"A Great Deal of Sickness"[1]

Introduced diseases among the Aboriginal People of colonial Southeast Australia 1788-1900

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[1] Buttfield 1874

Except where otherwise stated in the text, this thesis represents my own original work.

Peter J. Dowling

For Dianne

Saal na saol
Tús gá deiredh
Tá muid beo
Dá deo

Life of lives
Beginning without an end
We are alive
To the end of time

(Enya & Ryan 1987)

Abstract

Palaeopathological studies have sought to build up a picture of Australian Aboriginal health before European settlement in 1788, and epidemiological studies of Aboriginal health in the twentieth century are now legion. But, despite a growing body of literature on Aboriginal history in the intervening colonial period, this remains an under-studied period from the viewpoint specifically of Aboriginal health. This thesis is a contribution to filling that gap through an examination of documentary and skeletal evidence on the changing bio-medical situation experienced by Aboriginal populations of Southeast Australia from 1788 to 1900.

This thesis examines one of the major biological components of this change - the diseases that were introduced into Australian Aboriginal populations during the process of colonisation. The epidemiology, timing, diffusion of diseases are considered with specific attention given to infectious and respiratory diseases that were responsible for causing major epidemics of morbidity and mortality.

A medical model for the contact period in the late 18th and 19th centuries is proposed. This model considers three major stages in the disease environment of Aboriginal populations in Southeast Australia; a pre-contact stage with endemic pathogens causing chronic diseases and limited epidemics, an early contact stage where introduced exotic human diseases cause severe epidemics of infectious and respiratory diseases among Aboriginal populations, and a third stage where remaining Aboriginal populations were institutionalised on government and mission settlements and were subjected to a high level of mortality from the introduced diseases.

The major epidemic diseases during the early contact stage were smallpox, syphilis, tuberculosis, influenza, and measles. Each of these diseases were responsible for excessive morbidity and mortality. During the period of institutionalisation infectious and respiratory diseases were responsible for over 50% of recorded deaths on 8 separate Aboriginal settlements in Southeast Australia. The major diseases recorded as causes of death were tuberculosis, bronchitis, pneumonia, diarrhoea and dysentery.

Aboriginal and non-Aboriginal Australian infant mortality rates are calculated to provide an indicator to compare the state of health of the two populations. Aboriginal rates were high when compared to the non-Aboriginal populations of Victoria and South Australia. The rates reveal a substantial health differential between Aboriginal and non-Aboriginal populations. Aboriginal infant mortality has improved into the latter quarter of the twentieth century but the corresponding improvement in non-Aboriginal infant mortality has been at a much higher rate. The gap between the health status of each has widened rather than narrowed over the last one hundred years.

Acknowledgments

When I first began this project I was warned by several colleagues that finding nineteenth century information regarding the impact of introduced diseases on Aboriginal populations would be difficult. The sources of information I would need, if they existed at all I was warned, would most likely be scattered throughout archival texts in several capital cities and much of it would not be readily obtainable through current archival referencing aids. They were correct. Much of the material obtained came after seemingly endless days of searching through bundles of files, often without any success, in the libraries and public records offices of five capital cities. In order to find them, understand them, use them in the way I wished, cope with all the inevitable setbacks, and finally put them all together in what I hope is a coherent story required the help of many people.

At the top of the list is my wife Dianne. Once again she had to cope with this thing called a thesis which invaded her life just as much as it did mine. Without her understanding, help in the archives, and support throughout the good times and the bad times, this thesis would never have been written.

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Go raibh míle maith agat.

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CHAPTER ONE

Introduction

1.1 Introduction

This thesis begins an investigation and assessment of the disease environment of the Aboriginal people of Southeast Australia during the colonial period 1788 to 1900. Throughout, the discussion will centre around two propositions recurrent in biological anthropology. The first is that hunter-gatherers who have had little or no contact with the representatives of European industrial societies, their artefacts, ideas, and biology, tend to have a society which is well adapted to their environments. They tend to have levels of health that are able to sustain a state of population homeostasis with natality, mortality and morbidity not threatening its internal state. The second is a condition of rapid biological and social change. Almost immediately after such hunter-gatherer societies begin extensive and continuous contact with European colonisation, and experience sociocultural, medical and environmental changes, their relationships with their own environment is disrupted and their health is jeopardised (Wirsing 1985: 303). The causes of change are largely outside the control of the indigenous society, severe in their effects, and there is often little time for social adaptation. The consequence is an imbalance to the processes of natural and human control and the homeostatic state is lost.

The objective of this thesis is to examine one set of biological components of this change - the diseases that were introduced into Australia during the process of colonisation. The epidemiology, timing, and diffusion of diseases are considered with specific attention given to infectious and respiratory diseases that caused major epidemics of morbidity and mortality. This will be done mainly by examining historical documents from the colonial period of Southeast Australia and to a lesser extent skeletal indications of pathology.

1.2 Research to date

While many researchers have recognized the value of investigating the history of inter-racial contact in Australia, too few have sought to examine the nature of biological, and in particular health, changes that occurred following contact between Aboriginal societies and European colonists.

The first attempts were by Drs. J.B. Cleland (1928) and Herbert Basedow (1932). Cleland was then professor of pathology at the University of Adelaide and Basedow was Chief Medical Inspector, and for a short period Chief Protector of Aborigines, in Darwin. Both had made periodic exploratory surveys into central and northern Australia as naturalists and had medically examined Aboriginal people. Using historical sources, contemporary medical literature, and their own observations both wrote extensive reviews on diseases among Aboriginal communities. Both were concerned with the declining population of Aborigines as was a common preoccupation at the time, and sought to gain knowledge on the manifestation, symptoms and effects of introduced and endemic diseases among Aboriginal people with no genetic admixture with Europeans. Their coverage, while including many different diseases, was concentrated on populations in central and northern Australia, and temporally focused in the early twentieth century on Aboriginal communities who had had little contact with Europeans.

At the same time as Cleland and Basedow were writing on Aboriginal diseases, J.H.L. Cumpston was writing on the health and diseases in Australia (Cumpston 1989). His was a historically orientated, epidemiological review of diseases among the European population of Australia. Cumpston's focus was not on Aboriginal people but in several cases, for example when discussing smallpox, venereal diseases, and influenza, he briefly

considered them in the context of the geographical dissemination of infectious disease epidemics. His main work dealing with diseases among Aboriginal people was *The History of Smallpox in Australia, 1788-1908,* (Cumpston 1914) which brought together much of the historical literature dealing with three epidemics that affected the Aborigines and the cases of smallpox among Europeans. Hackett (1936a, 1936b, 1936c) followed with descriptions of treponemal infected bones, arguing that non-venereal syphilis and yaws were long established endemic diseases throughout much of Australia.

In 1975 Reynolds began to examine the history of race relations in Australia, a subject that had until then, been largely avoided by historians. His chapter 'Disease and Deprivation' in *Aborigines and Settlers,* was concerned with the diseases of contact. He acknowledged that exotic diseases caused severe mortality among Aboriginal populations all over the continent (Reynolds 1975: 71) and briefly covered the topics of smallpox, respiratory diseases, venereal diseases, malnutrition, mortality and morbidity, alcohol, and fringe-dwellers. He presented, verbatim, a selection of previously published discussions (often from secondary sources), and accounts by medical practitioners, missionaries, Aboriginal protectors, and government inquiries in the nineteenth century and early twentieth century. No analysis of the biomedical nature of these diseases was attempted.

Smallpox has continued to be the subject of most discussions and debates on the early diseases of contact, and has remained a major emphasis of the study of introduced diseases into Aboriginal Australia (Curson 1985: 41-53; Fenner 1984, 1985; Frost 1994; Reynolds 1981, 1987; Watt 1989; Webb 1995: 284-7; Wilson 1985). In 1983 Butlin published a controversial opinion on the origins and mortality of introduced diseases among the Aboriginal populations of Australia. His book entitled *Our Original Aggression* argued that the Aboriginal population, particularly that of Southeast Australia, was severely and repeatedly reduced by diseases introduced by British colonists and that

previous estimates of Aboriginal population in 1788 (then estimated to be approximately 300,000 for the entire continent) should be revised substantially upwards. He provided demographic models based on life-table analysis to support his case. Butlin made three assumptions to support his depopulation hypothesis: the first was that the 1789 smallpox epidemic was the first to hit the Aboriginal population of Australia and that they had had no previous exposure to the virus; the second was that the 1789 and the 1829-30 smallpox epidemics affected almost all the Aboriginal people of Southeast Australia; the third was that mortality in both epidemics followed the pattern of age-specific case fatality rates seen in unvaccinated communities in India but at a much higher level. In a discussion on the origins of the first recorded smallpox epidemic among Aboriginal populations in 1789 he suggested that it may have been started by deliberate release of the virus from the government's store of variolous material.

These arguments elicited further discussion and protest on the origin and extent of epidemic disease, particularly smallpox in early colonial Australia. Campbell (1983, 1985) argued that epidemics of smallpox originated on the north coast of Australia, introduced by Macassan sailors on their annual visits to collect trepang, a hypothesis that had previously been put forward by Cleland (1928, 1966). Curson (1985: 41-53) devoted a chapter to the 1789 epidemic of smallpox observed in the vicinity of Sydney and suggested that the disease may not have been smallpox but chickenpox and that the mortality, duration, and geographical extent of the epidemic had been exaggerated by previous authors. On the other hand, Frost (1994), also devoting a chapter to the 1789 epidemic, saw no reason to dispute the evidence that the disease was smallpox; an opinion shared by most other authors (e.g. Butlin 1983, 1985; Campbell 1983; Fenner 1984, 1985).

To the best of my knowledge there have been no attempts to document and discuss in detail other serious diseases such as measles, acute respiratory diseases, or sexually transmitted diseases, despite the effects they are perceived to have had on Aboriginal communities. In the *History of Tuberculosis in Australia, New Zealand and Papua New Guinea* (Proust 1991) the impact of tuberculosis among Aboriginal communities in colonial Australia received only three and a half pages in a chapter entitled 'Tuberculosis among Aborigines'. The majority of the chapter, however, dealt with tuberculosis in twentieth century Aboriginal populations in central and northern Australia.

Two other recent books dealing solely with Aboriginal health have not attempted to offer any substantial discussion on the colonial period. In one *The Health of Aboriginal Australia,* (ed. Reid & Trompf), Franklin and White (1991), in a chapter entitled 'The history and politics of Aboriginal health', presented a brief review of earlier published assessments on the demographic effects of introduced diseases before examining the politics surrounding Aboriginal health and social status in the nineteenth and twentieth centuries. Again, their emphasis was on smallpox. In the same publication Gray *et al.* (1991) wrote on 'Early epidemics and introduced diseases'. Again the emphasis was on smallpox and in particular Butlin's hypothesis on population decline. Brief mention was accorded to other diseases. In the other recent book to examine the history of Aboriginal health, *Aboriginal Health & Society*, Saggers and Gray (1991) tackled the topic of early introduced diseases in a chapter entitled 'Colonisation and its consequences'. Their concentration was on the changing social, economic and political impacts on Aboriginal health in the last two centuries. A brief review of early published material on introduced diseases was given as a backdrop to the main focus of the chapter.

Goldsmid (1984, 1988) provided an overview of the whole problem of the international spread of disease, with special emphasis on Australia. He discussed

diseases likely to have existed in precontact Australia and followed with a nine-page discussion on the entry of pathogens into the Australian continent but has only a few references to the effects of these disease on the Aboriginal people.

One writer to look further than the smallpox epidemics was Barwick (1971). Barwick examined archival sources on introduced diseases but restricted her analysis to Victoria and had population decline as her main issue. She argued that the spatial dissemination and mortality from smallpox, particularly the 1829-30 epidemic, had been exaggerated; and that other introduced diseases, particularly the chronic diseases such as pulmonary tuberculosis, and other respiratory diseases were the major cause for the decline in the population of Aborigines in Victoria (Barwick 1989).

Following on from Barwick, others have examined frontier contact in Victoria and the causes of Aboriginal depopulation. Christie (1979) discussed the period of contact in Victoria between 1835 and 1886 but only devoted a paragraph to diseases, preferring to emphasise European violence as the major cause of Aboriginal deaths. Critchett (1990) examined Aboriginal population decline in the western districts of Victoria between 1834-48. Her theme was directed towards the size of the Aboriginal populations just prior to settlement; the strength of their opposition to European expansion; and the social and demographic consequences of colonisation. Though she acknowledged that venereal disease and influenza caused high mortality, Critchett was more concerned with arguing for the presence of smallpox among Aboriginal groups of western Victoria than looking at the effects of other introduced diseases. Cannon (1990) further contributed to the topic of depopulation in *Who Killed the Koories?*, but made only brief references to diseases. The theme of Cannon's book was an emotive one as the subtitle - *The true and terrible story of Australia's founding years* - suggests.

