongue, simmered	0.5 cup	100
Sausages, frankfurters		
Beef, sausage, cooked	2 thick	110
Saveloy / hot dog	1 average	42
Delicatessen meats		
Beef, corned	1 slice	30
Beef, silverside, corned, boiled	1 slice	250
Luncheon meat	2 slices	50

Table A4.1 — Continued

Food group and subgroup ³	Portion ⁴	Weight ⁵
Mixed Dishes		
*Beef stew with vegetables	1 portion	170
*Chicken stew with vegetables	1 portion	170
*Curried chicken	1 portion	170
*Curried mince	1 portion	170
*Curried sausage with vegetables	1 portion	170
*Curried steak	1 portion	170
*Roast lamb with vegetables	1 portion	170
Take-aways (non-fish)		
*Beef burger, plain	1 portion	149
Beef rissoles	1 patty	75
Chicken, average, fried	1 piece	89
Chicken drumstick	1 piece	50
Chiko roll, deep fried	1 roll	170
Chiko / spring roll, fat out	1 roll	170
Dim sims	1 average	50
Meat pie, individual	1 pie	180
Pizza, supreme / commercial	1 slice	70
Sausage roll	1 roll	125
Fish, seafood, and their products		
Fish		
Fish, battered / crumbed, fried	1 fillet	100
Fish, cakes, deep fried	1 cake	93
Fish, finger, fried	1 whole	33
Fish, no coating, steamed	1 fillet	100
Fish, oven fried, coated	1 fillet	100
Crustaceans and molluscs		
Prawns, crumbed / battered	1 portion	180
Canned products, spreads, etc.		
Herring, canned	1 portion	100
Eggs		
Eggs		
*Eggs, scrambled	1 portion	108
Eggs, whole, cooked	1 large	52
Eggs, whole, cooked	1 medium	47
Nuts and seeds		
Peanuts, peanut paste, cashews		
Peanut paste, salted	1 portion	9

Table A4.1 — Continued

Food group and subgroup ³	Portion ⁴	Weight ⁵
Milk and milk products		
Milk		
Milk, fresh, whole	1 glass	260
Milk, fluid, flavoured	1 glass	260
Cheese		
Cheese, cheddar	1 slice	20
Cheese, cheddar, processed	1 slice	21
Fats		
Butter / margarine		
Butter, salted	1 portion	7
Margarine, table, vegetable based	1 portion	7
Other fats		
Fats, domestic, unknown cooking	1 tablespoon	20
*Gravy	1 portion	50
Snack foods		
Snack foods		
Potato crisps, plain	1 portion	50
Sugar, jam, honey and syrups		
Jam and lemon spreads / honey		
Jam, stone / other fruits	1 portion	10
Honey	1 portion	10
Sugar		
Sugar, white	2 teaspoons	7
Sugar, brown	1 teaspoon	4
Confectionery		
Filled and plain chocolate		
Confectionery, bounty bar	1 bar	50
Confectionery, mars bar	1 bar	65
Beverages, non-alcoholic		
Fruit juice and vegetable juices		
Fruit drink, commercial	1 glass	240
Juice, orange, commercial, unsweetened	1 glass	240
Other fruit and vegetable juices	1 glass	240

Table A4.1 — Continued

Food group and subgroup ³	Portion ⁴	Weight ⁵
Beverages, non-alcoholic (continued)		
Soft drinks, sweetened / cordials		
Cordial, concentrate, average	1 glass	250
Soft drinks, cola-based	1 glass	226
Soft drinks, dry ginger / other	1 glass	250
Soft drinks, lemonade	1 glass	230
Soft drinks, low energy	1 glass	250
Soft drinks, soda water	1 glass	250
Coffee / tea		
Coffee, instant, black	1 cup	250
*Coffee, instant, with milk and sugar	1 cup	283
*Coffee, instant, with sugar	1 cup	257
Tea, ceylon, black	1 cup	240
*Tea, ceylon, with milk	1 cup	266
*Tea, ceylon, with milk and sugar	1 cup	273
*Tea, ceylon, with sugar	1 cup	247
Beverages, alcoholic		
Beer / wine		
Beer, standard	1 glass	250
Wine, red	1 glass	100
Condiments, flavourings, and soups		
Sauces / salad dressings		
Chutney / relish, vegetable	1 teaspoon	5
*Curry sauce	1 portion	50
Dressing, Italian	1 tablespoon	20
Dressing, mayonnaise, rich, commercial	1 tablespoon	20
Sauce, tomato, ketchup	1 tablespoon	25
White sauce	1 tablespoon	25
Yeast and meat extracts		
Vegemite	1 portion	6
Soups		
Soup, broth canned / homemade	1 portion	226
Soup, light or vegetable	1 portion	226
Soup, medium canned	1 portion	226
Beverage flavourings, powdered		
Drinking chocolate, powdered	1 tablespoon	10

1. Aboriginal risk factor prevalence survey conducted in April and May 1989.

2. Based on the listing of food groups compiled by the Department of Health (1986).

3. See Table A4.2 for the standardized recipes of those food items marked with an asterisk (*). Salads, salad rolls, and sandwiches were broken up into their various constituents.

4. Average portion size.

5. Weight of food item in grams.

Table A4.2 A listing of the standardized recipes used in this study, by ingredient¹.

Ingredient list	Portion ²	Weight ³
Beef burger, plain		
Beef, burger, fried	1 patty	75
Bread roll, white	1 roll	60
Margarine, table, vegetable based	2 portions	14
Average portion size		149
Beef stew with vegetables		
Beef, chuck, simmered, lean and fat	1 portion	120
Onion, mature, peeled, boiled	0.5 cup	100
Potato, pale skin, peeled, boiled	1 medium	90
Pumpkin, peeled, boiled	0.5 cup	60
Carrot, peeled, boiled	0.3 cup	55
Average portion size		170
Chicken stew with vegetables		
Chicken, boneless, baked	3 slices	360
Onion, mature, peeled, boiled	0.5 cup	100
Potato, pale skin, peeled, boiled	1 medium	90
Carrot, peeled, boiled	0.3 cup	55
Cabbage, common, boiled	0.3 cup	56
Pumpkin, peeled, boiled	0.5 cup	60
Pea, green, canned, drained	0.3 cup	60
Bean, haricot, boiled	0.5 cup	91
Average portion size		170
Coffee, instant, with milk and sugar		
Coffee, instant, black	1 cup	250
Milk, fresh, whole	25 millilitres	26
Sugar, white	2 teaspoons	7
Average portion size		283

Table A4.2 — Continued

Ingredient list	Portion ²	Weight ³
Coffee, instant, with sugar		
Coffee, instant, black	1 cup	250
Sugar, white	2 teaspoons	7
Average portion size		257
Curried chicken		
Chicken, boneless, baked	2 slices	240
Potato, pale skin, peeled, boiled	1 medium	90
Onion, mature, peeled, boiled	0.5 cup	100
Carrot, peeled, boiled	0.3 cup	55
Bean, haricot, boiled	0.3 cup	55
Sweet corn, frozen, boiled	0.5 cup	68
Curry sauce	1 portion	50
Average portion size		170
Curried mince		
Beef, mince, regular, simmered	1 portion	120
Sweet corn, frozen, boiled	0.3 cup	41
Potato, new, canned, drained	1 portion	90
Pea, green, canned, drained	0.3 cup	60
Bean, haricot, boiled	0.3 cup	55
Curry sauce	1 portion	50
Average portion size		170
Curried rice		
Rice, white, boiled	1 cup	130
Potato, pale skin, peeled, boiled	1 medium	90
Onion, fried	0.5 cup	100
Carrot, peeled, boiled	0.3 cup	55
Cabbage, common, boiled	0.3 cup	56
Pumpkin, peeled, boiled	0.5 cup	60
Celery, boiled	0.5 cup	60
Curry sauce	1 portion	50
Average portion size		170

Table A4.2 — Continued

Ingredient list	Portion ²	Weight ³
Curried sausage with vegetables		
Beef, sausage, cooked	2 thick	110
Potato, pale skin, peeled, boiled	1 medium	90
Pumpkin, peeled, boiled	0.5 cup	60
Pea, green, boiled	0.3 cup	51
Sweet corn, frozen, boiled	0.3 cup	41
Capsicum, boiled	1 shell	60
Noodles, boiled	1 cup	150
Curry sauce	1 portion	50
Average portion size		170
Curried steak		
Beef, skirt steak, simmered	1 portion	120
Sweet corn, frozen, boiled	0.3 cup	41
Potato, pale skin, peeled, boiled	1 medium	90
Pea, green, canned, drained	0.3 cup	60
Bean, haricot / cannelloni, boiled	0.3 cup	55
Pumpkin, peeled, boiled	0.5 cup	60
Curry sauce	1 portion	50
Average portion size		170
Curry sauce		
Fats, domestic, unknown cooking	1 tablespoon	20
Flour, white, plain	0.3 cup	50
Curry powder	3 teaspoons	12
Sugar, brown	1 teaspoon	4
Chutney / relish, vegetable	2 teaspoons	10
Salt, table	1 portion	0
Average portion size		50
Damper		
Flour, white, plain	0.75 cup	113
Water, plain drinking	0.5 cup	125
Average portion size		55

Table A4.2 — Continued

Ingredient list	Portion ²	Weight ³
Damper, fried (Johnny cake)		
Flour, white, plain	0.75 cup	113
Water, plain drinking	0.5 cup	125
Fats, domestic, unknown cooking	9 tablespoons	180
Average portion size		55
Eggs, scrambled		
Eggs, whole, cooked	1 large	52
Margarine, table, vegetable based	2 portions	14
Milk, fresh, whole	2 tablespoons	42
Salt, table	1 portion	0
Average portion size		108
Gravy		
Fats, domestic, unknown cooking	3 tablespoons	60
Flour, white, plain	0.3 cup	50
Water, plain drinking	100 millilitres	100
Average portion size		50
Roast lamb with vegetables		
Lamb, leg, baked, lean and fat	1 portion	170
Tomato, canned	0.5 cup	120
Onion, mature, peeled, boiled	0.5 cup	100
Average portion size		170