1.3 Aims

These studies just discussed have gone some way towards an understanding of the changes in Aboriginal health during the nineteenth century. Smallpox has been the most commonly studied disease, particularly the 1789 epidemic. But there are still large gaps in our knowledge of the timing, dissemination, and effects of other introduced diseases during the nineteenth century. For example, chronic diseases which, while not killing swiftly in the way smallpox did, were nevertheless major causes of the ongoing mortality and population decline among Aboriginal people last century.

The central aim of this thesis is to fill the gaps of our knowledge of these diseases. A model (Table 2.2) of the changing medical circumstances in Aboriginal populations that began with the colonisation of Australia in 1788 and continued throughout the nineteenth century will be used as a structure in which to examine the introduced diseases. While the emphasis will be on the major diseases - smallpox, sexually transmitted disease (i.e. syphilis and gonorrhoea), tuberculosis, acute respiratory diseases, and measles - other disease states will be considered.

Historical literature concerning these diseases, and to a lesser extent skeletal evidence will be examined to:

- determine whether the epidemic events and annual outbreaks of introduced diseases among the European population showed a similar pattern of events among the Aborigines.
- establish in what regions and under what circumstances Aboriginal populations were affected by introduced diseases and epidemic events.
- ascertain whether introduced diseases became endemic among the sedentary groups on settlements between epidemic episodes

- demonstrate and analyse the evidence for morbidity and mortality.

1.4 Scope

The spatial scope of this study is referred to as Southeast Australia (Fig 1.1) covered today by the states of South Australia, Victoria, New South Wales, and Tasmania. There were several reasons for choosing this area of Australia. The first is that it covers the initial colonial settlement based at Sydney on the east coast and hence the first contacts between Europeans and Aboriginal people. Apart from a penal settlement on the Brisbane River (Queensland) established in 1825, the first thirty years of colonial expansion were contained within this area. Expansion of the colonial frontiers continued during the nineteenth and into the early twentieth centuries, with the north western part of Southeast Australia being the last to be settled by Europeans (Williams 1969: 12-44). As a consequence, the Southeast of Australia contained the majority of Australia's European population during the late eighteenth and nineteenth centuries, as it does today.

The Southeast also contained the largest pre-contact Aboriginal population densities of the continent (White & Mulvaney 1987:116-117) though they were concentrated in differing ecological regions. The eastern and southern coast and hinterlands of the mainland, the Murray Darling Basin, and the inland regions of the south, particularly those in western Victoria, were the areas of high biomass able to sustain large Aboriginal population densities (Butlin 1983; Lourandos 1983, 1987:293-307). In these areas population densities in 1788 are estimated to have ranged between 1.5 to 3.9 persons per square kilometre (Dowling 1990, Foster & Gara 1986, Lourandos 1987, Luebbers 1981, Smith 1980). Archaeological and ethnographic evidence has indicated that in western Victoria settlements consisting of stone hut clusters along water ways were occupied semi permanently (Critchett 1990: 26-27; Lourandos 1976, 1977, 1980, 1983, 1987:293-307;

White & Mulvaney 1987:116-117). In contrast with these high densities, the population density of the desert areas of central and northern South Australia have been estimated to be as low as 1 person per 50-60 square kilometres (Foster & Gara 1986).

The Southeast includes the whole or part of five of the eighteen proposed cultural regions of the continent existing prior to European colonisation (Horton 1994: 935; Peterson 1976: 50-72; McConvell 1990: 3-27). The population in the Southeast (the states of New South Wales, Victoria, Tasmania, and South Australia) has been calculated by Smith (1980: 69) at 82,500 or 26.2% of a total continental population of 310,000. This figure, however, should be seen as a minimum estimate of population as White & Mulvaney (1987:115-117), reviewing Butlin's (1983) calculations, have suggested a continental population of c.750,000 with the Southeast having at least 250,000 or 33.3%. A reasonable estimate of population in the Southeast would therefore lie between 85,500 and 250,000 or between 26 to 33% of the continental population.

Although I have included the present state of Tasmania as part of the Southeast, much of the historical literature regarding introduced diseases from this area (main island and Bass Strait islands) is restricted to those Aboriginal people confined on settlements (e.g. Plomley 1966; 1987, Ryan 1996). Unlike the mainland, few documents about the diffusion of introduced diseases among populations living beyond European frontiers exist. Because a major part of this thesis is concerned with the period prior to Aboriginal institutionalization on settlements, the available Tasmanian evidence on introduced diseases is used mainly in a comparative sense against that existing for the mainland.

The period to be covered begins in 1788 with the establishment of the colony of New South Wales and ends at 1900, the last year before Federation. The period of sustained direct contact between Aborigines and Europeans in Southeast Australia is

generally considered to have begun in 1788. Before the arrival of the First Fleet, sporadic contact between Europeans had occurred on the east coast of mainland Australia and Tasmania. Tasman sailed the southern coastline of Tasmania in 1642 and later D'Entrecasteaux in 1792. There is, however, circumstantial evidence that Portuguese mariners sailed down the east coast in the 1500s, but little is known of this venture (Frost 1987:369). British exploration began in 1770 when Captain James Cook in the *Endeavour* sighted the eastern coast of Australia near the present border of Victoria and New South Wales and then sailed north along the coast. In April of that year he sailed into Botany Bay and stayed for eight days. He later had to spend seven weeks at the mouth of the Endeavour River (the site of the present Cooktown), repairing *Endeavour* before leaving Australia via the Torres Strait. His contact with Aboriginal people in these places was limited and there is no record suggesting disease transferral.

The beginning of contact between Europeans and Aborigines differed in various regions of Southeast Australia in accordance with the spread of colonial settlements. After the establishment of New South Wales, Tasmania (Van Diemen's Land) was colonised in 1804, followed by Victoria (Port Phillip) in 1835, and South Australia in 1837. Exploration and settlement of the inland regions continued throughout the first half of the nineteenth century and the last Aboriginal groups to be contacted in were those of the northern Spencer and southern Eyre regions during the last three decades.

1.5 Significance of thesis

The significance of this thesis is two-fold.

1. It begins an examination of the biological and medical impact on Australian Aborigines in response to the changing social and cultural conditions that resulted when Europeans colonised the continent.

2. It adds to the growing body of literature regarding the biological consequences indigenous populations were subjected to as a result of the expansion of European powers into the Pacific and New World.

1.6 Sources and methods

The evidence used in this thesis comes primarily from European historical and ethnohistorical documentary sources and to a lesser extent from skeletal collections. For the most part quantitative evidence on morbidity and mortality could only be used after 1876 when recording of the Aboriginal population's vital statistics became more precise.

1.6.1 Historical sources

The major fund of information on the diseases and health of the Aborigines last century comes from a range of written sources. They include official correspondence of government bodies, parliamentary inquiries, reports tabled in colonial parliaments, journals and diaries of explorers, pastoralists, and travellers, letters and reports of missionaries and superintendents of Aboriginal settlements. Newspapers were a particularly productive source. They contain many of the government reports on Aboriginal matters reproduced verbatim in full or in part; letters to the editors from writers whose impressions of Aborigines were based on anything from long-standing personal contacts to a casual observation; and editorial opinions and judgements of European attitudes towards the Aboriginal people and the responses by Aboriginal people. These documents are housed in State Archives and State Libraries, in Sydney, Melbourne, Adelaide and Hobart; the National Library of Australia in Canberra; and the Institute of Aboriginal and Torres Strait Islander Studies in Canberra.

A two stage method of selection of suitable material from such sources was utilised in order to obtain the maximum information on the diseases from the most reliable observers. The first stage used a 'shot-gun' approach, *i.e.* any reference to disease or state of health of the Aborigines was noted regardless of its source or of its possible inaccuracy. The second stage involved culling this material in an attempt to establish some degree of accuracy and reliability in the reporting and the authority of those who observed or commented on diseases. This was done by selecting material originated by various sources:

- Reports by medical practitioners, particularly those who had cause to examine ill Aborigines on more than one occasion. Many of these reports originated during routine visits to Aboriginal missions and stations; others were on specially requested visits when higher than normal morbidity began to cause concern. They provide first-hand accounts of diseases and symptoms by contemporary qualified observers. In the majority of cases these reports were considered the most reliable of sources dealing with diseases.

- Routine reports by Aboriginal protectors, sub-protectors and mission superintendents appointed by the colonial governments. Reports were submitted to the colonial heads of the colonies covering either a three-monthly, six-monthly or annual period depending upon the official requirements of the time. Many of the references to disease in this material were by the protector or sub-protector who in most cases had no medical background. Often, however, they appear to have originated from either a verbal or written observation by medical practitioners and were often included verbatim or as statements within the body of the report.

- Reports by Aboriginal settlement superintendents. The majority of the mission settlements were under the administration of the various mission bodies who appointed their own superintendents. Regular (quarterly, annually) reports were submitted to the mission heads and to colonial officials responsible for the Aboriginal people. While the majority of missionaries had little or no medical training one stands out in his observations of diseases. George Taplin who founded the mission at Point McLeay on the Lower Murray River in South Australia spent twenty years (1859 to 1879) among the Narrindjeri until his death. Although he had no formal medical training his reports on the diseases experienced by these people provide a valuable insight on their changing health status and the timing and prevalence of the major introduced diseases in this region of the colony.

- Explorers who initially contacted Aboriginal groups. In a few cases explorers documenting the interior and coastlines of Australia paid particular attention to the Aboriginal people whom they met and recorded their observations in diaries, journals and letters. Although they often had no formal medical training themselves they were on occasions the first Europeans to see the clinical symptoms of active epidemics (as in the case of Charles Sturt who documented an epidemic of syphilis on the Murray River in 1829) or saw the sequelae of past epidemics (as in the case of Major Mitchell who recorded facial scarring from smallpox on the Darling River in 1836).

- Where none of the above sources were available, comments by observers who had no medical background or secondary advice; who had limited contact with Aboriginal people, or whose personal knowledge of the Aborigines could not be ascertained, were used. These came in the form of letters to the editors of newspapers, verbal statements to government representatives, or personal letters and diaries. In such cases I have used these sources only when they have been substantiated by other more reliable material.

For example, during epidemic years of influenza and measles a reasonable reliance could be ascribed to these sources when they refer to the same diseases which had been observed in the near vicinity during the same period by a more reliable source.

There were several problems in using the historical data. One of the major hurdles was that disease pathogenesis was poorly understood (and often misunderstood) by many members of the medical profession last century. This has led to symptoms of one disease often being diagnosed incorrectly as another entirely different disease. A case in point is sexually transmitted diseases. Often, the early symptoms of gonorrhoea were thought to be an aspect of the same disease entity as those of syphilis, mainly because of their common venereal means of transmission and the organs involved in their primary clinical symptoms. These two diseases, along with other sexually transmitted diseases, were often referred to by the medical profession by using the term 'venereal disease' or 'the venereal' This problem is further exacerbated by the very few surviving detailed clinical descriptions of these diseases.

Like all historical material written by Europeans that is used to reconstruct aspects of Aboriginal history, they must be used in a cautious and critical manner. An awareness of the ideological contexts in which they are written, and the character and background of the author should always be taken into consideration when using a source for evidence. When investigating the nature of diseases, which were poorly understood by the medical writers themselves, one must be additionally cautious.

1.6.2 Skeletal evidence

While the use of human skeletal remains in pathological interpretations of precontact and contact populations is well advanced, it is of limited value for this present

topic for a number of reasons. The main shortcoming is simply that the diseases that are of chief interest in this study either have no effect on the skeletal system, or infect the skeleton in only a small percentage of individual cases. For example, two of the major diseases causing high morbidity and mortality were smallpox and tuberculosis. In some cases of smallpox infection osteomyelitis variolosa occurs mainly in the three long bones (humerus, radius, and ulna) that meet at the elbow joint (Jackes 1983: 75, Zimmerman & Kelley 1982:109) and small necrotic foci involving the spine, sternum, and bones of the wrist and ankle. Zimmerman & Kelley (1982) found osteomyelitis variolosa occurring in as little as 2 to 5% of cases and almost entirely restricted to children under the age of fifteen. The severity of the disease also had no bearing on the extent of bone changes. The main reason for smallpox's restricted skeletal involvement is that it was often rapidly fatal, particularly in an immunologically vulnerable population, with death occurring often within 1 to 10 days of infection (Benenson 1990:395) giving little time for osteological infection and destruction.

Skeletal involvement in tuberculosis is usually a secondary development of the disease emanating from a primary focus in the lungs or lymph nodes. Steinbock (1976: 175) reviewed the incidence of bone lesions in tuberculosis patients and found it occurred in approximately 5 to 7% of cases. The most common form of skeletal involvement is Pott's disease of the lower thoracic and upper lumbar vertebrae, and inflammation and destruction of the articular surfaces of the knees and hips.

Previous palaeopathological examinations of skeletal collections in Australia (Dowling 1990; Webb 1984a, 1989, 1995) have failed to identify any smallpox or tuberculous lesions in bone. One reason for this lack of evidence for smallpox is the low frequency of osteomyelitis variolosa in smallpox cases and therefore the small likelihood of it appearing in skeletal collections. The likelihood of tuberculosis lesions being

represented in osteological collections is also low. Tuberculosis did not become a serious cause of morbidity among the Aboriginal people until the second half of the nineteenth century (see chapter five) when the greater part of the population was living permanently or temporarily on mission and government settlements. The majority of Aborigines dying from tuberculosis were buried within the grounds of these establishments and these individuals are not represented in the osteological collections.

Another problem in using skeletal remains in Australia is that the majority of the collections housed in museums were not excavated under strict archaeological control and are therefore poorly provenanced and dated. In many collections it has been difficult and often impossible to establish with clarity which individuals can be attributed to the precontact period and which come from the post contact period. This can only be resolved by individual dating of selected remains, a process that is destructive and largely undesired by both museum curators and Aboriginal custodians.

Syphilis, on the other hand, presents a clearer picture in the Australian skeletal collections (Dowling 1990; Webb 1984a; 1989, 1995). Untreated syphilis often develops from a primary infection located at the point of entry of the treponeme into a chronic systemic disease with severe lesions (gummatous osteoperiostitis) of the skeletal system. The frequency of bone involvement is greater than smallpox and tuberculosis, ranging from 10 to 20% of cases at the tertiary stage (Steinbock 1976: 109), probably higher when first introduced into virgin-soil populations. The lesions caused by the treponeme, particularly in the tertiary phase of the infection, can be readily identified in dry bone (Hackett 1976). Venereal syphilis was almost certainly one of the first diseases to have been introduced into Australia by European colonists and rapidly established itself throughout the Aboriginal populations of Southeast Australia. I have therefore limited the use of skeletal remains in this investigation to the discussion on venereal

syphilis along the Murray River. The material used is housed in the South Australian Museum and had been previously examined by me (Dowling 1990) and Webb (1984a, 1989, 1995).

1.7 Origin of diseases

This thesis is not about who was responsible for introducing new infections into the Aboriginal populations**.** This topic has been the subject of some contention among historians for some time (Butlin 1983; 1985; Campbell 1983, 1985) and continues to be debated (Frost 1994). For example, the case of who was responsible for the first introduction of smallpox in 1789, just one year after the British colonisation, has not been resolved. There is, however, little doubt that most of the exotic diseases introduced into Australia came with British colonisation and that these diseases were reintroduced several times over until effective quarantine methods were enforced.

1.8 Organisation

Chapter Two introduces a three stage medical model for late 18th century and 19th century Aboriginal Australians which acts as a framework for the remaining chapters. The first stage of the model deals with the pre-contact medical conditions which is discussed in Chapter Two. Chapters Three, Four, Five, Six, and Seven cover stage two of the model, the early contact period and diffusion of disease. Each of these chapters is concerned with a particular disease or group of diseases which reached epidemic proportions and caused wide-spread morbidity and mortality among the Aboriginal people. These are smallpox, sexually transmitted diseases, acute respiratory diseases, tuberculosis, and measles. Chapter eight is concerned with the third part of the medical model and discusses the principal diseases responsible for mortality of

Aboriginal people living on eight settlements in the colonies of Victoria and South Australia. Conclusions are presented in Chapter Nine.

CHAPTER TWO

Disease Environments and Epidemiological Considerations

2.1 Introduction

There can be little doubt that the Aboriginal people of Southeast Australia suffered severely from the stress brought about by European colonisation. They suffered the destruction and depletion of traditional food resources; epidemics of exotic diseases from which they had little or no immunological protection; wide-spread physical violence and genocide; the forced dispossession of lands; and in the later stages of contact, the confinement on settlements. Some groups could not recover from such a multi-pronged attack and ceased as culturally distinct units. Many other groups survived. After first suffering initial decline in population in the second and third stages of the contact period, the population reached a nadir which was sufficient for eventual recovery. Populations that recovered were nonetheless subject to considerable health problems and a severe reduction in individual psychological well-being.

This chapter presents a medical model in an attempt to identify the processes involved and the impact of new diseases that occurred among the Aboriginal populations of Southeast Australia after their contact with European colonists. The model has three stages relating to the colonisation of Southeast Australia. The first stage of the model (pre-contact) is discussed in this chapter and the second (early contact and diffusion) and third (settlement and acculturation) stages are the subjects of the remaining chapters.

2.2 A Medical Model for Contact in Southeast Australia

McElroy and Townsend (1989) developed a medical model based on changes in Canadian Inuit health subsystems during the 19th and 20th century that were brought about by European contact (Table 2.1). The table lists four stages of contact on one axis and the epidemiological, demographic, nutritional, and health care subsystems on the other axis. The circumstances presented by McElroy and Townsend in stage II and III are in many ways typical of the development of changes brought about by colonial occupation throughout the Americas (Verano & Ubelaker 1992) and the Pacific (McArthur 1967) - epidemics of exotic diseases following closely on the heels of colonial expansion, leading to extreme morbidity and mortality; population decline and in some cases a collapse beyond the threshold of survival; a collapse of long-standing social infrastructures; a decline in nutritional intake, leading to starvation in some cases; institutionalisation often away from traditional homelands; and a loss of control of individual and group lifestyles.

I have adapted the model to the Southeast Australian situation for the late 18th century and 19th century (Table 2.2). An extra subsystem representing changes to socio-economic circumstances has been added. This subsystem outlines the severe and often sudden changes to long-standing social and economic ways of life that occurred with the arrival of European colonists. Omitted from the model is McElroy & Townsend's Stage IV the equivalent of which began in Southeast Australia in the twentieth century (and to a large degree can be seen to exist today) and so is outside the scope of this discussion. The Aboriginal populations of Southeast Australia fit sufficiently well into the model proposed by McElroy & Townsend of indigenous decline and survival that followed colonisation.

Table 2.1 Changes in health subsystems of Canadian Inuit during stages of culture contact (McElroy & Townsend 1989: 312).

	Stage I Pre-Contact	**Stage II Early Contact and Diffusion**	**Stage III Settlement and Acculturation**	**Stage IV Modernization and Assimilation**
Epidemiological subsystem	Few pathogens in ecosystem; low immunities to infections	Epidemics of infectious diseases	Hyper-endemic infectious and nutritional diseases	Endemic infectious, nutritional, and stress-related diseases
Demographic subsystem	Births ≅ deaths, population stable	Births< deaths, population decline	Births > deaths population growth	Births ò deaths, slow population growth
Nutritional subsystem	High protein, low carbohydrate; fluctuating supply	Carbohydrate supplements; famine interacting with epidemics	High carbohydrate, low protein; food supply steady but nutritionally poor	High carbohydrate, low protein supply and quality varies by socioeconomic status
Health resources subsystem	Shamans and midwives fulfil limited medical & psycho-therapeutic needs	Shamans discredited in epidemics; missions provide relief	Government & missions provide modern medical care; health needs greatly increased	Modern medicine continues; birth control increases; health care and ethnic politics interconnected

Table 2.2 Medical model for 18th & 19th century Aboriginal Australians (after McElroy & Townsend 1989).

	Stage I Pre-Contact	**Stage II Early Contact and Diffusion**	**Stage III Settlement and Acculturation**
Epidemiology	Pathogens in ecosystem, chronic rather than epidemic; low immunities to exotic diseases	Epidemics of exotic infectious & respiratory diseases	Hyper-endemic infectious and respiratory diseases
Demography	Population homeostasis	Sharp population decline due to epidemics	Lessening population decline
Nutrition	Adequate or more than adequate nutritional requirements; often higher in carbohydrates than protein; fluctuating supply	Traditional food sources disrupted; lower protein & higher refined carbohydrate intake with introduction of sugar & flour; famine interacting with epidemics	High refined carbohydrate, low protein; food supply steady but nutritionally poor
Socio-economic	Hunter-gatherer economy; society based on kinship & spiritual ties to land	Economic disruption; displacement from land; social disintegration due to deaths from epidemics	Reliance on government & mission food distributions denial of land; social breakdown & destruction
Health care	Traditional medical practices fulfil psycho-therapeutic needs	Traditional medical practices unable to cope with epidemics & increased health needs	Health needs greatly increased; governments & missionaries provide limited but often ineffective medical care;

The pre-contact stage I is similar for each subsystem apart from the diet which was higher in carbohydrates for the Australian Aborigines than the Inuit (McElroy & Townsend 1989: 312). Stage II also shows a similar sequence in the subsystems but for the epidemiological subsystem where infectious and respiratory diseases were the main components for Southeast Australia. Stage III departs from McElroy and Townsend's model in the demographic sub-system. Population decline continued after Aboriginal people became institutionalised on mission and government controlled settlements. The official reports from these settlements show a common and continuous trend of declining population throughout the nineteenth century, with deaths continuing to outnumber births (Barwick 1971; Butlin 1983; Dowling 1990; Smith 1975, 1980). The respective colonial governments and missionary organisations in Southeast Australia were convinced of the eventual extinction of Aboriginal people. Population decline continued into the first decades of the twentieth century when the trend began to reverse (Smith 1975, 1980).

In accordance with the aims of this thesis which focus on introduced diseases the following discussion will centre on the epidemiological subsystem of the model. Discussions relevant to the other subsystems are incorporated within the following chapters.

2.3 Epidemiological Considerations

An infection, defined simply, is the invasion and multiplication of a infectious agent into body tissues (Benenson 1990: 501). If the invasion results in cellular injury and the manifestation of symptoms in the host an infectious disease is the result. The infectious agents (pathogens) capable of causing infectious disease range from minute and structurally simple viruses to large, structurally complex helminthic parasites. It is important to note, however, that infection by a specific pathogen does not necessarily

result in disease. The progression from initial infection by an agent capable of causing a pathogenic response in the host to the clinical symptoms of the disease depends upon a number of intervening variables. These include the pathogenicity of the agent, the route of transmission of the agent to the host, and the ability of the host's immune system to combat the infectious agent. All these factors, in turn, are affected by the natural and social environments in which the agent and host are juxtaposed; in some cases, the environments may promote the transmission of the agent to the host (e.g. crowded and poor hygienic living conditions), while in other cases it may limit or even prevent such transmission (e.g. small settlements and contact with fewer people). The transmission of tuberculosis provides a good example of such conditions.

2.3.1 Pre-contact disease.

In order to comprehend the effects of these previously unknown infections and how they influenced the colonial experience of Aboriginal people we must first delineate the disease environment that existed before Europeans arrived.

Traditional models of pre and post-contact health and disease proposed by anthropologists and historians have made two assumptions. The first is that indigenous populations, particularly those of the New World and Pacific, lived in a mainly disease-free environment prior to the arrival of Europeans with demographically and culturally stable populations (e.g. Bushnell 1993: 19-20; Ubelaker & Verano 1992: 279). This assumption has been based on an extensive body of recorded observations beginning at the times of initial contact between Old World and New World populations (Lucas Powell 1992: 41). In Australia the assumption was again made when early European observers consistently compared the poor state of health of Aboriginal people who had been in close contact with the colonists to the more healthier populations beyond the settlement frontiers. The latter were often described as strong, well-shaped, clean-limbed,

powerful, noble, and generally healthy (Marshall 1993: 483; Moodie 1973: 1; Sturt 1833: 126). The second assumption is that contact with Europeans was destructive to the health of indigenous people with epidemics sweeping through large regions and destabilising them demographically. While I have no quarrel with the second assumption (it is the subject of this dissertation) the first is deceptive.

The epidemiology of diseases in pre-agricultural hunter-gatherer populations has been frequently studied (Black 1980; Buikstra 1992; Cockburn 1971; Cohen 1989; Cohen & Armelagos 1984; Dobyns 1983, 1992: Fenner 1980; Lucas Powell 1992; Merbs 1992; McKeown 1991; McNeill 1976). Webb (1984a, 1984b, 1989, 1995) has discussed in depth the paleopathology expressed in the skeletons of Aboriginal Australians prior to European arrival. While these have largely put to rest the assumption of a 'disease-free paradise' (Ubelaker & Verano 1992: 279) among New World populations the assumption, however misleading, still lingers for Aboriginal Australia (Franklin & White 1991:3; Marshall 1993: 483).

Here I wish only to outline briefly the epidemiological nature of the pre-contact disease environment in Southeast Australia. In Stage I of the medical model (Table 2.2) I have summarised the disease environment as having autochthonous pathogens causing disease but rarely reaching an epidemic level. The epidemiology and mortality of infectious diseases and their effects on the demography of hunter-gatherer societies are inextricably linked to their social systems and the ecosystem in which they exist (Polunin 1977). The major characteristics of Aboriginal society and ecology which would have exerted an influence on the pattern of disease are summarised in Table 2.3.

Table 2.3 Major characteristics of Aboriginal society and ecology (compared to Europeans) exerting an influence on the pattern of disease (Polunin 1977:7).