Table A4.2 — Continued

Ingredient list	Portion ²	Weight ³
Salad roll with corned beef		
Bread roll, white	1 average	60
Margarine, table, vegetable based	2 portions	14
Beef, silverside, corned, boiled	0.5 slice	125
Lettuce, common, raw	2 leaves	30
Beetroot, peeled, boiled	2 slices	30
Average portion size		259
Salad		
Tomato, raw	2 slices	40
Cucumber, common, raw, peeled	3 slices	30
Onion, mature, peeled, raw	0.2 cup	40
Celery, raw	0.5 cup	60
Cheese, cheddar	1 slice	20
Average portion size		190
Salad, chicken		
Lettuce, common, raw	2 leaves	30
Tomato, raw	2 slices	40
Cucumber, common, raw, peeled	3 slices	30
Onion, mature, peeled, raw	0.2 cup	40
Celery, raw	0.5 cup	60
Chicken, boneless, baked	2 slices	240
Average portion size		190
Salad with luncheon meat		
Lettuce, common, raw	2 leaves	30
Tomato, raw	2 slices	40
Cucumber, common, raw, peeled	3 slices	30
Onion, mature, peeled, raw	0.2 cup	40
Celery, raw	0.5 cup	60
Luncheon meat	2 slices	50
Average portion size		190

Table A4.2 — Continued

Ingredient list	Portion ²	Weight ³
Sandwich, baked bean		
Bread, white	2 slices	66
Margarine, table, vegetable based	2 portions	14
Baked beans, canned in sauce	0.5 cup	120
Average portion size		200
Sandwich, cheese		
Bread, white	2 slices	66
Margarine, table, vegetable based	2 portions	14
Cheese, cheddar, processed	1 slice	21
Average portion size		101
Sandwich, chicken, lettuce and tomato		
Bread, white	2 slices	66
Margarine, table, vegetable based	2 portions	14
Chicken, boneless, baked	0.75 slice	90
Tomato, raw	2 slices	40
Lettuce, common, raw	2 leaves	30
Average portion size		240
Sandwich, corned beef and ham		
Bread, white	2 slices	66
Margarine, table, vegetable based	2 portions	14
Beef, corned	1 slice	30
Ham, lean and fat	1 slice	30
Average portion size		140

Table A4.2 — Continued

Ingredient list	Portion ²	Weight ³
Sandwich, corned beef		
Bread, white	2 slices	66
Margarine, table, vegetable based	2 portions	14
Beef, corned	1 slice	30
Tomato, raw	2 slices	40
Average portion size		150
Sandwich, curry		
Bread, white	2 slices	66
Margarine, table, vegetable based	2 portions	14
Curried mince	0.5 portion	85
Average portion size		165
Sandwich, egg		
Bread, white	2 slices	66
Margarine, table, vegetable based	2 portions	14
Eggs, whole, cooked	1 medium	47
Dressing, mayonnaise, rich, commercial	1 tablespoon	20
Average portion size		147
Sandwich, ham, cheese and lettuce		
Bread, white	2 slices	66
Margarine, table, vegetable based	2 portions	14
Ham, lean and fat	1 slice	30
Cheese, cheddar, processed	1 slice	21
Lettuce, common, raw	2 leaves	30
Average portion size		161

Table A4.2 — Continued

Ingredient list	Portion ²	Weight ³
Sandwich, ham, cheese and tomato		
Bread, white	2 slices	66
Margarine, table, vegetable based	2 portions	14
Ham, lean and fat	1 slice	30
Cheese, cheddar, processed	1 slice	21
Tomato, raw	1 slice	20
Average portion size		151
Sandwich, jam		
Bread, white	2 slices	66
Margarine, table, vegetable based	2 portions	14
Jam, stone / other fruits	2 portions	20
Average portion size		100
Sandwich, luncheon meat		
Bread, white	2 slices	66
Margarine, table, vegetable based	2 portions	14
Luncheon meat	2 slices	50
Average portion size		130
Sandwich, luncheon meat and tomato		
Bread, white	2 slices	66
Margarine, table, vegetable based	2 portions	14
Luncheon meat	2 slices	50
Tomato, raw	2 slices	40
Average portion size		170

Table A4.2 — Continued

Ingredient list	Portion ²	Weight ³
Sandwich, peanut butter / paste		
Bread, white	2 slices	66
Margarine, table, vegetable based	2 portions	14
Peanut paste, salted	2 portions	18
Average portion size		98
Sandwich, roast silverside		
Bread, white	2 slices	66
Margarine, table, vegetable based	2 portions	14
Beef, silverside, baked, lean and fat	2 slices	70
Average portion size		150
Sandwich, salad		
Bread, white	2 slices	66
Margarine, table, vegetable based	2 portions	14
Lettuce, common, raw	2 leaves	30
Cheese, cheddar	1 slice	20
Beetroot, peeled, boiled	2 slices	30
Average portion size		160
Sandwich, sausage		
Bread, white	2 slices	66
Margarine, table, vegetable based	2 portions	14
Beef, sausage, cooked	1 thick	55
Average portion size		135

Table A4.2 — Continued

Ingredient list	Portion ²	Weight ³
Sandwich, steak		
Bread, white	2 slices	66
Butter, salted	2 portions	14
Beef, steak, fried, lean and fat	1 portion	120
Onion, fried	0.3 cup	60
Average portion size		260
Tea, ceylon, with milk		
Tea, ceylon, black	1 cup	240
Milk, fresh, whole	25 millilitres	26
Average portion size		266
Tea, ceylon, with milk and sugar		
Tea, ceylon, black	1 cup	240
Sugar, white	2 teaspoons	7
Milk, fresh, whole	25 millilitres	26
Average portion size		273
Tea, ceylon, with sugar		
Tea, ceylon, black	1 cup	240
Sugar, white	2 teaspoons	7
Average portion size		247
Toast with honey		
Bread, white	2 slices	66
Margarine, table, vegetable based	2 portions	14
Honey	2 portions	20
Average portion size		100

1. Categorized according to the listing of food groups compiled by the Department of Health (1986).

2. Average portion size.

3. Weight of food item in grams.

Table A4.3 Recommended dietary intakes for Australian males — 1982, 1984, and 1985 revisions¹⁻².

	Age in years	
	18	19-64
Retinol equivalents (μg)	750	750
Thiamin (mg)	1.2	1.1
Riboflavin (mg)	1.9	1.7
Niacin equivalents (mg)	20-22	18-20
Vitamin C (mg) ³	50	40
Iron (mg)	10-13	7
Calcium (mg)	1000	800
Zinc (mg)	12-18	12-16
Magnesium (mg)	320	320

1. Source: National Health and Medical Research Council (1987: 20-21, Table 1).

2. Expressed as mean daily intake.

3. Source: National Health and Medical Research Council (1987: 19, Table 1a).

Table A4.4 Recommended dietary intakes for Australian females — 1982, 1984, and 1985 revisions¹⁻³.

	Age in years		
	18	19-54	55+
Retinol equivalents (µg)	750	750	750
Thiamin (mg)	0.9	0.8	0.7
Riboflavin (mg)	1.4	1.2	1.0
Niacin equivalents (mg)	15-17	12-14	10-12
Vitamin C (mg) ⁴	30	30	30
Iron (mg)	10-13	12-16	5-7
Calcium (mg)	800	800	1000
Zinc (mg)	12-18	12-16	12-16
Magnesium (mg)	270	270	270

1. Source: National Health and Medical Research Council (1987: 20-21, Table 1).

2. Expressed as mean daily intake.

3. Excluding pregnant and lactating women.

4. Source: National Health and Medical Research Council (1987: 19, Table 1a).

REFERENCES CITED

- Abbie, A.A., and J. Schroder
1960 Blood pressures in Arnhem Land Aborigines. *The Medical Journal of Australia*. 2: 493-496.
- Aboriginal Health Organization
n.d. *Renal Survey Report*. Adelaide: South Australian Ministry of Health and Welfare.
- Altman, J.C., and J. Niewenhuysen.
1979 *The Economic Status of Australian Aborigines*. Cambridge: Cambridge University Press.
- Anderson, I.
1988 *Koorie Health in Koorie Hands. An Orientation Manual in Aboriginal Health for Health Care Providers*. Melbourne: Koorie Health Unit, Health Department of Victoria.
- Aronoff, S.L., P.H. Bennett, P. Gorden, N. Rushforth, and M. Miller
1977 Unexplained hyperinsulinaemia in normal and "prediabetic" Pima Indians compared with normal Caucasians. *Diabetes*. 26: 827-840.
- Australian Bureau of Statistics
1976 *Deaths Australia 1976*. Canberra: Australian Government Printer.
1987a *Deaths Australia 1986*. Canberra: Australian Government Printer.
1987b *Estimated Resident Population by Sex and Age: States and Territories of Australia June 1981 to June 1987*. Canberra: Australian Government Printer.
1988a *Regional Statistics New South Wales 1988*. Canberra: Australian Government Printer.
1988b *Cause of Death, New South Wales 1986*. Canberra: Australian Government Printer.
1988c *Deaths New South Wales 1986*. Canberra: Australian Government Printer.
1988d *Overseas Born Australians 1988*. Canberra: Australian Government Printer.
1988e *Cause of Death, Australia 1986*. Canberra: Australian Government Printer.
1989a *Census 86: Data Quality Aboriginal and Torres Strait Islander Counts*. Canberra: Australian Government Printer.
1989b *Deaths Australia 1988*. Canberra: Australian Government Printer.