Population distribution	Lower population density Smaller residential/foraging groups Wider spatial distribution Large group aggregation occurs infrequently Greater residential mobility Shorter range of individual movements More contact with fewer people
Stability	Greater resistance to changes Greater vulnerability to external influences
Complexity	Fewer formal roles and occupations More rigidity in maintaining social and subsistence roles Simpler technology
Ecology	Closer association with, and adaptations to the ecosystem Less close association with other animal species Less degradation of ecosystem

The main characteristic of Australian Aboriginal epidemiology before European contact would have been the presence of human and zoonotic pathogenic agents causing endemic diseases (Cockburn 1971; McNeill 1976; Black 1980; Fenner 1980; Verano & Ubelaker 1992). Epidemics may have occurred but they would have been limited temporally and spatially (Black 1975,1980).

The evolution of the relationship between humans and disease in Australia spans the 50,000 years (Roberts *et al.* 1990) to 116,000 years (Fullagar *et al.* 1996) of human occupation of Australia. The first human diseases to enter would have been those already well established in the populations of Southeast Asia. Humans arriving on the Australian continent would have brought with them many of the pathogens that were able to be transmitted directly from one human host to another and able to be maintained among the groups moving across the island chains to the Australian continent. They would have left behind all the zoonoses and infections that required vectors, or obligatory alternative hosts found only in Southeast Asia. When they arrived in Australia the first people encountered the marsupials, a completely new type of fauna. The infectious diseases carried by the marsupials would have differed considerably from those of the Asian mammals and as a result the zoonotic diseases acquired would have been of a kind not previously experienced (Cockburn 1971: 48). After many years of interaction between the human hosts and the infectious agents a state of equilibrium would have then resulted. Many of the diseases would have contributed, either on their own or collectively, to mortality among Aboriginal people; usually at low levels of incidence and mainly to the very young and the elderly. Only under exceptional circumstances would diseases have reached epidemic proportions and caused excessive mortality among the infected population. It was left to another group of humans arriving in Australia from Europe to introduce new and lethal pathogens and thus upset the balance.

Table 2.4 Probable major infections of Aboriginal communities of Southeast Australia prior to European contact (Benenson 1990; Goldsmid 1984, 1988; Merbs 1992; Stevenson & Hughes 1980).

Disease	Reservoir/Host
Worldwide	
Herpes	Human
Mononucleosis	Human
Hepatitis B	Human
Trachoma	Human
Whipworm	Human
Roundworm	Human
Ringworm	Human
Scabies	Human
Pediculosis - lice infection	Human
Streptococcal diseases	Human
Staphylococcal diseases	Human
Diarrhoea, acute bacterial	Human
Salmonellosis	Human/Animals
Tetanus	Human/Animals/Soil
Autochthonous	
Hydatids?	Dingo/kangaroo
Botulism	Spores in soil/fish
Australian encephalitis (MVE)	?Mosquito eggs
Ross River Fever (RRV)	Macropods
Query Fever (Q fever)	Bandicoots
Cryptococcosis	Human
Scrub Typhus	Mites
Queensland tick typhus	Ticks
Leptospirosis	Bandicoots, marsupials

Table 2.4 lists probable major infections of Australian Aborigines prior to European contact. The list consists of worldwide diseases that are known, or have been suspected of, having a long association with humans to the extent that humans are the major reservoir for the pathogens, and pathogens that are known to infect humans and are autochthonous or suspected to be autochthonous to Australia.

Many of these infections are capable of inflicting illness ranging from mild to acute life-threatening symptoms in individuals. Chronic infection may have caused anaemia in up to 45.6% of individuals as diagnosed by Webb (1995: 122) in one area of the central Murray River prior to European arrival. In some cases localised epidemics infecting a number of people simultaneously were likely to have occurred, particularly among communities on the east coast and the Murray, Murrumbidgee, and Darling River corridors where population densities were high and people were becoming more sedentary (Luebbers 1978; Webb 1995: 274-291).

Absent from this list are the 'urban' or 'crowd' infectious diseases that were endemic among the populations of Europe and Asia - smallpox, measles, influenza, chickenpox, poliomyelitis, typhoid; and the chronic diseases syphilis and tuberculosis. There is no evidence, circumstantial or otherwise, that these latter diseases existed among the Aboriginal population of Southeast Australia before 1788 (see following chapters).

Table 2.5 lists non-infectious disease states that were likely to have affected Aboriginal populations in Southeast Australia. Webb (1984a, 1984b, 1989, 1995) has documented the wide-spread prevalence of nutritional and degenerative diseases, and trauma in the form of cranial fractures, trephination and broken limbs and ribs in skeletal collections Australia wide. Neoplastic disease in the form of multiple myeloma, metastatic carcinoma, nasopharyngeal carcinoma, and osteomas (benign neoplasms) have

been identified in at least five Australian Aboriginal skeletons (Webb 1995: 217-234). Evidence of neoplastic disruption of bone tissue in prehistoric skeletal samples is rare (Ortner & Putschar 1981), mainly because the death of the individual usually occurs with the destruction of vital soft tissue before the bone is affected. The prevalence of neoplasms in precontact Australia can then be expected to be more than is represented in the skeletal collections.

Table 2.5 Probable non-infectious disease profile of Australian Aborigines before contact with Europeans (Thomson pers comm.).

Disease type	Occurrence
Metabolic	Some
Nutritional	Some deficiencies, anaemia (famine, parasitic)
Traumatic	Non-intentional - falls, bites, stings, burns
	Intentional - human aggression
Degenerative	Arthritis, dental attrition
Neoplasms	Little - idiopathic, viral
Allergies	Little
Toxic	Some
Psychological	Some
Psychiatric	Some

The extent and prevalence of the other states are unknown, but on the basis that they are known to be present in all historic and modern human populations there is no reason to assume their absence in precontact Australia.

Saunders *et al.* (1992: 117-118) suggest that the acceptance of pre-contact health has lead to the distorted use of the epidemiological concept 'virgin-soil epidemic' (Crosby 1976) by many researchers. Virgin-soil populations have often been described as having had no previous exposure to particular pathogens and thus lacking the necessary immunological response when infected. Mauser and Bahn (1974: 27), however, define

virgin-soil populations as those 'in which an organism has not been present for many years, if ever'.

When an infectious disease occurs in a population it will usually cause death or leave the survivors immunologically protected. If the protection from the disease is long-term and the disease does not reappear, future generations will have had no exposure to the disease and will be unable to develop antibodies to the pathogen. This new or younger virgin-soil population is then susceptible to the disease should it reappear, while the older generations may still be able to produce antibodies to the pathogen and escape serious clinical disease.

An example of a new virgin-soil sub population being created is cited by Black (1990: 59) among the Amazonian Parakana after exposure to hepatitis A. The Parakana were first exposed to hepatitis A in 1927 which was then present in a neighbouring and rival tribe which they continually raided for a period of ten years before breaking off contact and isolating themselves in the forest. In 1984, after they emerged from the forest and re-established contact, Black and his team were able to test them immunologically. The results showed an age specific pattern in positive reaction to hepatitis A. They found that everybody over the age of fifty had antibody to hepatitis A but nobody younger. The virus had not persisted in the Parakana after the raiding period when they lived isolated in the forest and a new virgin-soil subgroup of the population susceptible to hepatitis A had been created within the next generations.

We do not know whether the Aboriginal people of Southeast Australia had any previous experience with an introduced disease before the arrival of Europeans in 1788. Diseases such as smallpox and tuberculosis may have been introduced to Australia from Asia (Butlin 1983, 1985). If they had, the excessive morbidity and mortality that

later resulted from diseases such as these after European settlement, strongly suggests that they had little or no immunological protection and that any experience with these diseases would have been long in the past. The Aboriginal people of Southeast Australia had no herd immunity to the introduced diseases and the region was virgin-soil for many pathogens endemic among the Europeans.

The epidemiological concept of a virgin-soil population can therefore cover situations where contact with a particular pathogen has caused an epidemic, but the disease did not become endemic among the populations and eventually ceased to exist. The herd immunity or immunological protection to the pathogen would have been lessened and eventually lost with the succeeding generations having little or no exposure to the pathogen (Saunders *et al.* 1992:117).

To conclude I can see no problem with the assumption that new pathogens caused increased morbidity and mortality to Australian Aborigines. No-one can dispute the fact that the diseases introduced into the New World populations were horrifically destructive to the indigenous people because of their lack of immune experience with the new pathogens.

2.3.2 Post-contact Epidemics

The process by which an epidemic of an infectious disease develops and the period for which it can sustain a high level of incidence, are dependent on several factors which are related to the success of the pathogen's ability to invade, establish itself in a new host, multiply, and then spread into a new host.

- The presence of a source of the fully virulent microorganism. Introduction and maintenance of the source of pathogens is often through actual cases, convalescent cases, and apparently healthy carriers able to pass the pathogens on to further susceptible individuals. Once introduced, the pathogen may be able to reside in animal hosts, re-emerging when conditions are favourable, to infect human victims.

- The release from the source of infection of a large number of viable pathogenic agents. The larger the number of agents able to be released from an infected host, and the longer the period of time the host remains infectious, the greater the chances are of the disease finding a new host and maintaining itself in a population.

- The survival ability of the pathogen outside the infected host. The microorganism must retain its pathogenic ability between the time it leaves the host source to the time it is able to enter and become established in the body of the new host.

- A high number of susceptible individuals must be present in the population exposed to the pathogen; or put another way - the herd immunity to a particular pathogen must be low enough to maintain it at an epidemic level. Many diseases are density-dependent and need a high number of susceptible individuals in a group or community to maintain their presence. Smallpox was an example of a density dependent disease. If the disease is to maintain a high incidence within a population there must be a large number of potential humans available who can host the pathogen. In turn, their immune systems must allow the pathogen to multiply and to develop the ability to exit one host and infect the next. If the herd immunity to a particular disease is high then the chance of the disease becoming epidemic are low.

- The frequency of contact between infected hosts and susceptible individuals must be suitable to the pathogen, enabling it to transfer from one to the other. This may be achieved by direct means (e.g. person to person contact) or by indirect means (e.g. an insect vector). The more personal contacts that are achieved, the greater the period of time the contacts are maintained, and the more intimate the contacts, the greater will be the spread of the pathogen from host to host and thus the better the ability to maintain the development of the epidemic.

The effects of introduced infectious diseases upon an indigenous population would vary according to several factors. Previous exposure to the pathogen, the point of entry and route of transmission, the population density, and the health and nutritional status of individuals would have strong bearings on the degree of morbidity and mortality of a disease on the indigenous population. Cultural practices and intergroup social relations also affect the dissemination of new pathogens.

Exotic diseases introduced into a population with no immunological experience of them often display particularly high virulence of the pathogen and the fulminate character of the symptoms (Crosby 1976; McNeill 1976). Black (1992: 1739-40) has noted that an individual who catches measles from a family member or near relative faces nearly twice the risk of death as an individual infected by someone unrelated. His hypothesis for this is that the virus grown in one infected host is preadapted to a genetically similar host and so increases its virulence. Histocompatibility (MHC) antigens in a host infected by the virus present a restricted set of viral peptides to the immune system which in turn selects against viruses with these peptide sequences. Under natural selection the viruses that survive immune system attack are then preadapted to a closely related host and are better able to survive the immune response and replicate within the host. When the virus enters successive unrelated hosts it encounters a new

genetic background (new MHC genes) to which it is not efficiently adapted and while not being as virulent within that host may nevertheless be able to survive and reinfect.

The cycle of adaptation and re-adaptation continues as a virus spreads throughout the populations. Black (*ibid*) has estimated that among South Amerindians there is a 32% chance that a virus will not encounter a new MHC type at either the A or B locus when passing from one host to another. As new pathogens entered Southeast Australia, the chances of finding successive closely related hosts with similar genomes would also have been high. A gene pool more restricted among the Australian Aborigines than among the Europeans together with many common lines of descent within clans would have enabled many of the pathogens to retain a highly virulent state as they passed through the populations.

In examining the biological consequences of New World colonisation Crosby (1986), Dobyns (1983) and Ramenofsky (1987) identified eighteen viral, bacterial, and protozoal diseases most likely to have been introduced for the first time to the indigenous populations of the Americas. Table 2.6 lists these diseases and compares them with those that were introduced into Southeast Australia during the nineteenth century.

With the exception of yellow fever which has not been recorded on shore in Australia (Cumpston 1989: 221), malaria which when introduced was most probably confined to Northern Australia (Black 1956: 136; Cumpston 1989: 221; Goldsmid 1984: 181), and bubonic plague which was first recorded in Australia in the initial months of 1900 (Cumpston 1989:192), all of the diseases introduced to the Americas were subsequently introduced into Southeast Australia following the arrival of the First Fleet in 1788 (Black 1956: 136; Cumpston 1989: 221; Goldsmid 1984: 181).

Table 2.6 Diseases suggested to have been introduced by European colonists into Aboriginal populations of Southeast Australia (post 1788) and the Americas .

Disease	**Post 1788 Southeast Australia**	**America (Ramenofsky 1987)**	**America (Crosby 1986)**	**America (Dobyns 1983)**
Chickenpox	X	X	X	
Dengue fever	X		X	
Influenza	X	X	X	X
Measles	X	X	X	X
Mumps	X	X		X
Rubella	X	X		
Smallpox	X	X	X	X
Cholera	X		X	X
Diphtheria	X		X	X
Pneumonia	X	X		
Pertussis	X	X	X	
Scarlet fever	X	X	X	X
Typhoid fever	X		X	X
Anthrax	X	X		
Plague		X		X
Malaria		X		X
Typhus	X	X		X
Yellow fever		X	X	X
Syphilis	X			
Gonorrhoea	X			
Tuberculosis	X			

There are, however, three notable diseases omitted from the Americas lists which were introduced into Australia during the process of colonisation.

Treponematoses (venereal syphilis, non-venereal syphilis, yaws) has been the subject of a long-standing debate concerning its endemicity in pre-Columbian America (e.g. Cockburn 1961; Hudson 1963, 1965; Crosby 1969, 1972, 1986; Hackett 1963). The debates have centred around the presence or absence of treponematoses in pre-Columbian America, and their spread throughout Europe after the return of Columbous from the New World. In her review on pre-Columbian disease environments Buikstra (1994) sees the debate now swinging more convincingly to endemic forms of treponematoses in the Americas having both a venereal and non-venereal mode of transmission. While the debate is of interest to this project it is not a direct concern and I shall not contribute to it here. I do, however, challenge the argument made by Webb (1984a, 1989, 1995) of an endemic form of non-venereal syphilis in Southeast Australia prior to European arrival (see Chapter Three).

Gonorrhoea is one of the oldest known and most common human diseases, with a history dating back 5,500 years (Rothenberg 1993: 759). Its presence in pre-contact Australia is unknown. An introduction cannot, however, be discounted in Australia's north where contact between Macassan fisherman and Aboriginal groups living on the coast was a frequent event, at least from the middle of the eighteenth century and before European colonisation of the region (Macknight 1976; Mulvaney 1989: 22).

The third omission from the Americas lists is tuberculosis. This is a disease transmitted mainly by airborne droplets carrying the bacilli and is often characterised by acute and chronic symptoms. In the crowded and confined spaces of shipboard life tuberculosis was an excellent traveller and was carried across the world time and time

again (Proust 1991a: 5-26). Erosive spinal, longbone and rib lesions in skeletal populations dated to c.2,000 BP have now confirmed the presence of tuberculosis as an endemic disease in pre-Columbian America (Buikstra 1981, 1994: 311; Lucas Powell 1992). There is no such evidence of the disease among pre-contact Australian populations (Dowling 1990; Webb 1984a, 1989) and it was almost certainly absent in Southeast Australia before 1788 (Thomson 1991:62). Again, it may have been introduced into north Australia before 1788 through the long-standing annual contact between Macassan fishermen and Aboriginal people but there is no positive evidence for this.

Most of the infectious diseases introduced into Australia would have originated from Britain. The length of ocean voyages, the place of departure and subsequent ports of call, however, are other major considerations of transcontinental disease dissemination. The First Fleet left Portsmouth on 13 May 1787 and sailed into Port Jackson on 27 January 1788, a voyage lasting thirty-six weeks; a period probably too long for diseases with short periods of infectivity originating in Britain to retain their communicability. On its passage the First Fleet called in at Rio de Janeiro in August 1787 and the last port of call was Capetown before a nine week crossing of the Indian Ocean to the east coast of Australia. It is conceivable then that some short-term infectious diseases may have originated from South Africa. Chronic infectious diseases, such as tuberculosis and syphilis, could have survived the journey from Britain.

Once the colony had become established in the nineteenth century the frequency of ship arrivals increased and improved sailing technology reduced the time of passage between ports of origin and destination (Blainey 1982a: 173) allowing the short-term infectious diseases a better chance of surviving the trip. This allowed a chain of infection to become established, throughout the initial colony and into other new

settlements which, until effective quarantine practices were in place, would have been difficult to break.

Throughout the nineteenth century the European population of Southeast Australia increased and movements across that part of the continent became complex. New settlements were established at Hobart in 1803; Melbourne in 1835, and Adelaide in 1836. The spread of pastoralism in the new colonies opened up the interior lands, extended the European frontiers and increased contact with Aboriginal communities. During the 1850s a population boom occurred following the discovery of gold. The European population of Victoria increased three-fold during the decade and in New South Wales it more than doubled between 1851 and 1861 (Blainey 1982c: 158-163). Also, in 1852 and 1853 a total of about 18,000 Chinese arrived in Victoria, and by the late 1850s more than 40,000 lived on the diggings (Blainey 1982c: 164-165). The frequency of disease entry into Australia would have increased with migrant arrivals and the new diseases became established in the high population centres leading to endemicity of introduced pathogens.

Historical documentation provides ample evidence of the impact of Europeans on the Aboriginal people. Dispossession of their lands, addiction to tobacco and alcohol, loss of access to traditional foods and incarceration in government and mission institutions often in unhygienic conditions, predisposed them to severe mortality and morbidity from the new diseases. Chronic malnutrition completed the well established connection between social disruption and infectious diseases. The new diseases flourished in the environment of unestablished immune responses and decreased immuno-efficiency of individuals, often suffering from multiple chronic and acute infections.

2.4 European and Aboriginal concepts of disease and curing

When the European colonisation of Australia began in 1788 both they and the Aborigines had well established sets of beliefs in regard to sickness and curing. It is therefore important to sketch the underlying concepts regarding disease and curing of both western Europe and Aboriginal Australia because together they helped to determine firstly, the Aboriginal response to the invasion of Old World pathogens and secondly, the response by the European colonial system and its attitude towards the diseases among the Aborigines.

Every human society is subject to illness in one form or another. Each in turn has evolved concepts that define the various physical and psychological conditions that effect personal well-being, explain the cause of illness, and proffer cures. While the European and Aboriginal medical systems, as they were in the late 18th century, differed in many respects, both were more socially than medically therapeutic. Neither could adequately cope with the morbidity and mortality of infectious diseases and neither could offer an effective cure to those infected.

At the time of colonisation, the arriving European medical community largely subscribed to the long standing humeral theories of sickness and health. These had been first advanced by Hippocrates in the fifth century BC, and later championed by Galen to such an extent that they became a part of a quasi-religious dogma (Duin & Sutcliffe 1992: 22; McNeill 1976: 218-219). The idea that minute organisms might be the cause of disease ('germ theory') had been postulated as early as the second century but it had little impact on the medical establishment's conception of disease aetiology (Duin & Sutcliffe 1992:56). It was not until the nineteenth century breakthroughs of Pasteur and Koch that the role of microbes in the causation of disease began to be accepted. These theories did

not go unchallenged in Europe and the medical profession was slow to come to terms with the new ideas of illness and the new ways of treatment. The concepts were even slower to become accepted in the distant Australian colonies (Cumpston 1978: 1-8). The 'tyranny of distance' (Blainey 1982a) which so often influenced the political, social and commercial history of Australia in the nineteenth century also encumbered the dissemination of medical knowledge. So, at the beginnings of the colonial period and well into the nineteenth century, the understanding of disease aetiology and therapy was still based on the long-held theories of humeral causes (Cumpston 1978: 1-8).

The traditional view held by Galen was that the four humours; blood, phlegm, white bile and black bile, present in the body, coincided with the four elements that comprised the world; air, water, fire, and earth. Each of the humours had qualities of hot/cold and wet/dry. As the theory passed through the centuries it became subtly altered and adorned with new connotations but the core concepts persisted. Illness in a previously healthy person was perceived to be the result of an imbalance between the bodily humours brought about by changing climatic or environmental factors (Howe 1972: 7; Duin & Sutcliffe 1992: 56). Most epidemic diseases were understood in much the same way. But some, for example smallpox, were seen to be rather different and appeared to result from a tangible contagion that was passed through the air from an infected person to others. This was the presumption underlying the development of variolation in the early eighteenth century and later Jenner's idea of inoculation. An infective agent was also suspected by some to be the cause of tuberculosis (Ruddock 1873; Johnston 1993:1064).

The underlying principle behind treating the ill and affecting a cure was to restore the balance of the humours. One of the most common practices up to the late 19th century was to bleed the patient to expel the corruption within. This may actually have

been extremely deleterious to the health of the patient because it reduced the body's own ability to fight the infection by the loss of the very medium, the blood supply, that carries the antibodies to the infected area. Other methods used to restore the bodily balance were medicinal. The drugs used for the treatment of diseases were, however, generally ineffective in combating the infectious agents and in most cases had only limited effect on the symptoms. For example, a range of drugs was prescribed for the treatment of diarrhoea. Acute bacterial diarrhoea was a prevalent complaint of the times, mainly due to the lack of knowledge regarding the cause of putrefaction and the poor hygienic conditions that commonly prevailed. Conversely, drugs purported to relieve constipation were used for almost all the non-specific illnesses e.g. debility, headache, anxiety, rheumatism etc. Along with the practice of blood letting, purgation was considered to be one of the best treatments to rid the infectious matter from the body and restore the balance of the body humours (Duin & Sutcliffe 1992).

In many cases drugs were prescribed to relieve pain symptoms. Before modern analgesics, the most effective were opium and its derivative morphine, often causing problems of dependence and withdrawal symptoms when the drugs were no longer available (Duin & Sutcliffe 1992). Others were given for the relief of respiratory symptoms, e.g. coughs, colds, and the effects of bronchitis, influenza, and tuberculosis (Potterton 1983). Perhaps the most dangerous of the forms of chemotherapy of the time was the use of mercury, particularly in the treatment of syphilis symptoms. Until the introduction of arsenic derivative treatments and the development of penicillin antibiotics in the twentieth century, mercury chemotherapy had been the mainstay of treatment for syphilis for four hundred years. Despite such a long faith by the medical profession in its effectiveness, the drug had little effect on the symptoms of syphilis and was completely ineffective against the causative agent of the disease. But if taken over a long period, as it customarily was by the sufferers of chronic syphilis, it induced severe iatrogenic illnesses,

the cause of which frequently went unrecognised by doctors and often resulted in the victims' demise.

Many of the drugs used in the nineteenth century were patent medicines of little use in reducing the symptoms of infection. A highly popular concoction of the day was 'Holloway's Pills' reputed to cure gout, rheumatism, inveterate ulcers, sore breasts, sore heads, bad legs and so on. 'Doctor Morse's Indian Root Pills' were another invention that claimed to purify the blood and to be a positive and permanent cure for 'biliousness, constipation, indigestion, headaches, kidney troubles, piles, pimples, and female ailments etc' (Cumpston 1978: 3). Even as late as the turn of the twentieth century little effective treatment was available for most of the known infectious diseases and doctors treating the ill were helpless because of their lack of knowledge of the true causes of the diseases (Cumpston 1978: 3).

At the time of colonisation and for many years afterwards, Western anthropologists had little or no knowledge of Aboriginal medical theories. Because of the rapid destruction or transformation of many Aboriginal societies, many of the then existing medical practices and beliefs have been lost for ever. But by drawing upon more recent anthropological studies of contemporary Aboriginal medical beliefs and practices we can gain some insight into how diseases and their cures may have been perceived. Aboriginal approaches to health and illness were most probably holistic ones, recognising spiritual, physical, and biological aetiologies and therapies (Reid 1982: 91). For the most part, however, the theories of illness were based on magicoreligious beliefs (Taylor 1977:423; Saggers & Gray 1991:47). Blame for an illness was often attributed to the machinations of rivals or to the will of angry supernatural beings intent on revenge for the alleged misdeed of the patient. Divine anger was seen by some as the cause of the 1828-30 smallpox epidemic which swept along the river systems of south eastern Australia

(Dawson 1881:60). Others placed the blame on the first European they had seen. The explorer Charles Sturt was present in the land of the Wiradjuri just as smallpox began and was seen as the cause of the epidemic (Mair 1831). The Ngarrindjeri and Meru along the Murray River in South Australia blamed those further upstream for unleashing the epidemic (Chapter Three). Determining the cause was an important part of coping with illness (Saggers & Gray 1991: 41-52). It allowed the victims or their families to vent their anger and frustration and so alleviate their feeling of helplessness which often arose from being unable to control the disease. Finding a cause also transferred the responsibility for the condition to someone or something else (Goodall 1994: 68-72; Mobbs 1991:303-308).

The practice of healing the sick was often vested in special people. Doctor-sorcerers were able to evoke and control alternative supernatural forces to combat those already causing the illness. Not all of these practitioners, however, used their skills for healing. They in turn could use their power and knowledge to cause illness and death among others. The pointing of a bone by a sorcerer accompanied by a magic ritual, chant, or song was widely used across the continent to invoke an illness, or to cause the death of the unfortunate recipient (Berndt & Berndt 1988: 307-322). Frequently the process of healing involved an act of purification or cleansing, such as sucking the skin to remove the underlying pathogen, blood-letting, rubbing and massaging the skin to purge the illness from the body. Heat was often applied to the body by burying the victim in warm sand and ashes or suspension over a fire. This induced perspiration which was to sweat the illness out. In other methods the illness could be ritually transferred to another object and then cast out.