- Avogaro, P.
1985 Apolipoproteins, the lipid hypothesis, and ischemic heart disease. In Behavioral Epidemiology and Disease Prevention. Kaplan, R. and M. Criqui, eds. Pp. 57-65. New York: Plenum Press
- Bailar, J.C., and F. Ederer
1964 Significance factors for the ratio of a poisson variable to its expectation. *Biometrics* 20: 639-643.
- Balough, M., H.A. Kahn, and J.H. Medalie
1971 Random repeat 24-hour dietary recalls. *American Journal of Clinical Nutrition* 24: 304-310.
- Barr, D.P., E.M. Ross, and H.A. Eder
1951 Protein-lipid relationship in human plasma II. In atherosclerosis and related conditions. *American Journal of Medicine*. 11: 480-484.
- Barwick, D.
1974 The Aboriginal family in south-eastern Australia. In *The Family in Australia: Social, Demographic, and Psychological Aspects*. J. Krupinski and A. Stoller, eds. Pp. 154-167. Sydney: Pergamon Press.
- 1988 Aborigines of Victoria. In *Being Black. Aboriginal Cultures in 'Settled' Australia*. I Keen, ed. Pp. 27-32. Canberra: Aboriginal Studies Press.
- Bastian, P.
1979 Coronary heart disease in tribal Aborigines — the west Kimberley survey. *Australian New Zealand Journal of Medicine*. 9: 284-292.
- Beaglehole, R., I. Prior, C. Salmond, and E. Eyles
1978 Coronary heart disease in Maoris: Incidence and case mortality. *The New Zealand Medical Journal*. 88: 138-141.
- Beaglehole, R., I.A.M. Prior, E. Eyles, and V. Sampson
1979 High density lipoprotein cholesterol and other serum lipids in New Zealand Maoris. *The New Zealand Medical Journal*. 90: 139-142.
- Beard, T.C., and R.F. Heller
1987 Relevance of the salt-hypertension hypothesis to the community control of hypertension. *The Medical Journal of Australia*. 147: 29-38.
- Becker, T.M., C. Wiggins, C.R. Key, and J.M. Samet
1988 Ischaemic heart disease mortality in Hispanics, American Indians, and non-Hispanic Whites in New Mexico. *Circulation*. 78: 302-309.
- Beckett, J.
1958a *A Study of a Mixed-Blood Minority in the Pastoral West of New South Wales*. An unpublished MA Thesis. Canberra: The Australian National University.
- 1958b Marginal men: a study of two half caste Aborigines. *Oceania*. 29: 91-108.
- 1964 Aborigines, Alcohol, and Assimilation. In *Aborigines Now*. M. Read, ed. Pp. 32-47. Sydney: Angus and Robertson.

- 1988 Kinship, mobility, and community in rural New South Wales. In *Being Black. Aboriginal Cultures in 'Settled' Australia*. I Keen, ed. Pp. 117-136. Canberra: Aboriginal Studies Press.
- Bell, J.H.
1964 Assimilation in New South Wales. In *Aborigines Now*. M. Reay, ed. Pp. 59-71. Sydney: Angus and Robertson.
- Bennett, P.H.
1983 Diabetes in developing countries and unusual populations. In *Diabetes in Epidemiological Perspective*. J. Mann, K. Pyörälä, and A. Teusher, eds. Pp. 43-57. Edinburgh: Churchill Livingstone.
- Bennett, P.H., and W.C. Knowler
1980 Increasing prevalence of diabetes in the Pima (American) Indians over a ten year period. In *Diabetes 1979*. W.K. Walddhäusl, ed. Pp. 507-511. Amsterdam: Excerpta Medica.
- Bingham, S.A.
1987 The dietary assessment of individuals; methods, accuracy, new techniques and recommendations. *Nutrition Abstracts and Reviews (Series A)*. 57: 705.
- Birkbeck, J.A.
1977 *New Zealanders and Their Diet*. A Report to the National Heart Foundation of New Zealand on the National Diet Survey 1977.
- Böttiger, L.E., and L.A. Carlson
1980 Risk factors for ischaemic vascular death for men in Stockholm Prospective Study. *Arteriosclerosis*. 36: 389-408.
- Brand, J.C.
1978 *Lactose malabsorption and its relation to human growth and nutrition*. An unpublished Ph.D. thesis. Sydney: University of Sydney.
- Brand J.C., and V. Cherikoff
1985 Australian Aboriginal bushfoods: the nutritional composition of plants from arid and semi-arid areas. *Australian Aboriginal Studies*. 2: 38-46.
- Brand, J.C., V. Cherikoff, and A.S. Truswell
1985 The nutritional composition of Australian Bushfoods. 3. seeds and nuts. *Food Technology in Australia*. 37: 275-279.
- Brand, J.C., C. Rae, J. McDonnell, A. Lee, V. Cherikoff, and A.S. Truswell
1983 The nutritional composition of Australian bushfoods 1. *Food Technology in Australia*. 35: 293-298.
- Bray, G.A.
1978 Definition, measurement and classification of the syndromes of obesity. *International Journal of Obesity*. 2: 99-112.
- 1985 Obesity: definition, diagnosis and disadvantages. *The Medical Journal of Australia Special Supplement*. 142: S2-S8.
- Brody, H.
1977 Alcohol, change and the industrial frontier. *Inuit Studies*. 1: 31-46.

- Bruhn, J.G., B. Chandler, M.C. Miller, S. Wolf, and T.N. Lynn
 1966 Social aspects of coronary heart disease in two adjacent, ethnically different communities. *American Journal of Public Health*. 56: 1493-1506.
- Cameron, W.I., P.S. Moffitt, and D.R.R. Williams
 1986 Diabetes mellitus in the Australian Aborigines of Bourke, New South Wales. *Diabetes Research and Clinical Practice*. 2: 307-314.
- Casley-Smith, J.R.
 1959 Blood pressures in Australian Aborigines. *The Medical Journal of Australia*. I: 627-633.
- Cass, B.
 1988 *Income Support for the Unemployed in Australia: Towards a More Active System*. *Social Security Review*. Canberra: Australian Government Publishing Service.
- Castle, R.
 1987 *Employment Prospects for Aborigines in Rural Labour Markets*. Report to the Australian Council for Employment and Training. Canberra: Australian Government Publishing Service.
- Castle, R., and J. Hagan
 1983 Seasonal work and Aboriginal employment in two rural area of New South Wales, 1921-1978. *Immigrants and Minorities*. 2: 117-134.
- Chandra, V. Szklo, M., R. Goldberg, and J. Tonascia
 1983 The impact of marital status on survival after an acute myocardial infarction: a population-based study. *American Journal of Epidemiology*. 117: 320-325.
- Charnock, J.S., J. Casey-Smith, and C.J. Schwartz
 1959 Serum magnesium-cholesterol relationships in the central Australian Aborigine and in Europeans with and without ischaemic heart disease. *Australian Journal of Experimental Biology*. 37: 509-516.
- Cherikoff, V., J.C. Brand, and A.S. Truswell
 1985 The nutritional composition of Australian Aboriginal bushfoods. 2. Animal foods. *Food Technology in Australia*. 37: 208-211.
- Chiang, C.L.
 1977 *Life Table and Mortality Analysis*. Geneva: World Health Organization.
- 1984 *The Life Table and Its Applications*. Malabar: Robert E. Krieger Publishing Company.
- Christian, C.M., M. Dufour, and D. Bertolucci
 1989 Differential alcohol-related mortality among American Indian tribes in Oklahoma, 1968-1978. *Social Science and Medicine*. 28: 275-284.
- Coolican, R.E.
 1971 *Morbidity in an Australian Rural Practice*. An unpublished M.D. Thesis. Sydney: University of New South Wales.

- 1974 The role of the general practitioner in a rural part-Aboriginal community. In *Better Health For Aborigines*. B. Hetzel, M. Dobbin, L. Lippmann, and E. Eggleston, eds. Pp. 127-132. St Lucia: University of Queensland Press.
- Corner, J.
1989 *dSURVEY User Manual*. Canberra: Helkorn Pty Ltd.
- Cowlishaw, G.
1988 *Black, White or Brindle. Race in rural Australia*. Melbourne: Cambridge University Press.
- Crimmins, E.M.
1981 The changing pattern of American mortality decline, 1940-77, and its implications for the future. *Population and Development Review*. 7: 229-254.
- Criqui, M.H.
1985 Alcohol and cardiovascular mortality. In *Behavioral Epidemiology and Disease Prevention*. R. Kaplan and M. Criqui, eds. Pp. 67-90. New York: Plenum Press.
- Cunneen, C., and T. Robb
1987 *Criminal Justice in North-West New South Wales*. Sydney: N.S.W. Bureau of Crime Statistics and Research.
- Dahl, L.K., and B.A. Love
1954 Evidence for relationship between sodium (chloride) intake and human essential hypertension. *Arch Intern Medicine* 94: 525-531.
- Dargin, P.
1976 *Aboriginal Fisheries of the Darling-Barwon Rivers*. Brewarrina Historical Society. Dubbo: Dubbo Printing Works.
- Dasvarma, G.L.
1980 *Differential Mortality in Australia with special reference to the period 1970-72*. An unpublished Ph.D. Thesis. Canberra: The Australian National University.
- Davidson, S. Sir, R. Passmore, J.F. Brock, and A.S. Truswell
1979 *Human Nutrition and Dietetics*. Edinburgh: Churchill Livingstone.
- Dawber, T.R.
1980 Incidence of coronary heart disease, stroke and peripheral arterial disease. In *The Framingham Study: The Epidemiology of Atherosclerotic Diseases*. Dawber, T.R., ed. Pp. 59-75. Cambridge: Harvard University Press.
- Department of Health
1986 *National Dietary Survey of Adults: 1983, No. 1 Foods Consumed*. Canberra: Australian Government Publishing Service.
- Department of Community Services and Health
1987 *National Dietary Survey of Adults: 1983, No. 2 Nutrient Intakes*. Canberra: Australian Government Publishing Service.
- 1988 *National Dietary Survey of Adults: 1983, No. 3 Nutrient intakes by capital city*. Canberra: Australian Government Publishing Service.

- 1989 *Composition of Foods Australia 1989*. Canberra: Australian Government Publishing Service.
- 1990a *Composition of Foods Australia 1990. Volume 2 Cereal and Cereal Products*. Canberra: Australian Government Publishing Service.
- 1990b *Composition of Foods Australia 1990. Volume 3 Dairy Products, Eggs and Fish*. Canberra: Australian Government Publishing Service.
- Department of Health and Social Security
 1984 *Diet and Cardiovascular Disease*. Report on Health and Social Subjects 28. London: Committee on Medical Aspects of Food Policy.
- Department of Health and Social Security Medical Research Council
 1976 *Research on Obesity*. A Report of the DHSS/MRC Group. London: Her Majesty's Stationery Office.
- Department of Statistic
 1986 *New Zealand Life Tables 1980-82*. Wellington: Department of Statistics.
- Dimsdale, J.E., B.S. Alpert, and N. Schneidermann
 1986 Exercise as a moderator of cardiovascular reactivity. In *Handbook of Stress, Reactivity, and Cardiovascular Disease*. K. Matthews, S. Weiss, T. Detre, T. Dembroski, B. Falkner, S. Manuck, and R. Williams, eds. Pp. 365-384. New York: John Wiley and Sons.
- Divakaran-Brown, C., and M. Honari
 1990 Aboriginal mortality in South Australia. In *A Matter of Life and Death. Contemporary Aboriginal Mortality*. A. Gray, ed. Pp. 95-102. Canberra: Aboriginal Studies Press
- Dooley, J., and J. Rodrigues
 1984 Prevalence of diabetes mellitus and sequelae in Indians of eastern Manitoba. A paper presented at the 6th Circumpolar Health Conference, Alaska, 1984.
- Ducimetiere, P., E. Eschwege, L. Papoz, J.L. Richard, J.R. Caude, and G. Rosselin
 1980 Relationship of plasma insulin levels to the incidence of myocardial infarction and coronary heart disease mortality in middle-aged population. *Diabetologia*. 19: 205-210.
- Duff, W.
 1965 *The Indian History of British Columbia. Volume 1: The Impact of the White Man*. Anthropology in British Columbia, Memoir No. 5. Victoria: Provincial Museum of British Columbia.
- Duffy, P., H. Morris, and G. Nelson
 1981 Diabetes mellitus in the Torres Strait region. *The Medical Journal of Australia Special Supplement*. 1: 8-11.
- Dunt, D.R.
 1982 Recent mortality trends in the adult Australian population and its principal ethnic groupings. *Community Health Studies*. 6: 217-222.
- Durnin, J.V.G.A.
 1988 Diet and obesity. In *A Balanced Diet?* J. Dobbing, ed. Pp. 27-44. London: Springer-Verlag.

- Dwyer, T.
1988 Defining the study, measuring the study parameters. In *Research on Health in Human Populations. The Menzies Foundation Technical Report Number Three — 1988*. S. Leeder, ed. Pp. 77-86. Melbourne: Menzies Foundation.
- Eason, R.J., J. Pada, R. Wallace, A. Henry, and R. Thornton
1987 Changing patterns of hypertension, diabetes, obesity and diet among Melanesians and Micronesians in the Solomon Islands. *The Medical Journal of Australia*. 146: 465-473.
- Eaton, C.
1977 Diabetes, culture change, and acculturation: a biocultural analysis. *Medical Anthropology*. 1: 41-63.
- Eaton, S.B., M. Konner, and M. Shostak
1988 Stone agers in the fast lane: chronic degenerative diseases in evolutionary perspective. *The American Journal of Medicine*. 84: 739- 749.
- Edwards, C., and P. Read, eds.
1989 *The Lost Children*. Sydney: Doubleday.
- Edwards, L.
1970 Malnutrition and disease in pre-school Aboriginal children. in the Walgett are of New South Wales. *The Medical Journal of Australia*. 2: 1007-1012.
- Edwards, L.D., and L.J. Craddock
1973 Malnutrition and intellectual development a study of school-age Aboriginal children at Walgett N.S.W. *The Medical Journal of Australia*. 1: 880-884.
- Ekelund, L., W.L. Haskell, J.L. Johnson, F.S. Whaley, M.H. Criqui, and D.S. Sheps.
1988 Physical fitness as a predictor of cardiovascular mortality in asymptomatic American men. *The New England Journal of Medicine*. 319: 1379-1384.
- Epstein, L.H., and L.R. Jennings
1986 Smoking, stress, cardiovascular reactivity and coronary heart disease. In *Handbook of Stress, Reactivity, and Cardiovascular Disease*. K. Matthews, S. Weiss, T. Detre, T. Dembroski, B. Falkner, S. Manuck, and R. Williams, eds. Pp. 291-309. New York: John Wiley and Sons.
- Fink, R.
1955 Social Stratification: A Sequel to the Assimilation Process in a Part-Aboriginal Community. An unpublished M.A. Thesis. Sydney: University of Sydney.
- 1957 The cast barrier — an obstacle to the assimilation of part-Aborigines in north-west New South Wales. *Oceania*. 28: 100-112.
- Finlay-Jones, R.A., and M.J. McComish
1972 Prevalence of diabetes mellitus in Aboriginal lepers. The Derby survey. *The Medical Journal of Australia*. 2: 135-137.

- Fisher, A.D.
1987 Alcoholism and race: the misapplication of both concepts to North American Indians. *Canadian Review of Sociology and Anthropology*. 24: 81-98.
- Fisher, V.J., and F. Favaler
1975 The action of ethanol upon the action potential and contraction of ventricular muscle. *Recent Advances in Studies on Cardiac Structure and Metabolism*. 5: 415-422.
- Fitzgerald, T.K.
1986 Dietary changes among Cook Islanders in New Zealand. In *Shared wealth and symbol. Food, culture, and society in Oceania and Southeast Asia*. L. Manderson, ed. Pp. 67-86. Cambridge: Cambridge University Press.
- Fuller, J.H.
1985 Causes of death in diabetes mellitus. In *Macrovascular Disease in Diabetes Mellitus. Pathogenesis and Prevention*. E. Pfeiffer, and M. Lipsett, eds. Pp. 3-9. New York: George Thieme Verlag Stuttgart.
- Garcia, M.J., P.M. McNamara, T. Gordon, and W.B. Kannel
1974 Morbidity and mortality in diabetics in the Framingham population. Sixteen year follow-up study. *Diabetes*. 23: 105-111.
- Garcia-Palmieri, M.R., J. Tillotson E. Cordero R. Costas, P. Sorlie, T. Gordon, W.B. Kannel, and A.A. Colon
1977 Nutrient intake and serum lipids in urban and rural Puerto Rican men. *American Journal of Clinical Nutrition*. 30: 2092-2100.
- Garnick, M.B., P.H. Bennett, and T. Langer
1979 Low density lipoproteins metabolism and lipoprotein cholesterol content in southwestern Indians. *Journal of Lipids Research*. 20: 31-39.
- Gillon, A., and E. Knight
1986 *Bourke Bush Foods. Traditional Foods of Bourke Aborigines*. Dubbo: Development and Advisory Publications of N.S.W.
- Goldberg, H.I., and C.W. Warren
1990 Health risk behaviors in five American Indian populations [Abstract]. Paper presented at the 1990 Annual Meeting of the Population Association of American, Toronto Canada.
- Goodall, H.
1982 *A History of Aboriginal Communities in New South Wales, 1909-1939*. An unpublished Ph.D. Thesis. Sydney: University of Sydney.
- Gould, L., C.V. Reddy, W.H. Becker, K.H. Oh, and S.G. Kim
1978 Electrophysiologic properties of alcohol in man. *Journal of Electrocardiology*. 11: 219-226.
- Gracey, M.
1986 The nutrition of Australian Aborigines. In *Recent Advances in Clinical Nutrition: 2*. M. Wahlqvist and A. Truswell, eds. Pp. 57-68. London: John Libbey.

- Gracey, M., and R.M. Spargo
 1987 The state of health of Aborigines in the Kimberley Region. *The Medical Journal of Australia*. 146: 200-204.
- Graham, P., R. Jackson, R. Beaglehole, and G. de Boer
 1989 The validity of Maori mortality statistics. *The New Zealand Medical Journal*. 102: 124-126.
- Gray, A.
 1983 *A Baseline Study of the Demography of Aboriginal People in New South Wales*. Sydney: New South Wales Department of Health.
- 1984a Aboriginal marriage and survival. *Journal of the Australian Population Association*. 1: 18-29.
- 1984b Changes in aboriginal family formation and fertility. In *Family Formation, Structure, Values, Proceedings of Australian Family Research Conference: Volume 1*. Canberra: The Australian National University.
- 1985 Some myths in the demography of Aboriginal Australians. *Journal of the Australian Population Association*. 2: 136-149.
- 1986 An introduction to Aboriginal health statistics. In *Aboriginal Health Statistics*. N. Thomson, ed. Pp. 4-13. Canberra: Australian Institute of Health.
- 1987a *The 'death bird' aspects of adult Aboriginal mortality*. Aboriginal Family Demography Study Working Paper No. 7. Canberra: The Australian National University.
- 1987b *Need a New House*. Aboriginal Family Demography Study Working Paper No. 4. Canberra: The Australian National University.
- 1990a Australian Aboriginal Families and the Health Care System. In *What We Know About Health Transition: The Cultural, Social and Behavioural Determinants of Health*. Canberra: J. Caldwell, S. Findley, P. Caldwell, G. Santow, W. Cosford, J. Braid, D. Broers-Freeman, eds. Pp. 378-399. Canberra: The Australian National University.
- 1990b National estimates of Aboriginal mortality. In *A Matter of Life and Death. Contemporary Aboriginal Mortality*. A. Gray, ed. Pp. 147-158. Canberra: Aboriginal Studies Press.
- Gray, A., and R. Hogg
 1989 *Mortality of Aboriginal Australians in western New South Wales 1984-1987*. Sydney: New South Wales Department of Health.
- Gray, A., and J. Vesper
 1987 *Housing at Gunderimba*. Aboriginal Family Demography Study Working Paper No. 1. Canberra: The Australian National University.
- Grim, C.E., F.C. Luft, J.Z. Miller, G.R. Meneely, H.D. Batterbee, C.G. Hames, and L.K. Dahl
 1980 Racial differences in blood pressure in Evans County, Georgia. *Journal of Chronic Diseases*. 33: 87-94.