More profane (and potentially dangerous) methods of treatment were used by the doctor-sorcerers. During the 1830 smallpox outbreak in the Lachlan and Wellington

Valley region of New South Wales a victim had his hair scorched from his head, probably in an attempt to relieve a severe headache which was a common symptom of the disease. The *Kradjee* (healer), who had previously observed other cases of smallpox where the pustules had burst, then began to prick the pustules with a sharp-pointed fish bone and squeezed out the fluid contained in them with the flat part of the instrument (Bennett 1834: 154-156). The outcome of this treatment on the patient and the doctor-sorcerer is unknown, but considering the highly contagious nature of the disease, a favourable result for both would not be expected.

Patient's participation in their own or their family's treatment was a socially important aspect of the healing process. This allowed the sick to assert some measure of control over the state of their body and their future well-being. With the breakdown of the social fabric following the incursions of European colonisation, and the loss of faith in the powers of the healing doctor-sorcerers to cure the new illnesses, this was often the only way open to healing. An extreme method was used by one Aboriginal group in South Australia in the late 19th Century who were probably suffering from the maculopapular rash of venereal syphilis. They approached a group of Europeans who were treating a flock of sheep with an arsenic based dip. One of the men was in a poor state and in jest it was suggested by the Europeans that he should be dipped like the sheep.

> The scabby black was put in the vat, and after a short time he became so ill that it was feared he would not recover, and lost his hair, toe nails and finger nails; but in time an improvement was observable. His skin peeled off, and he was described as a magpie when moulting. Eventually he quite recovered, his hair grew again, and his skin became as smooth and as glossy as marble. When this became known among the tribes others having the same complaint presented themselves at Cannowie, and begged to be "jipped like it other feller;" but the experiment was considered too hazardous, and no one cared to risk a trial for murder or manslaughter, if, as was by no means unlikely, a patient should die under the treatment (*Adelaide Observer* 1904).

While sorcery and supernatural agents probably formed the most important component of medical concepts, a relationship of physical cause, medical consequence, and biological treatment was widely acknowledged. Accidental and deliberate trauma, diet and weather fluctuations were seen as direct causes for certain medical conditions and specific methods were used to reduce their effects on well-being (Scarlett *et al.* 1984; Saggers & Gray 1991). Together with this knowledge Aboriginal societies could draw upon an extensive knowledge of medical resources and treatments. Scarlett *et al.* (1982) have documented medical practices of the Yolngu from northern Australia and have revealed an extensive pharmacopoeia based mainly on plant species but also using animal and mineral preparations. Preparations were used for the treatment of coughs, colds, disorders of the lungs, eyes, teeth, and healing agents for surface and penetrating wounds. Most of the people were aware of these 'bush medicines', in particular the elderly women who regularly collected the species and prepared the medicines. Many of the treatments were used on specific ailments, others had multiple uses, while others were more generalised treatments.

Australian Aboriginal pharmacopoeia was quite adequate for dealing with wounds, burns, bites, gastro-intestinal ailments and body aches that were likely to occur in the daily life of any group. But there were no traditional remedies for the Old World pathogens such as tuberculosis, influenza, and smallpox that caused high morbidity and mortality after 1788. This is not to say that European medicine could cope any better with these diseases. In reality the European medical system could offer no cure and very little relief from the major infectious diseases.

CHAPTER THREE

Smallpox

3.1 Introduction

Until it was officially eradicated in 1979 (Fenner *et al*. 1988: vii) smallpox had been a highly infectious and serious systemic disease of humans, erupting in populations throughout the world in particularly fatal epidemic episodes. The causative agent of smallpox, variola virus, now no longer occurs in its natural environment and exists in only two laboratories, one in the United States and the other in Russia.

The origin of smallpox is not clear. It most probably developed as a zoonosis during the Old World Neolithic period when agricultural societies grew sufficiently large enough to provide a supporting human reservoir for the virus (Crosby 1993a: 1008). The disease, as it was known in modern times (a human disease with no animal reservoir), required a large population reservoir to maintain itself and so almost certainly did not exist among the hunting and gathering peoples of the Palaeolithic (Crosby 1993a: 1008).

Descriptions of smallpox, or a disease of a similar nature, are known from China during the 4th century AD and south-west Asia in the 7th century AD. One of the earliest victims of the disease may have been Ramses V of Egypt who died in 1157 BC (Crosby 1993a: 1009; Fenner *et al*. 1988: 209-211). As large centres of population developed in Europe, Asia, and Africa and movement of people increased, smallpox developed from the occasional epidemic to become a well established endemic disease - one of the most deadly known to humans of the Old World.

Its trans-oceanic spread began in the early 16th century when it was carried across the Atlantic by Spanish expeditions to the New World populations of the West Indies, Mexico, South and North America (Crosby 1986: 196). Mortality among these populations was extreme (Dobyns 1983: 251-289; Stannard 1992: 77-136). Early Spanish estimates suggest a 25% to 50% case-fatality rate among the Indians of Central America (Crosby 1993a: 1010). Similar rates, or higher, were consistently reported throughout the continent for the next three hundred years. Among the Amerindian populations, smallpox was reported to have devastating effects in the Andes beginning in the early 1540s (Cook, N.D. 1992: 208-213); in Massachusetts during the seventeenth century (Crosby 1986:198; Cook, S.F. 1973: 491); among the Mohawk, Iroquois, and Huron of the Northeast of North America from 1634 on (Snow 1992: 183); in the Southeast during the early sixteenth century (Thornton *et al.* 1992: 191-193); in the Southwest during the fifteenth and sixteenth centuries (Stodder & Martin 1992: 67); on the Northern Plains and Missouri River Valley during 1837-1838 (Trimble 1992: 257-264); and among Californian mission populations during 1844 (Walker & Johnson 1992: 135).

Molnar (1983: 225) estimates that 1.5 million deaths occurred in the large population centres of Mexico following the arrival of the Spanish. Other high estimates include 60% mortality among the Northern Plains and Great Lakes populations (Joralemon 1982); 74% among the Hopi Indians of Arizona (Dobyns 1983: 13;), 85-90% in Texas (Aten 1983); 98% among the Cree of Ontario (Young 1979) and 62% among the populations of the Northwest (Boyd 1990: 144). In reviewing the evidence for mortality from smallpox both Dobyns (1983: 13-14) and Cook (1973: 501) suggest an overall figure of 75% case-mortality among the virgin-

soil populations for the first smallpox epidemics throughout central and northern America.

The first known introduction into the western Pacific was to the Palau Islands in 1783. In 1853 smallpox killed up to 8% of the indigenous population of Hawaii; in the Carolines it was responsible for the death of up 40%; and on Guam 33%. Other introductions were to Easter Island in 1863-64 and New Guinea sometime before 1872 and later in 1893 and 1895. Quarantining of ships allowed Fiji to escape the introduction of smallpox and the disease killed only six in New Zealand when two ships carrying active cases arrived in 1872 (Fenner *et al.* 1988: 241-242).

Smallpox among the Aboriginal people of Australia was first recorded soon after the arrival of British colonists. The aim of this chapter is to document its spread among the Aboriginal populations of Southeast Australia, compare its dispersion to that of the European colonists, and make some assessment of the demographic effect on the Aboriginal populations.

3.2 Biology of smallpox

Smallpox was an acute disease specific to humans which often led to death within the first two weeks from the appearance of clinical signs. Those victims who survived, however, obtained a lifelong immunity to the disease and neither chronic nor recurring infection followed. Immunity to the disease was not as well guaranteed by vaccination, but in vaccinated individuals who developed the disease, it often expressed a much milder symptomatology and had lower case-fatality rate in its victims (Fenner *et al*. 1988: 272). The disease could only become endemic within a population when there was a large reservoir of susceptible hosts. This was regularly

achieved by new births and immigration of susceptibles in the larger population regions of the Old World.

Two main clinical-epidemiological varieties of smallpox have been recognized - variola major, the classic type of smallpox, and variola minor (Table 3.1).

Table 3.1 A classification of clinical types of variola virus infection (WHO 1977; Fenner *et al*. 1988).

WHO Classification	Clinical Manifestation
050.0	**Variola major** Ordinary type Modified type Haemorrhagic (pustular) smallpox Flat type Variola sine eruptione
050.1	**Variola minor** (alastrim)

The two strains of variola virus differed quite significantly in their virulence to humans. Variola minor, as its name suggests, causes mild symptoms in its human host and produces a case-fatality rate in unvaccinated individuals ranging from 0.1 to 2%. The more virulent variola major strain is a life-threatening disease. It often manifests severe symptoms, and produces a case-fatality rate among unvaccinated victims up to and in excess of 50%.

The virus is transmitted from person to person with no other vector involved. In most cases the virus enters the body via the oropharynx or respiratory tract passed on by respiratory discharges from infected individuals. Other means of transmission

are: direct inoculation, as in variolation; by the skin lesions of patients, by material which has been contaminated by the virus, by placental transmission, and by airborne spread (Benenson 1976:440, 1990:396; Fenner *et al*. 1988:186). The period of communicability is greatest during the first week of initial infection and continues for approximately 21 days. Susceptibility to the disease is universal but long-term immunity usually follows recovery and second attacks are rare.

There are five main sub-types of variola major (Table 3.1) distinguished by their clinical symptoms and prognoses. Ordinary type variola major is the most common. In typical cases of this type of infection there is an incubation stage that ranges from seven to seventeen days but usually between ten to twelve (Benenson 1990: 396). During this period of infection the virus replicates and spreads throughout the body via the lymphoid organs (spleen, bone marrow and lymph nodes). The onset of clinical symptoms is sudden. Fever, malaise, headache, severe backache, prostration and in some instances abdominal pain are the first to appear. These symptoms can easily be confused with influenza, meningitis and pneumonia (Benenson 1976: 443). After two to four days the fever is reduced and the characteristic stages of the smallpox rash begin. Starting as maculopapules first on the face, hands or forearms and then by centrifugal distribution to the trunk and lower limbs. The lesions are more abundant on the face and extremities than on the trunk. The papules then become vesicular within a day or two and then form pustules within 48 hours. The fever often returns during this stage. The pustules dry and begin forming scabs within eight to ten days of initial eruption. The matured lesions may be confluent, semi confluent, or discrete. Finally the scabs fall off at the end of the third to fourth week if the patient has survived. This type of variola major has been documented as having case-fatality rates of up to 62% (Rao 1972 cited in Fenner *et al*. 1988).

The flat type of variola major, distinguished by the lesions which project little, if at all, above the surrounding skin surface, is relatively rare but has a case-fatality rate reaching 98% (Benenson 1976:444; Fenner *et al*. 1988: 5). Equally as fatal is the haemorrhagic type which causes, either late or early in its development, subconjunctival bleeding, bleeding from the mouth, gums, nose, and blood in the urine (haematuria). In this type of variola major pregnant females are more susceptible than males and non-pregnant females. The modified type and variola sine eruptione are rare and occur principally in vaccinated individuals (Benenson 1976: 444; Fenner *et al*. 1988: 22-38)

The symptoms of variola minor were similar but much less severe than variola major. The onset was, like variola major, sudden with a fever, headache, backache, and sometimes vomiting. The sequence of development, and distribution of the skin lesions were also similar but their evolution was much more rapid. The eruptions became vesicular on the third day after the appearance of the papular stage and developed into pustules within twenty four hours. The final crusting stage was established on the sixth or seventh day of the rash. The facial lesions were often more sparse than in variola major and rarely reached the confluent stage. Secondary fever was rare and the victims usually remained ambulant and less affected during the course of the disease (Fenner *et al*. 1988: 38-39).

3.3 Smallpox among the European population of Southeast Australia

In 1984 Fenner stated that Australia has not loomed large in the global history of smallpox. A major reason for this may be seen in the slight effect the disease had on the European population of Australia. Since the arrival of the First Fleet in 1788 there

have been seven outbreaks of smallpox among the European population that could be considered serious (Table 3.2).

Table 3.2 Major smallpox outbreaks during nineteenth century in European population of Australia (Cumpston 1914, 1989; Curson 1985).

Year	Region	Cases	Deaths	Case Fatality
1857	Melbourne	16	4	25
1868-69	Melbourne	43	10	23
1871	Sydney, Melbourne	7		
1881-82	Sydney	154	40	25
1884-86	Sydney, Melbourne, Adelaide	123	10	8
1887	Launceston		11	
1893	Perth	52	9	17

All the outbreaks were due to importations by passengers arriving by ship. The 1881-82 outbreak which spread throughout the port suburbs of Sydney had the highest number of individual cases and a case-fatality rate of 25%. Most of the other cases were confined among ship's passengers who were quarantined. The containment of the outbreaks was largely due to the success of the various colonial government bodies who emphasized a strategy of quarantine and vaccination of victims and contacts rather than the routine vaccination of children (Hopkins 1983: 134). While this strategy was suited to a colonial population who had recently arrived from an endemic to a non-endemic country where the major threat was the periodic importation of the disease by sea, it was not suited to the Aboriginal populations who had no previous experience with smallpox and no means of prevention.