- Harris, M., and M. Davies
1984 *Health and Housing for Aboriginal People in Bourke*. An unpublished report based on a survey conducted by Matt Davies and Jenny Edwards in February/March 1984.
- Harris, M., B. Nolan, and A. Davidson
1984 Early childhood pneumonia in Aborigines of Bourke, New South Wales. *The Medical Journal of Australia*. 140: 705-707.
- Health and Welfare Canada
1988 *Health Indicators Derived from Vital Statistics for Status Indian and Canadian Population 1978-1986*. Ottawa: Health and Welfare Canada.
- Health Department of Western Australia
1985 *Aboriginal Morbidity and Mortality in Western Australia*. Perth: Health Department of Western Australia.
- Hetzel, B., and T. McMichael
1989 *The LS Factor: Lifestyle and Health*. Maryborough: Penguin Books.
- Heywood, P.F., and C.A. Zed
1977 Dietary and anthropometric assessment of the nutritional status of Aboriginal and White school children in Walgett, N.S.W. *Proceedings of the Nutrition Society of Australia*. 2: 21-27.
- Hicks, D.G.
1990 Aboriginal mortality rates in Western Australia. In *A Matter of Life and Death. Contemporary Aboriginal Mortality*. A. Gray, ed. Pp. 71-76. Canberra: Aboriginal Studies Press
- Hillary Commission
1990 *Lifestyle in New Zealand. A Survey of Lifestyles of New Zealanders. Access to Results*. Wellington: University of Otago.
- Hitchcock, N.E., and M. Gracey
1975 Dietary patterns in a rural Aboriginal community in south-west Australia. *The Medical Journal of Australia Special Supplement*. 2: 12-16.
- Honari, M.
1990 Causes of Aboriginal mortality. In *A Matter of Life and Death. Contemporary Aboriginal Mortality*. A. Gray, ed. Pp. 139-146. Canberra: Aboriginal Studies Press.
- Howell, N.
1979 *Demography of the Dobe !Kung*. New York: Academic Press.
- Hugo, G.
1986 *Australia's Changing Population Trends and Implications*. Canberra: Commonwealth Department of Immigration and Ethnic Affairs.
- Indian and Inuit Health Services, Medical Services Branch
1985 *Issues For Health Promotion in Family and Child Health — A Source Book*. Ottawa: Health and Welfare Canada.

- Indian and Northern Affairs Canada
1988 *Basic Departmental Data*. Ottawa: Indian and Northern Affairs Canada.
- James, K.W.
1983 Analysis of indigenous Australian foods. *Food Technology in Australia*. 35: 342-343.
- James, R., C. Tyler, and D. Henrikson
1988 An evaluation of the accuracy of the Reflotron system in the field. *The Medical Journal of Australia*. 149: 130-131.
- Jarret, R.J.
1985 Risk factors of macrovascular disease in diabetes mellitus. In *Macrovascular Disease in Diabetes Mellitus*. E. Pfeiffer, and M. Lipsett, eds. Pp. 1-3. New York: George Thieme Verlag Stuttgart.
- Jarvis, G.K., and M. Boldt
1980 *Death Styles Among Canada's Indians*. Discussion Paper No. 24. Edmonton: Population Research Laboratory, the University of Alberta.
- Julienne, A., L. Smith, N. Thomson, and A. Gray
1983 Summary of Aboriginal mortality in New South Wales country regions, 1980-1981. *Health Services Information Bulletin No. 2*. Sydney: New South Wales Department of Health.
- Kamien, M.
1975a *The Doctor as an Agent of Change: An Action Oriented Epidemiological and Sociological Study of the Health of a Rural Aboriginal Community*. An unpublished Ph.D. thesis. Sydney: University of New South Wales.
- 1975b Aboriginal and alcohol intake, effects and social implications in rural community in western New South Wales. *The Medical Journal of Australia*. 1: 291-298.
- 1975c Cultural chasm and chaos in the health care services to Aborigines in rural New South Wales. *The Medical Journal of Australia Special Supplement*. 2: 6-11.
- 1976 The physical health of Aboriginal adults in Bourke, New South Wales. *The Medical Journal of Australia Special Supplement*. 1: 38-44.
- Kamien, M., S. Nobile, P. Cameron, and P. Rosevear
1974 Vitamin and nutritional status of a part Aboriginal community. *Australian and New Zealand Journal of Medicine*. 4: 126-137.
- Kamien, M., J.M. Woodhill, S. Nobile, P. Rosevear, P. Cameron, and J.M. Winston
1975a Nutrition in the Australian aborigine dietary survey of two Aboriginal families. *Food Technology in Australia*. 27: 93-103.
- Kamien, M., J.M. Woodhill, S. Nobile, P. Cameron, and P. Rosevear
1975b Nutrition in Australian Aborigines — effects of the fortification of white flour. *Australian and New Zealand Journal of Medicine*. 5: 123-133.

- Kannel, W.B., W.P. Castelli, T. Gordon, and P.M. MacNamara
1971 Serum cholesterol, lipoproteins and the risk of coronary heart disease: The Framingham Study. *Annals of Internal Medicine*. 74: 1-12.
- Kannel, W.B., and T. Gordon, eds.
1970 *The Framingham study. An epidemiological investigation of cardiovascular disease. Section 26: 16-year follow-up*. Washington D.C.: US Government Printing Office.
- Kannel, W.B., and T. Gordon
1974 Obesity and cardiovascular disease: the Framingham study. In *Obesity*. W. Barland, P. Sameul, and J. Yudkin, eds. Pp. 24-51. London: Churchill Livingstone.
- Kannel, W.B., M.C. Hjartland, P.M. McNamara, and T. Gordon
1976 Menopause and risk of cardiovascular disease: the Framingham study. *Annals of Internal Medicine*. 85: 447-452.
- Kay, R.M., A.I. Sabry, and A. Csima
1980 Multivariate analysis of diet and serum lipids in normal men. *American Journal of Clinical Nutrition*. 33: 2566-2572.
- Keen, I.
1988 Introduction. *Being Black. Aboriginal Culture in 'Settled' Australia*. I. Keen, ed. Pp. 1-26. Canberra: Aboriginal Studies Press.
- Kenen, R.H.
1987 Health status: Australian Aborigines and Native Americans — a comparison. *Australian Aboriginal Studies*. 1: 34-45.
- Kenen, R., and C.R. Hammerslough
1987 Reservation and non-reservation American mortality in 1970 and 1978. *Social Biology*. 34: 26-36.
- Kessler, I.L.
1971 Mortality experience of diabetic patients. A twenty-six year follow-up study. *American Journal of Medicine*. 51: 715-724.
- Keys, A.
1952 The cholesterol problem. *Voeding*. 13: 539-555.
1970 Heart disease in seven countries. *Circulation*. 41: Supplement I1-I211.
- Keys, A., J.T. Anderson, and F. Grande
1965 Serum cholesterol response to changes in the diet. IV. Particular fatty acids in the diet. *Metabolism*. 14: 776-787.
- Keys, A., and R.W. Parlin
1966 Serum-cholesterol response to changes in dietary lipids. *American Journal of Clinical Nutrition*. 19: 175-181.
- Khalidi, N.A.
1989 *The Aboriginal Population of Alice Springs: A Demographic Study*. An unpublished Ph.D. Thesis. Canberra: The Australian National University.

- Khaw, K.T., and E. Barrett-Connor
1987 Dietary potassium and stroke-associated mortality. *New England Journal of Medicine*. 316: 235-240.
- Khaw, K.T., and S. Thom
1982 Randomised double-blind cross-over trial of potassium on blood pressure in normal subjects. *Lancet*. 2: 1127-1129.
- King, H., P. Zimmet, R. Raper, and B. Balkau
1984 Risk factors for diabetes in three Pacific populations. *American Journal of Epidemiology*. 119: 396-409.
- Kinlay, S.
1988 Comparison of Reflotron and laboratory cholesterol measurements. *The Medical Journal of Australia*. 149: 126-129.
- Kitagawa, E.M., and P.M. Hauser
1973 *Differential Mortality in the United States: A Study in Socioeconomic Epidemiology*. Cambridge: Harvard University Press.
- Kitaoji, Y.
1976 *Family and Social Structure Among Aborigines In Northern New South Wales*. An unpublished Ph.D. Thesis. Canberra: The Australian National University.
- Klatsky, A.L., G.D. Friedman, A.B. Siegelau, and M.J. Gérard
1977 Alcohol consumption and blood pressure: Kaiser-Permanente multiphasic health examination data. *New England Journal of Medicine*. 296: 1194-1200.
- Knowler, W.C., D.J. Pettitt, P.H. Bennett, and R.C. Williams
1983 Diabetes mellitus in the Pima Indians: genetic and evolutionary considerations. *American Journal of Physical Anthropology*. 62: 107-114.
- Knowler, W.C., D.J. Pettitt, P.J. Savage, and P.H. Bennett
1981 Diabetes in Pima Indians: contributions of obesity and parental diabetes. *American Journal of Epidemiology*. 113: 144-156.
- Knowler, W.C., P.J. Savage, M. Nagulesparan, B.W. Howar, B.W. D.J. Pettitt, J.R. Lisse, S.L. Aronoff, and P.H. Bennett
1982 Obesity, insulin resistance and diabetes mellitus in the Pima Indians. In *The Genetics of Diabetes Mellitus*. J. Köbberling and R. Tattersall, eds. Pp. 243-250. London: Academic Press.
- Kromhout, D.
1985 Dietary components and the risk of coronary heart disease mortality. In *Behavioral Epidemiology and Disease Prevention*. R. Kaplan, and M. Criqui, eds. Pp. 121-134. New York: Plenum Press.
- Kromhout, D., E.B. Bosschieter, and C. Lezenne Coulander
1982 Dietary fibre and 10-year mortality from coronary heart disease, cancer and all causes. (The Zutphen Study). *Lancet*. 2: 518-522.
- Kunitz, S.J.
1983 *Disease Change and the Role of Medicine: The Navajo Experience*. Berkeley: University of California Press.

- 1990a The impact of public policy and health care on the mortality
(In Press) experience of indigenous populations in Northern America and Australasia. *Population and Development Review*. 16: [no page numbers].
- 1990b *Disease and the Destruction of Indigenous Populations*. National Centre For Epidemiology and Population Health Working Paper No. 15. Canberra: The Australian National University.
- Kunitz, S.J., J. Levy, C. Odoroff, and J. Bollinger
1971 The epidemiology of alcoholic cirrhosis in two southwestern Indian tribes. *Quarterly Journal of Studies in Alcohol*. 32: 706-720.
- Lee, J.F.
1984 The effects of a smoking prevention program for Alaskan youth. Paper presented at sixth Circumpolar Health Conference, Alaska.
- Lew, E.A., and L. Garfinkel
1979 Variations in mortality by weight among 750,000 men and women. *Journal of Chronic Diseases*. 32: 563-576.
- Lillioja, S., and C. Bogardus
1988 Insulin resistance in Pima Indians. *Acta Med Scand, Supplement*. 723: 103-119.
- Lincoln, R.A., J.M. Najman, P.R. Wilson, and C.E. Matis
1983 Mortality rates in 14 Queensland Aboriginal reserve communities. *The Medical Journal of Australia*. 1: 357-360.
- Long, J.P.M.
1970 *Aboriginal Settlements: A Survey of Institutional Communities in Eastern Australia*. Canberra: The Australian National University Press.
- Lynch, J.J.
1977 *The Broken Heart: The Medical Consequences of Loneliness*. New York: Basic.
- MacGregor, G.A.
1988 Salt and blood pressure. In *A Balanced Diet?* J. Dobbing, ed. Pp. 77-92. London: Springer-Verlag.
- Mao, Y., H. Morrison, R. Semenciw, and D. Wigle
1986 Mortality on Canadian Indian reserves, 1977-1982. *Canadian Journal of Public Health*. 77: 263-268.
- Marmot, M.G., and L.S. Syme
1976 Acculturation and coronary heart disease in Japanese-Americans. *American Journal of Epidemiology*. 104: 225-247.
- Martin, F.I.R., G.B. Wyatt, A.R. Griew, M. Haurahelia, and L. Higginbotham
1980 Diabetes mellitus in urban and rural communities in Papua New Guinea. Studies of prevalence and plasma insulin. *Diabetologia*. 18: 369-374.
- Mathews, J.
1977 *The Two Worlds of Jimmie Barker*. Canberra: Australian Institute of Aboriginal Studies.

- Matthews, K.A.
 1986 Summary, conclusions, and implications. In *Handbook of Stress, Reactivity, And Cardiovascular Disease*. K. Matthews, S. Weiss, T. Detre, T. Dembroske, B. Falkner, S. Manuck, R. Williams, eds. Pp. 461-473. New York: John Wiley and Sons.
- Mattson, F.H., and S.M. Grundy
 1985 Comparison of effects of dietary saturated, monounsaturated, and polyunsaturated fatty acids on plasma lipids and lipoproteins in man. *Journal of Lipid Research*. 26: 194-202.
- McCarron, D.A., C.D. Morris, and C. Cole
 1982 Dietary calcium in human hypertension. *Science*. 217: 267-269.
- McGarvey, S.T.
 1980 *Modernization and Cardiovascular Disease Among Samoans*. An unpublished thesis in Anthropology. College: The Pennsylvania State University.
- McIntyre, L., and C.P. Shah
 1986 Prevalence of hypertension, obesity and smoking in three Indian communities in northwestern Ontario. *Canadian Medical Association Journal*. 134: 345-349.
- Millar, B.D., and T.C. Beard
 1988 Avoidance of dietary sodium — a simple questionnaire. *The Medical Journal of Australia*. 149: 190-192.
- Millar, W.J., and D.T. Wigle
 1986 Socioeconomic disparities in risk factors for cardiovascular disease. *Canadian Medical Association Journal*. 134: 127-132.
- Miller, C.J., and N.E. Miller
 1975 Plasma high-density lipoprotein concentration and development of ischaemic heart disease. *Lancet*. 1: 16-19.
- Mitchell, J.D., J. Brand, and J. Halbisch
 1977 Weight-gain inhibition by lactose in Australian Aboriginal children: a controlled trial of normal and lactose hydrolysed milk. *Lancet*. 1: 500-502.
- Moodie, P.M.
 1973 *Aboriginal Health*. Canberra: The Australian National University.
- Mouratoff, G.J., N.V. Carroll, and E.M. Scott
 1967 Diabetes mellitus in Eskimos. *Journal of American Medical Association*. 199: 107-112.
- 1969 Diabetes mellitus in Athabaskan Indians in Alaska. *Diabetes*. 18: 29-32.
- Morris, J.N., J.W. Marr, and D.G. Clayton
 1977 Diet and heart a postscript. *British Medical Journal*. 2: 1307-1314.
- Muir, B.L.
 1988 *Health Status of Canadian Indians and Inuit update 1987*. Ottawa: Community Health Services, Indian and Northern Health Services, Medical Services Branch, Health and Welfare Canada.

- National Aboriginal Health Strategy Working Party
1989 *A National Aboriginal Health Strategy*. [No publication details provided].
- National Academy of Sciences
1980 *Recommended Dietary Allowances, ninth edition*. Washington: National Academy of Sciences.
- National Advisory Committee on Nutrition Education
1983 *A discussion paper on proposals for nutritional guidelines for health education in Britain*. London: The Health Education Council.
- National Health and Medical Research Council
1984 *Report of the working party on sodium in the Australian diet*. Canberra: Australian Government Publishing Service.

1987 *Recommended Dietary Intakes for use In Australia*. Canberra: Australian Government Publishing Service.
- National Heart Foundation
1985 *Risk Factor Prevalence Study, Report No. 2 — 1983*. Canberra: National Heart Foundation.
- Neel, J.V.
1962 Diabetes mellitus: A "thrifty" genotype rendered detrimental by "progress"? *American Journal of Human Genetics*. 14: 353-362.

1982 The thrifty genotype revisited. In *The Genetics of Diabetes Mellitus*. J. Köbberling and R. Tattersall, eds. Pp. 283-293. New York: Academic Press
- New South Wales, Ministry of Aboriginal Affairs
1987 *The Bourke Disturbances of August 1986*. Final report of the Consultative Committee. Sydney: Ministry of Aboriginal Affairs.
- New South Wales Health Commission, Aboriginal Policy Committee
1979 *Working paper No. 2: Aboriginal mortality in country areas of New South Wales, 1978, 1979*. Sydney: New South Wales Health Commission.
- Nichols, A.B., C. Ravenscroft, D.E. Lamphiear, and L.D. Ostrander
1976 Independence of serum lipid levels and dietary habits. The Tecumseh Study. *Journal of the American Medical Association*. 236: 1948-1953.
- Nobile, S.
1974 Blood vitamin levels in Aboriginal children and their mothers in western New South Wales. *The Medical Journal of Australia*. 1: 601-607.
- Obermann, A., N.E. Lane, W.R. Harlan, A. Graybiel, and R.E. Mitchell
1967 Trends in systolic blood pressure in the thousand aviator cohort over a twenty-four-year period. *Circulation*. 36: 812-822.
- O'Dea, K.
1984 Marked improvement in carbohydrate and lipid metabolism in diabetic Australian Aborigines after temporary reversion to traditional diet. *Diabetes*. 33: 596-603.

- O'Dea, K., R.M. Spargo, and P.J. Nestel
1982 Impact of westernization on carbohydrate and lipid metabolism in Australian Aborigines. *Diabetologia*. 22: 148-153.
- O'Dea, K., N.G. White, and A.J. Sinclair.
1988 An investigation of nutrition-related risk factors in an isolated Aboriginal community in Northern Australia: advantages of a traditionally-orientated life-style. *The Medical Journal of Australia*. 148: 177-180.
- Oliver, W.J., L.C. Edwin, and J.V. Neel
1975 Blood pressures, sodium intake, and sodium related hormones in the Yanomamo Indians, a "no-salt" culture. *Circulation*. 52: 146-151.
- Omran, A.R.
1971 The epidemiologic transition: a theory of the epidemiology of population change. *Milbank Memorial Fund Quarterly*. 49: 509-538.
- Pain, R.W.
1988 Are your laboratory's cholesterol results reliable enough? *The Medical Journal of Australia*. 149: 113-115.
- Parkinson, J.
1982 *An Exercise in Perspective: A Black History of Bourke*. An unpublished honours paper. Canberra: The Australian National University.
- Parliament of the Commonwealth of Australia
1979 *Aboriginal Health. House of Representatives Standing Committee on Aboriginal Affairs*. Canberra: Australian Government Publishing Service.
- Passel, J.S., and P.A. Berman
1986 Quality of 1980 census data for American Indians. *Social Biology*. 33: 163-182.
- Patrick, R.C., I.A.M. Prior, J.C. Smith, and A.H. Smith
1983 Relationship between blood pressure and modernity among Ponapeans. *International Journal of Epidemiology*. 12: 36-44.
- Pell, S., and C.A. d'Alonzo
1970 Factors associated with long-term survival of diabetics. *Journal of American Medical Association*. 214: 1833-1840.
- Perkins, C.
1976 *Alcohol and Drug Problem among Aborigines*. Canberra: National Alcohol and drug Dependency Multidisciplinary Institute.
- Péron, Y., and Strohmenger, C.
1985 *Demographic and Health Indicators. Presentation and Interpretation*. Ottawa: Statistics Canada, Health Division, Research and Analysis Section.
- Phillips, S., L. Wyndham, J. Shaw, and S.F. Walker
1988 How accurately does the Reflotron dry-chemistry system measure plasma total cholesterol levels when used as a community screening device? *The Medical Journal of Australia*. 149: 122-125.