3.4 Smallpox among the Aboriginal populations - source material

In great contrast to its minor presence among the European population, smallpox has had a lethal history in Aboriginal Australia. Three unconnected epidemics attributable to smallpox were recorded among the Aboriginal populations of Southeast Australia (Fig 3.1). The first began in March or April 1789. A severe epidemic disease was seen by European colonists to be causing high mortality among the Aboriginal population living around the new settlement of Sydney (e.g. Butlin 1983: 63-68; Frost 1994: 190-210; Curson 1985: 41-53). It appeared quickly and lasted in the Sydney region for a few weeks. Later evidence suggests that it occurred along the coast line both north and south of Sydney.

Apart from a handful of written accounts by members of the First Fleet (e.g. Collins 1798; Hunter 1793; Phillip 1790b; Tench 1793) little is known about this epidemic. A few contemporary accounts survive, written by those who saw the disease active among the Eora around Sydney Harbour, and its effects in surrounding groups. The First Fleeters attribute the disease they saw to smallpox, although this diagnosis has not always been accepted by historians (Watt 1989: 145). Most writers (e.g. Butlin 1983; Cumpston 1914, 1989; Day 1996; Fenner 1984, 1985; Fenner *et al.* 1988; Frost 1994), however, have accepted that the disease was indeed smallpox, but debates mainly concerned with the origin of the disease, have continued (Butlin 1985; Curson 1985). Several possibilities have been put forward including; the accidental or deliberate release of variolous matter brought with the colonists of the First Fleet in January 1788 (Butlin 1983: 19-24; Day 1996: 62-64); a visiting French squadron which arrived at Botany Bay; seasonal fishermen reaching northern Australia from Makassar where the disease was endemic; and a type of 'native pox' presumed to be an endemic disease of Australia (Tench 1793; Cumpston 1914; Butlin 1983; Fenner 1985; Curson 1985). While each in turn have made articulate use of historical records citing evidence for their particular case, and in some cases speculating on evidence that does not exist, no one author or theory has

in the end prevailed over the others and the question of origin has remained unresolved. The limiting factor has been the historical records themselves which show no conclusive evidence either way. As far as this thesis is concerned the origin of the first smallpox epidemic, and indeed the subsequent outbreaks among the Aboriginal populations is not a central issue. It is far more relevant to examine the nature of the epidemics and to establish their spatial and temporal extent, and the demographic and social effects they had on the Aboriginal populations than to deliberate about their origin on evidence that does not sustain such an approach.

The second epidemic occurred forty years later, between 1828 and 1832 and was documented more extensively. It was first observed by European exploration parties (Sturt 1833 vol I: 93) on the river systems west of the Great Dividing Range in New South Wales, beyond the limits of the European settlements. Later, as the frontiers extended, observations of Aboriginal people bearing smallpox scarring, and oral accounts from those who survived, revealed that the epidemic had extended further inland along major river corridors to South Australia, and to the eastern coastline of the continent (Fig 3.1).

A third outbreak of smallpox was observed to be spreading among the Nawu, Wirangu, and Banggaria people along the western coast and inland regions of South Australia in 1866-67 (Fenner 1984: 732; Gething 1867). This epidemic was most likely the southern extent of an outbreak which was spreading among Aboriginal groups on the north and west coast of Australia and through the centre between 1860 and 1869 (Fenner 1984, 1985; Fenner *et al.* 1988; Cumpston 1989: 180).

3.4.1 The first epidemic - 1789

One year after the first European colonists established their settlement at Sydney Cove they noticed a severe mortality among the Eora seemingly caused by a deadly disease. The few contemporary accounts referring to the incident describe it as an epidemic of smallpox.

The 1789 outbreak of smallpox among the Aboriginal people was the first epidemic in Australia to be recorded. It remains among the most poorly recorded. Potentially, the most informative sources on the epidemic would have been the medical journals of the First Fleet Surgeons which to a large extent have not survived. The majority of written records that have survived are by contemporary observers who had no medical education or background and which generally consist of accounts within official reports, personal memoirs and letters. Nevertheless, these diarists wrote accounts of the epidemic based on their visual observations and on information they received from those who were medically trained. They should be accorded some credibility.

In April 1789 the Judge-Advocate and Secretary of the Colony, David Collins (1798: 53) wrote:

> Early in the month, and throughout its continuance, the people whose business called them down the harbour daily reported, that they found, either in excavations of the rock, or lying upon the beaches and points of the different coves which they had been in, the bodies of many of the wretched natives of this country.

Almost every boat that sailed on the harbour going about the daily business of the colony saw bodies lying about as if they had been abandoned where they had died. Returning from the Cape of Good Hope in the *Sirius*, Bradley (cited in Cobley 1963: 35) sailed in from the sea entrance of Port Jackson to the settlement at Sydney Cove and saw 'a great number of dead Natives ... in every part of the Harbour'. According to Newton

Fowell, a naval officer on board *Sirius,* in some cases the bodies were found with the remains of small fires on either side of them and containers of water left within reach (Irvine 1988: 113). Bradley saw none of the Aboriginal fishing canoes that had previously been a common sight on the harbour. In fact, the waters and shores of Port Jackson appeared to the Europeans to be devoid of any living Aboriginal person.

Collins visited the harbour shores to investigate Bradley's observations and reported:

> At that time a native was living with us; and on taking him down to the harbour to look for his former companions, those who witnessed his expression and agony can never forget either. He looked anxiously around him in the different coves we visited; not a vestige on the sand was to be found of human foot; ... not a living person was anywhere to be met with. It seemed as if, flying from the contagion, they had left the dead to bury the dead. He lifted up his hands and eyes in silent agony for some time; at last he exclaimed, `All dead! all dead!' and then hung his head in mournful silence (Collins 1798: 496).

The only Aboriginal people that were seen by Collins that day were the 'putrid bodies of those who had fallen victim to the disorder' (*ibid*: 496).

At first the cause of the mortality was unknown but the repeated accounts of Eora bodies about the settlement environs aroused the interest of the staff of the colony's hospital. A number of bodies were collected and brought into the settlement for the purpose of post mortem examination (Tench 1793: 145). No description of these post mortems survives.

Tench, a marine captain, who accompanied Collins around the shorelines gives a brief description of the symptoms in his account of the settlement:

> On inspection, it appeared that all the parties had died a natural death: pustules, similar to those occasioned by the small pox, were thickly spread on the bodies... (Tench 1793: 146).

In mid April a small group of Eora was found suffering from the disease in a cove near to the settlement. The Governor of the colony, together with a surgeon and Arabanoo (a captured Eora then living in the settlement), went immediately by boat to the place. Tench again recorded in his account that:

> Here they found an old man stretched before a few lighted sticks, and a boy of nine or ten years old pouring water on his head, from a shell which he held in his hand: near them lay a female child dead, and a little farther off, its unfortunate mother... eruptions covered the poor boy from head to foot; and the old man was so reduced, that he was with difficulty got into the boat (Tench 1793: 146).

The old man and the boy were then brought back into the settlement and placed under the care of the surgeons. The old man lived only a few hours after being brought into the settlement:

> By the encouragement of Arabanoo, who assured them of protection, and soothing behaviour of our medical gentlemen, they became at once reconciled to us and looked happy and grateful at the change of their situation. Sickness and hunger had, however, so much exhausted the old man, that little hope was entertained of his recovery. As he pointed frequently to his throat, at the instance of Arabanoo, he tried to wash it with a gargle which was given him; but the obstructed, tender state of the part rendered it impracticable (Tench 1793: 146)

Despite being 'covered from head to foot' with eruptions when he was brought into the settlement the young male survived the disease. Collins (1798: 54) attributes his survival to the care he received from Surgeon White during the course of his illness. There is no surviving record by White of this incident.

Towards the end of April Governor Phillip (1790b) records that two more Eora, an elderly man and a young female of ten or eleven years of age, were brought into

the settlement suffering from smallpox. Tench (1793: 148) recorded them as 'a young man, and the other his sister, a girl of fourteen years old'. Other accounts differ as to how many Aboriginal people were found suffering with the disease and were brought into the settlement. Collins (1798: 496) reported that 'two elderly men, a boy, and a girl were brought up, and placed in a separate hut at the hospital'; and Hunter (1793:134) recorded that 'two children, a boy of six or seven years of age, and a girl about ten, were lately picked up labouring under the same disease; two old men; whom we had reason to believe were the fathers of the two children, were picked up at the same time, and much care taken of them.' Whatever the number may have been, it is recorded by Tench (1793: 147), Collins (1798: 53), and Phillip (1790b) that, despite the care afforded them, the adults died of smallpox. Two children, however, showed signs of improvement soon after their arrival in the settlement and survived the disease.

Although there are no surviving descriptions of the symptoms of the disease by the surgeons who attended the Eora brought into the settlement's hospital, it is possible to reconstruct, with some detail, the course of the symptoms from the diarists. Among the first group of Eora brought into the settlement in mid April was the young male with eruptions covering most of his body (Tench 1793: 146). If this observation is accurate then the boy was past the initial symptoms of fever, malaise, and prostration which would have occurred in the first two to four days of symptomatic onset. Tench's account is not clear as to which stage the 'eruptions' were at, but because he was able to sit up and offer some assistance to the adult male, and because they covered most of his body we can assume that he was in an advanced stage of clinical signs with the lesions reaching maturity and was over the worse stages of the disease. No more is said of him in this respect other than that he recovered and was adopted into the family of the Surgeon-General of the colony. The accompanying adult male may have been in about the fourth day of clinical signs as Tench describes him in a condition of immobility and exhaustion

when he was brought in. The next day his condition deteriorated with the onset of secondary fever accompanied by periodic bouts of shivering. He remained prostrate and was unable to eat but drank water when it was offered. He remained conscious until he died. Of the second pair that was brought into hospital we know less. We can assume that they were in an advanced stage of the disease as Tench (1793: 148) describes them both as being in 'a most deplorable state of wretchedness from the small-pox'. After three days the young male died and the female recovered to be adopted into the family of a clergyman (Tench 1793: 148).

Arabanoo, who had closely attended the dying Aboriginal patients, also succumbed to the disease. He began to show the symptoms on May 12 and died six days later (Tench 1793: 149; Phillip 1790b). An incubation period of between 7-17 days, most commonly 10-12 days, before the onset of symptoms would suggest that Arabanoo caught the infection from the second couple to be brought in. According to Tench (*ibid*: 148) Arabanoo had shown more sympathy and care for them than he had for the former group brought in, and most likely had had more physical contact with them. A period of six or eight days followed during which the symptoms 'burst forth with irresistible fury' (Collins 1798: 54; Tench 1793: 149) before he died:

> During his sickness he reposed entire confidence in us. Although a stranger to medicine, and nauseating the taste of it, he swallowed with patient submission innumerable drugs, which the hope of relief induced us to administer to him. The governor, who particularly regarded him, caused him to be buried in his own garden, and attended the funeral in person (Tench 1793: 150).

The infection was limited almost entirely to the Aboriginal population. The only non-Aboriginal person to be infected was, according to Collins (1798: 496), 'a North-American Indian', a sailor belonging to the *Supply*, who manifested symptoms on May 2 and died a few days later (Phillip 1790a: 145). The source of his infection is not

known. Nothing is recorded of this man's movements in the days preceding his infection, or of his contact with the Aboriginal people within and without the settlement. Nor is there record of the treatment he received during his illness or whether he was isolated from the rest of the community. But, even though the disease is highly communicable during the first week after the onset of symptoms, there is no record of the sailor transmitting the infection on board the *Supply* nor within the settlement.

That the settlement escaped the epidemic was largely due to the low level of personal contact the Europeans had recently had with the Eora. Governor Phillip (1790b: 159) was concerned with the lack of friendly contact and rapport that had so far been established. One of his tasks on establishing the settlement was to initiate a friendly relationship with the Aboriginal people, to conciliate their affections, and to learn as much about them as he could for the benefit of the colony (George III: 1787). At first he was able to meet them on amicable terms but the Eora remained generally aloof from the Europeans and avoided the settlement. No mutual understanding of their respective cultures developed and soon the goodwill, which had at best been tenuous, deteriorated into a series of violent skirmishes around the settlement lasting several years (Clark 1981: 116; Day 1996:: 49-68; Reynolds 1987: 32-40). Because of this poor relationship Phillip had decided to capture one of the Eora and bring him into the settlement in order to break down the communication barrier that had resulted and try once again to establish close association. Arabanoo, then, was the first of several Eora whom Phillip captured.

Nor did the ill Eora who were brought into the settlement hospital transmit smallpox further. They were immediately isolated in an unoccupied building next to the hospital. The colonists were well aware of the contagious nature of smallpox as Collins comments:

> It was not a desirable circumstance to introduce a disorder into the colony which was raging with such fatal violence among the natives of the country (1798: 53)

It is not known how many of the Europeans (about 1,000 in all) were immune to smallpox, having either previously survived an infection or having been successfully inoculated against it. There was, however, one potentially susceptible group in the settlement - the children and infants who had either been born in the settlement or on the voyage out. There is no record of any inoculation procedure performed on them. Collins (*ibid*: 53) reveals that:

> Notwithstanding the town of Sydney was at this time filled with children, many of whom visited the natives that were ill of this disorder, not one of them caught it...