- Plant, A.J.
1988 *Aboriginal Mortality in the Northern Territory, 1977-1983*. An unpublished Master of Public Health Thesis. Sydney: University of Sydney.
- Pointing, J.R., and R. Gibbons
1980 *Out of Irrelevance: A Socio-Political Introduction to Indian Affairs in Canada*. Toronto: Butterworth.
- Pomare, E.W., and G.M. de Boer
1988 *Hauora Maori Standards of Health. A study of the years 1970-1984*. Special Report Series 78. Wellington: National Health Statistic Centre.
- Pool, I.
1983 Changing patterns of sex differentials in survival: an examination of data for Maoris and non-Maoris in New Zealand. In *Sex Differentials in Mortality: Trends, Determinants, and Consequences*. A. Lopez and L. Ruzicka, eds. Pp. 193-220. Canberra: Department of Demography, The Australian National University.
- Prior, I.A.M., R. Beaglehole, F. Davidson, and C.E. Salmond
1978 The relationship of diabetes, blood lipids and uric acid levels in Polynesians. In *Advances in Metabolic Disorders*. P. Bennett, and M. Miller, eds. Pp.241-261. New York: Academic Press.
- Prior, I.A.M., and F. Davidson
1966 The epidemiology of diabetes in Polynesians and Europeans in New Zealand and the Pacific. *New Zealand Medical Association Journal*. 65: 375-383.
- Prior, I., and C. Tasman-Jones
1981 New Zealand Maori and Pacific Polynesians. In *Western Diseases: their emergence and prevention*. H. Trowell and D. Burkitt, eds. Pp. 227-267. London: Edward Arnold.
- Pyörälä, K.
1979 Relationship of glucose tolerance and plasma insulin to the incidence of coronary heart disease: results from two population studies in Finland. *Diabetes Care*. 2: 131-141.
- Rate, R.G., W.C. Knowler, H.G. Morse, M.D. Bonnell, J. McVey, C.L. Chervenak, M.G. Smith, and G. Pavnich
1983 Diabetes mellitus in Hopi and Navajo Indians. Prevalence of microvascular complications. *Diabetes*. 32: 894-899.
- Read, P.
n.d. *The Stolen Generations: The Removal of Aboriginal Children in New South Wales 1883 to 1969*. New South Wales Ministry of Aboriginal Affairs Occasional Paper No. 1.
- 1988 *A Hundred Years War*. Canberra: The Australian National University Press.
- Reay, M.
1945 A half-caste Aboriginal community in north-western New South Wales. *Oceania* 15: 296-323.

- 1949 Native thought in rural New South Wales. *Oceania*. 15 20: 89-118.
- Reay M., and G. Sitlington
1948 Class and status in a mixed-blood community. *Oceania*. 18: 179-207.
- Reid, J., and C. Kerr
1983 Trends in Aboriginal mortality. *The Medical Journal of Australia*. 1: 348-350.
- Reid, J.M., S.D. Fullmer, K.D. Pettigrew, T.A. Burch, P.H. Bennett, M. Miller, and G.D. Whedon
1971 Nutrient intake of Pima Indian women: relationships to diabetes mellitus and gall bladder disease. *The American Journal of Clinical Nutrition*. 24: 1281-1289.
- Rhoades, E.R., A.J. D'Angelo, and W.B. Hurlburt
1987a The Indian Health Service record of achievement. *Public Health Reports*. 102: 356-360.
- Rhoades, E.R., J. Hammond, T.K. Welty, A.O. Handler, and R.W. Amler
1987b The Indian burden of illness and future health interventions. *Public Health Reports*. 102: 361-368.
- Ringrose, H., and P. Zimmet
1979 Nutrient intake in an urbanized Micronesian population with a high diabetes prevalence. *The American Journal of Clinical Nutrition*. 32: 1334-1341.
- Rode, A., and R.J. Shepard
1984 Ten years of "civilization": fitness of Canadian Inuit. *Journal of Applied Physiology*. 56: 1472-1477.
- Rogers, R.G., and R. Hackenberg
1988 Extending epidemiologic theory: a new stage. *Social Biology*. 34: 234-243.
- Rose, G.A.
1962 The diagnosis of ischaemic heart pain and intermittent claudication in field surveys. *Bulletin of the World Health Organization* 27: 645-658.
- Ross, R.T.
1988 *The Labour Market Position of Aboriginal People in Non-metropolitan New South Wales*. Social Welfare Research Centre Discussion Paper No. 1. Kensington: University of New South Wales.
- Ross, R., and J.A. Glomset
1976 The pathogenesis of atherosclerosis. *The New England Journal of Medicine*. 297: 369-377, 420-425.
- Rowely, C.D.
1982 *Equality by Instalments: the Aboriginal householder in rural New South Wales, 1965 and 1980*. Canberra: Australian Institute of Aboriginal Studies.
- Rutishauser, I.H.E.
1988 Making measurements: diet. *Menzies Technical Report*. 3: 89-120.

- Sanders, T.A.B.
1988 Dietary fat and coronary heart disease. In *A balanced diet?* J. Dobbing, ed. Pp. 93-115. London: Springer-Verlag.
- Schmitt, N., L.W. Hole, and W. Barclay
1966 Accidental deaths among British Columbia Indians. *Canadian Medical Association Journal*. 94: 228-234.
- Schwartz, A., A.J. Day, and A. Peters
1957 Serum cholesterol and phospholipid levels of Australian Aborigines. *Australian Journal of Experimental Biology*. 35: 449-456.
- Shekelle, R.B., A. MacMillan Shyrock, O. Paul, M. Lepper, J. Stamler, S. Liu, and W.J. Raynor
1981 Diet serum cholesterol, and death from coronary heart disease. The Western Electric Study. *New England Journal of Medicine*. 304: 65-70.
- Shryock, H., and J. Siegel
1971 *The Methods and Materials of Demography*. Washington: U.S. Department of Commerce, Bureau of Census.
- Sibthorpe, B.
1988 *'All Our People Are Dyin'': Diet and Stress in an Urban Aboriginal Community*. An unpublished Ph.D. thesis. Canberra: The Australian National University.
- Sievers, M.L.
1968 Serum cholesterol levels in southwestern American Indians. *Journal of Chronic Diseases*. 21: 107-115.
- Simons, L., P. Whish, B. Marr, A. Jones, and J. Simons
1981 Coronary risk factors in a rural community which includes Aborigines: Inverell heart disease prevention programme. *Australian and New Zealand Journal of Medicine*. 11: 386-390.
- Sladden, T.J., P. Coster, P. Reaney, and A.J. Tidemann
1986 Prevalence of cardiovascular risk factors in an Aboriginal community. *Proceedings of the Nutrition Society of Australia*. 11: 104.
- Smith, A.H., and N.E. Pearce
1984 Determinants of differences in mortality between New Zealand Maoris and non-Maoris aged 15-64. *The New Zealand Medical Journal*. 97: 101-108.
- Smith, D.P.
1988 *User's Guide For Program SURVIVAL Version 2.03*. Houston: University of Texas School of Public Health.
- Smith, L., N. Thomson, and A. Gray
1983 *Aboriginal Mortality in NSW country regions 1980-1981*. Sydney: New South Wales Department of Health.
- Sobolski, J., M. Kornitzer, G. De Backer, M. Dramaix, Abramowicz, S. Degre, and H. Denolin
1987 Protection against Ischaemic heart disease in the Belgian Physical Fitness Study: physical fitness rather than physical activity? *American Journal of Epidemiology*. 125: 601-610.

South Australia

- 1987 *Report of Uwankara Palyanyku Kanyintjaku: An Environmental and Public Health Review within the Anangu Pitjantjatjara Lands.* Adelaide: Nganampa Health Council, South Australian Health Commission, and Aboriginal Health Organization of South Australia.
- Stanton, K.G., V. McCann, M. Knuiman, I.J. Constable, and T. Welborn,
1985 Diabetes in part-Aborigines of Western Australia. *Diabetologia*. 28: 16-21.
- Stenhouse, N.S., and M.G. McCall
1970 Differential mortality from cardiovascular disease in migrants from England and Wales, Scotland and Italy, and native-born Australians. *Journal of Chronic Diseases*. 23: 423-431.
- Stephenson, J., and P. Lenz
1990 *Cardiovascular Disease Risk Factor Screening, Wilcannia, October, 1989.* Orana and Far West Region, N.S.W. Health Report Number 5. Dubbo: Health Promotion Unit.
- Stratton, R.
1981 Relationship between prevalence of alcohol problems and socioeconomic conditions among Oklahoma native Americans. In *Currents in Alcoholism, Vol VIII*. M. Galanter, ed. Pp. 315-325. New York: Plenum Press.
- Strong, J.P.
1972 Atherosclerosis in human populations. *Atherosclerosis* 16: 193-200.
- Suarez, L., and E. Barrett-Conner
1984 Interactions between cigarette smoking and diabetes mellitus in the prediction of death attributed to cardiovascular disease. *American Journal of Epidemiology*. 120: 670-675.
- Sugarman, J.R., M. Hickey, T. Hall, and D. Gohdes
1990 The changing epidemiology of diabetes mellitus among Navajo Indians. *The Western Journal of Medicine*. 153: 140-145.
- Sullivan, H., M. Gracey, and V. Hevron
1987 Food costs and nutrition of Aborigines in remote areas of northern Australia. *The Medical Journal of Australia*. 147: 334-337.
- Sykes, B.
1986 Australia Day address. Canberra: National Press Club.
- Szathmary, J.E., and N. Holt
1983 Hyperglycaemia in Dogrib Indians of the Northwest Territories, Canada: association with age and a centripetal distribution of fat. *Human Biology*. 55: 493-515.
- Szathmary, J.E., C. Ritenbaugh, and C.M. Goodby
1987 Dietary change and plasma glucose levels in an amerindian population undergoing cultural transition. *Social Science and Medicine*. 24: 791-804.
- Task Force on Aboriginal Health Statistics
1985 *Towards a national system of Aboriginal health Statistics.* Canberra: Commonwealth Department of Health.

- Taylor, R., N.D. Lewis, and S. Levy
 1989 Societies in transition: mortality patterns in Pacific island populations. *International Journal of Epidemiology*. 18: 634-646.
- Taylor, R., and K. Thoma.
 1985 Mortality patterns in the modernized Pacific island of Nauru. *American Journal of Public Health*. 75: 149-155.
- Taylor, R., P. Zimmet, S. Levy, and V. Collins
 1985 Group comparisons of blood pressures and indices of obesity and salt intake in Pacific populations. *The Medical Journal of Australia*. 142: 499-501.
- Thomson, N.
 1984 Australian Aboriginal health and health care. *Social Science and Medicine*. 18: 939-948.
 1986 Recent developments in Aboriginal Health Statistics. In *Aboriginal Health Statistics*. N. Thomson, ed. Pp. 14-19. Canberra: Australian Institute of Health.
 1989 *Inequalities in Aboriginal health*. An unpublished Master of Public Health Thesis. Sydney: University of Sydney.
 1990 *Overview of Aboriginal health status*. Report to the Royal Commission into Aboriginal Deaths in Custody. Canberra: Australian Institute of Health.
- Thomson, N., F. Paden, and G. Cassidy
 1990 *Identification of Aborigines in hospital admissions in the North Coast Health Region, New South Wales*. Canberra: Australian Institute of Health and the North Coast Health Region of the New South Wales Department of Health.
- Thomson, N., and L. Smith
 1985 An analysis of Aboriginal mortality in NSW country regions, 1980-1981. *The Medical Journal of Australia Special Supplement* 143: S49- S64.
- Thorburn, A.W., J.C. Brand, and A.S. Truswell
 1987a Slowly digested and absorbed carbohydrate in traditional bushfoods: a protective factor against diabetes? *American Journal of Clinical Nutrition*. 45: 98-106.
- Thorburn, A.W., J.C. Brand, K. O'Dea, R.M. Spargo, and A.S. Truswell
 1987b Plasma glucose and insulin responses to starchy foods in Australian Aborigines: a population now at high risk of diabetes. *American Journal of Clinical Nutrition*. 46: 282-285.
- Thouez, J.P., A. Rannou, and P. Foggin
 1989 The other face of development: native population, health status and indicators of malnutrition — the case of the Cree and Inuit of northern Quebec. *Social Science and Medicine*. 29: 965-974.
- Trigger, D.S., C. Anderson, R.A. Lincoln, and C.E. Matis
 1983 Mortality rates in 14 Queensland Aboriginal reserve communities: association with 10 socioeconomic variables. *The Medical Journal of Australia*. 1: 361-365.

- Truett, J., J. Cornfield, and W. Kannel
 1967 A multivariate analysis of the risk of coronary heart disease in Framingham. *Journal of Chronic Diseases*. 20: 511-524.
- United Nations
 1983 *Manual X Indirect Techniques For Demographic Estimation*. New York: Department of International Economic and Social Affairs Population Studies.
- Urbano-Marquez, A., R. Estruch, F. Navarro-Lopez, J.M. Grau, L. Mont, and E. Rubin
 1989 The effects of alcoholism on skeletal and cardiac muscle. *The New England Journal of Medicine*. 320: 409-415.
- U.S. Department of Health and Human Services, Public Health Service, Indian Health Service
 n.d. *A Comprehensive Health Care Program for American Indians and Alaska Natives*. Washington: U.S. Government Printing Office.
- 1988 *Indian Health Service Chart Series Book April 1988*. Washington: U.S. Government Printing Office.
- Verbrugge, L.M.
 1983 The social role of the sexes and their relative health and mortality. In *Sex Differentials in Mortality: Trends, Determinants, and Consequences*. A. Lopez and L. Ruzicka, eds. Pp. 221-245. Canberra: Department of Demography, The Australian National University.
- Vital Events Staff, Program Statistics Branch, Evaluation and Legislation, Indian Health Service
 1984 *American Indian and Alaska Native Life Expectancy, 1979-1981 and Life Expectancy for Selected U.S. Populations by Race, 1980*. Rockville: Indian Health Service.
- Waldram, J.B.
 1985 Hydroelectric development and dietary delocalization in northern Manitoba, Canada. *Human Organization*. 44: 41-49.
- Waldron, I.
 1983 The role of genetic and biological factors in sex differences in mortality. In *Sex Differentials in Mortality: Trends, Determinants, and Consequences*. A. Lopez and L. Ruzicka, eds. Pp. 141-164. Canberra: Department of Demography, The Australian National University.
- Watson, C., J. Fleming, and K. Alexander
 1988 *Survey of Drug Use Patterns in Northern Territory Aboriginal Communities: 1986-1987*. Canberra: Department of Health and Community Services.
- Way, G.L., H.D. Ruttenberg, E. Eshaghpour, J.J. Nord, J.J., and R.R. Wolfe
 1976 Hypertrophic obstructive cardiomyopathy in infants of diabetic mothers. [Abstract 0411] *Circulation*. 54: 105. Supplement II.
- Weiss, K.M., R.E. Ferrell, and C.L. Hanis
 1984 A New World syndrome of metabolic diseases with a genetic and evolutionary basis. *Yearbook of Physical Anthropology*. 27: 157-178.

- Welborn, T.A., and K. Wearne
 1979 Coronary heart disease incidence and cardiovascular mortality in Bussleton with reference to glucose and insulin concentrations. *Diabetes Care*. 2: 154-160.
- West, K.M.
 1978 *Epidemiology of Diabetes and its Vascular Lesions*. New York: Elsevier.
- Wienblatt, E., W. Rubermann, G.D. Goldberg, C.W. Frank, S. Shapiro, and B.S. Chaudhary
 1978 Relation of education to sudden death after myocardial infarction. *New England Journal of Medicine*. 299: 60-65.
- Wilson, W.
 1953 A dietary survey of Aborigines in the Northern Territory. *The Medical Journal of Australia*. 2: 559-605.
- Wise, P.H., F.M. Edwards, D.W. Thomas, R.B. Elliot, L. Hatcher, and R. Craig
 1970 Hyperglycaemia in the urbanized aboriginal. The Davenport survey. *The Medical Journal of Australia*. 2: 1001-1006.
- Wise, P.H., F.M. Edwards, R.J. Craig, D. Evans, J.B. Murchland, B. Sutherland, and D.W. Thomas
 1976 Diabetes and associated variables in the South Australian Aboriginal. *Australian and New Zealand Journal of Medicine*. 6: 191-196.
- Williams, D.R.R., P.S. Moffitt, J.S., Fisher, and H.V. Bashir
 1987 Diabetes and glucose tolerance in New South Wales coastal Aborigines: possible effects of genetic admixture. *Diabetologia*. 30: 72-77.
- Wolfe, R.R., and G.L. Way
 1977 Cardiomyopathies in infants of diabetic mothers. *The John Hopkins Medical Journal*. 140: 177-180.
- Wood, C.S., and L.P. Gans
 1986 Contrasting patterns of blood pressure and related factors within a Maori and European population in New Zealand. *Social Science and Medicine*. 23: 439-444.
- Woodward, J.
 1989 *Native Law*. Toronto: Carswell.
- Woolf, B.
 1955 On estimating the relation between blood group and disease. *Annals of Human Genetics*. 19: 251-253.
- World Health Organization
 1959 Study group on atherosclerosis and ischaemic heart disease. *Technical Report Series No. 117*.
- 1977 *Manual of the International Statistical classification of diseases, injuries and causes of death, 1975 revision*. Geneva: World Health Organization.
- 1980 World Health Organization Expert Committee on Diabetes Mellitus, Second Report. *Technical Report Series*. No. 646.

- 1982 Prevention of coronary heart disease. Report of a WHO Expert Committee. *Technical Report Series*. No 678.
- World Health Organization, Health Education Unit
1986 Life-styles and health. *Social Sciences and Medicine*. 22: 117-124.
- World Health Organization / International Society of Hypertension
1986 Hypertension guidelines for the treatment of mild hypertension. *The Medical Journal of Australia*. 144: 575-577.
- Xyris Software
1989a *DIET / 1 Nutrient Calculation Software User's Guide*. Highgate Hill: Xyris Software.
- 1989b *DIET / 1 Nutrient Calculation Software Data Reference*. Highgate Hill: Xyris Software.
- Yano, K., D.M. Reed, and D.L. McGee
1984 Ten-year incidence of coronary heart disease in the Honolulu heart program. *American Journal of Epidemiology*. 119: 653-666.
- Young, C.M.
1986a *Selection and Survival. Immigration Mortality in Australia*. Department of Immigration and Ethnic Affairs, Adult Migrant Education Program. Canberra: Australian Government Publishing Service.
- 1986b Differential mortality of birthplace groups in Australia during 1980-82. *Journal of the Australian Population Association*. 3: 144-168.
- Young, E.A.
1982 Aboriginal town dwellers in New South Wales. In *The Aboriginal Component in the Australian Economy. Town Populations*. E. Young and E. Fisk, eds. Pp. 1-26. Canberra: Development Studies Centre, The Australian National University.
- Young, T.K.
1982 Self-perceived and clinically assessed health status of Indians in northwestern Ontario: analysis of a health survey. *Canadian Journal of Public Health*. 73: 272-277.
- 1983 Mortality pattern of isolated Indians in northwestern Ontario: a 10-year review. *Public Health Report*. 98: 467-475.
- Young, T.K., and G. Sevenhuysen
1989 Obesity in northern Canadian Indians: patterns determinants, and consequences. *American Journal of Clinical Nutrition*. 49: 786-793.
- Young, T.K., L.L. McIntyre, J. Dooley, and J. Rodriguez
1985 Epidemiological features of diabetes mellitus among Indians in northwestern Ontario and northeastern Manitoba. *Canadian Medical Association Journal*. 132: 793-797.
- Yusuf, F., and A. Hamilton
1982 Hospital morbidity among Aboriginal Australians in New South Wales. *Journal of Biosocial Science*. 14: 445-463.

- Zimmet, P.
1979 Epidemiology of diabetes and its macrovascular complications in Pacific populations: the medical effects of social progress. *Diabetes Care*. 2: 144-153.
- 1982 Type 2 (non-insulin-dependent) diabetes — an epidemiological overview. *Diabetologia*. 22: 399-411.
- Zimmet, P., S. Faaiuso, J. Ainuu, S. Whitehorse, B. Milne, and W. De Boer
1981 The prevalence of diabetes in the rural and urban Polynesian population of Western Samoa. *Diabetes*. 30: 45-51.
- Zimmet, P., P. Taft, A. Guinea, W. Guthrie, and K. Thoma
1977 The high prevalence of diabetes mellitus on a Central Pacific island. *Diabetologia*. 13: 111-115.
- Zimmet, P., and S. Whitehouse
1981 Pacific islands of Nauru, Tuvalu and Western Samoa. In *Western Diseases: their emergence and prevention*. H. Trowell and D. Burkitt, eds. Pp. 204-224. London: Edward Arnold.
- Zimmet, P., S. Whitehouse, and J. Kiss
1979 Ethnic variability in the plasma insulin response to oral glucose in Polynesian and Micronesian subjects. *Diabetes*. 28: 624-628.