While the arrival of Eora in the settlement would have been the cause of great curiosity for adults and children alike it is unlikely that they would have risked, or been allowed, close contact with them when they clearly showed signs of a disease that was feared and known to be highly contagious.

The epidemic existed in the Sydney region during March and April 1789 and appears to have subsided by early May. Its extent, however, appears to have been far greater than the environs of Sydney. Cumpston (1914: 2) refers to evidence of its existence far to the south-west in South Australia and Butlin (1983: 24) refers to evidence of its presence to the north in the Hunter Valley region; but neither author indicates a source for these assertions. The diarists of Sydney no longer report seeing active cases of smallpox among the Aboriginal people after May 1789, but the characteristic traces of the disease were often seen in survivors.

In June 1789 Governor Phillip led an exploration party to Broken Bay, a large inlet, and mouth of the Hawkesbury River, twenty five kilometres north of the settlement. They left their boat and crew at the northern end of Port Jackson and travelled first along the beaches then overland using the well established Aboriginal trails to meet their boat at Broken Bay. Collins (1798: 52) writes that the path they followed 'was in many places covered with skeletons, and the same spectacles were met with in the hollows of the rocks of that harbour.' Hunter, who also accompanied the party, recounts their meeting with a group who had been fishing in the waters of the bay. On seeing the boat approaching:

> ...they all made their escape, except this miserable girl, who had just recovered from the small-pox, and was very weak, and unable, from a swelling in one of her knees, to get off to any distance ... she appeared to be about 17 or 18 years of age, and had covered her debilitated and naked body with the wet grass, having no other means of hiding herself (Hunter 1793: 138-139)

Governor Phillip (1790b) commented that they had seen the traces of smallpox wherever they had been. Further to the north-west in the Wellington Valley some 300 Km from Sydney and on the western side of the Great Dividing Range, Mair, a military surgeon, reported seeing three old men with pock marks when he was investigating the second epidemic in 1830 (Mair 1830, & see below). Each claimed to have had the disease when they were young and each in turn was among the few who were not infected by this outbreak.

Well south of the Sydney-Botany Bay region the situation was similar. In 1803 Lieutenant Grant, aboard the *Lady Nelson*, entered Jervis Bay, 160 kilometres south of Port Jackson. He remarked that many of the Aboriginal men and women he saw were marked with smallpox scars (Grant 1803). In February of the same year Flemming of the *Cumberland* was on a voyage of exploration between Sydney and King Island in Bass Strait. He landed at Port Phillip Bay on the south coast where he met a group of eleven

Aboriginal people, probably Woiworung. Two of them he states appeared to be marked with smallpox sequelae (Flemming 1878: 16). Although Flemming does not say so, the scarred individuals he saw were most likely adults and probably males as they were carrying spears. If these reports are accurate, and there appears little reason for doubt, then these Aboriginal people would have been children when the 1789 smallpox epidemic reached the lower east and south coast of Australia. It is highly likely then that they were the survivors of the 1789 epidemic.

If these observations at Broken Bay, Wellington Valley, Jervis Bay, and Port Phillip are correct, then smallpox appears to have spread among the Aboriginal populations, at least as far inland as the Wellington Valley in the north and Port Phillip on the southern coast. Both points were well beyond the European frontier in 1789. Apart from Cumpston's (1914:2) assertion of the 1789 smallpox epidemic reaching South Australia there is no evidence to suggest that this epidemic broke out of the coast and highlands belt and penetrated into the interior of the continent (Curson 1985: 51).

The epidemic was severe for the Eora living around the Sydney settlement. The First Fleet records leave us with a stark picture of high morbidity and mortality from the disease. Governor Phillip (1790b: 159) wrote:

> It is not possible to determine the number of natives who were carried off by this fatal disorder. It must be great; and judging from the information of the native now living with us... one half of those who inhabit this part of the country died...

While this was a crude estimate by Phillip, it may have not have been so inaccurate. If this was the first episode of epidemic smallpox among this population then they would not have been immunologically protected against it and high morbidity and

mortality would be expected. The effects of variola major among other unprotected and virgin-soil populations elsewhere in the world has resulted in extraordinarily high mortality (Black 1975: 517; Butlin 1983: 65; Dobyns 1983; Fenner 1984: 730-733; Fenner *et al*. 1988: 1069-1102; Snow & Lanphear 1987; and above). In these circumstances the disease affects both sexes and all age groups of the population and case-fatality rates ranging from 25 to 90% can be expected (Koplan & Foster 1979: 440; Fenner *et al*. 1988:5)

How severe then was the mortality among the Eora around the Sydney settlement? If we accept Phillip's estimate of 50% mortality then how many deaths does this represent? In May 1788, just weeks after the settlement was established, Governor Phillip (1788a) estimated the number of Aboriginal people living in the vicinity of Botany Bay, Port Jackson and Broken Bay, to be '... no less than one thousand five hundred'. There is no way of knowing how accurate this was, but once again Phillip may have been close in his estimate (White & Mulvaney 1987: 115). More recent estimates by Kohen and Lampert (1987: 345) based on area/density calculations suggest a population of between two and three thousand people belonging to three main linguistic groups (Tharawal, Dharug, and Kuring-gai) living between Jervis Bay in the south to Broken Bay in the north. Other estimates (see Turbet 1989: 26-28) have a population of between 2,600 and 5,200 for an area of 1,300 square kilometres of coastal strip around the settlement. While there are certainly inaccuracies in these estimates they point to a substantial number of people inhabiting the coastal plains and hinterland either side of the Sydney settlement. With a postulated fatality rate of 50% (similar to the Americas) a likely number of deaths from the smallpox epidemic would therefore lay somewhere between 750 and 2,600.

The realisation that many had died in this epidemic remained with the European colonists. In 1822 a Russian visiting the Sydney recorded:

> Many of those who had settled in close proximity to the English colonists became infected with smallpox and thousands died, several generations perished (Hotimsky 1967: 93).

Whatever the real extent of mortality may have been it was certainly high. Apart from seeing the many bodies around the shores of Port Jackson, the colonists had the accounts of the friendly Eora to rely on for an estimation of the severity of mortality:

> As a proof of the numbers of those miserable people who were carried off by this disorder, Bennillong told us, that his friend Cole-be's tribe being reduced by its effects to three persons... (Collins 1798: 497).

Again in April 1791, when the first exploration expeditions reached the Nepean River region some fifty kilometres inland from the settlement, they were informed by their two Aboriginal guides that:

> this part of the country was inhabited by the Bidigals, but that most of the tribe were dead of the smallpox (Hunter 1793: 340-341).

The other writers seemed equally shocked by the epidemic and report indications of high mortalities (Irvine 1988: 113; Tench 1793: 146-149). The mortality in regions further away from the settlement remains unrecorded and so unknown.

The response to the epidemic by those who did not die immediately was to abandon the coastal regions near the settlement. Immediately after the epidemic was noticed by the Europeans Port Jackson and the environs of the settlement appeared deserted by the Aboriginal people. How far and to where they fled is unknown, but whatever the case it was not for long. Small groups of Aboriginal people were soon

sporadically sighted around Sydney and Botany Bay by exploration groups travelling outside the settlement (Hunter 1793: 165-167). Some four to six weeks after the epidemic Aboriginal people began to be seen again on the waters of Port Jackson. On 2nd June Bradley wrote in his journal that:

> Twenty canoes passed Sydney Cove going down the Harbour; this was the first time any number of them had been seen together since the small Pox having been among them (Cobley 1963: 43).

In November Hunter (1793: 167) recorded a group of twenty to thirty on the shores in a hostile encounter. Later that month a group was met under more friendly terms and two males, Benelong and Colbee, were enticed back into the settlement. One year after the epidemic a group of three hundred Aboriginal people, including Benelong and Colbee, who had escaped from the settlement, was seen at Manly Cove, Port Jackson, feasting on a whale that had recently become stranded on the beach (Collins 1798: 109; Willey 1979: 111). However high the mortality had been, it would appear that large numbers of Aboriginal people were again occupying the coastal regions of Port Jackson, as little as twelve to eighteen months after the epidemic was first noticed.

3.4.2 The second epidemic - 1828-32

In early 1829 the explorer Charles Sturt returned from an expedition into the north west of New South Wales and brought back with him what was probably the first indication of a second epidemic of smallpox among the Aboriginal populations. He and his party had traced the course of the Macquarie River into the interior and had reached the Darling River near the present town of Bourke. On February 5th while he was following the Darling southward he encountered a group of seventy Aboriginal huts which appeared to Sturt to be permanent habitations. Further on he encountered a group

of Barundji which he believed to be the inhabitants of the village. Sturt and his party were approached by an elderly male. Sturt recorded:

> As his tribe gathered around him, the old chief threw a melancholy glance upon them, and endeavoured, as much as he could, to explain the cause of that affliction which, as I had rightly judged, weighed heavily upon him. It appeared, then, that a violent cutaneous disease raged throughout the tribe, that was sweeping them off in great numbers. He called several young men to Mr. Hume and myself, who had been attacked by this singular malady (Sturt 1833 vol I: 93)

Four or five days later while resting in camp another group of about seventy approached Sturt's party. The women and children of the group passed the camp by, but Sturt noticed that several of the men who came nearer were afflicted with what he believed to be the same 'violent cutaneous eruptions' he had just recently seen. Sturt believed that this disease was smallpox (1838: 147). He was of the opinion that it was causing a severe decline in the population as the numbers of Barundji he saw on the Darling River bore no comparison to the size and numbers of the huts he had seen (*ibid*: 105).

Further indications that a second epidemic of smallpox was occurring among the Aboriginal populations west of the Great Dividing Range were brought to the notice of the Colonial Government in 1830 (Mair 1831). In August of that year five Wiradjuri were observed by a local pastoralist, Andrew Brown, suffering from what was thought to be smallpox. Two were described as being in the incipient stages of the disease and the other three in the more advanced stages. One of these was later seen with smallpox sequelae on his body, and when questioned he indicated that the others had died. Two months later, in October, the same symptoms, together with a high mortality were again reported among the Wiradjuri. The disease was active until at least December (Mair 1831). In August of 1831 the same symptoms were again witnessed by Brown in three

Wiradjuri who had been in contact with others recently arrived from the Lachlan River to the south. The disease was reported to be causing high mortality among the Lachlan River Wiradjuri, many of whom were fleeing the region.

A further indication that the disease was smallpox was recorded by Mair (1831). A European family residing near Bathurst was struck with the infection (Mair 1831). Four of the family showed eruptive and febrile symptoms and one, a two year old female, died. The first of the symptoms to appear was the rapid onset of fever to be followed by the successive stages of the rash reaching the confluent stage after ten days. The child's eyes were closed after the sixth day and the eruptions extended over most of her torso, feet, face. The eruptions were particularly severe on the gums, lips and tongue. The mother described the disease as being like a number of scalds running into each other and a discharge emanating from them (Mair 1831). The disease had arisen after the family had sheltered in their house a Wiradjuri child, whose parents had died from a disease with similar symptoms, and who likewise later died of the same disease.

A concerned New South Wales administration sent Dr John Mair, a military physician attached to the 39th Regiment, to investigate and report on the disease in October 1831. Mair was too late to witness first-hand the disease among the Wiradjuri but from accounts by eyewitnesses, whom he deemed to be credible observers, he pieced together the symptoms of the disease (Mair 1831). While there appeared to be a variety of forms of the disease in different individuals, the following description of the clinical symptoms were common to all. The initial pre-eruptive stages were characterised by a general malaise and fever lasting from 2 to 8 days accompanied by loss of appetite, headaches, chest and abdominal pains. The eruptive stage followed, with focal lesions of small 'red spots' (papules) resembling 'flea bites' commencing on the face and gradually spreading over the head, breast and extremities. Enanthema occurred at this time with

lesions developing on the tongue and lips. The rash spread and in many instances the soles of the feet were observed to be studded with lesions. After the eruption had developed, usually observed to have occurred in twenty-four hours, the fever subsided. At this time the patient experienced pain in the throat, most likely from enanthema on the pharynx, making it difficult to swallow solid foods. After three to eight days the 'red spots' developed into raised vesicles containing a milky fluid in some, and in others a 'yellowish' or 'straw coloured' fluid. At their height the lesions were the size of a 'small or large pea'. At this stage it was recognized by observers, who were familiar with smallpox in England, to be that disease. Scabs formed from the vesicles at different periods according to the length of time it took to reach maturation. These were occasionally confluent on the nose and cheeks and frequently left permanent scars or indurations on the skin. Mair observed these on some of the subjects and stated they 'cannot be distinguished from the pits of Small Pox'.

Mair's conclusions were, however, not unchallenged. Dr George Busby (1831) was also requested by the Government to furnish particulars on the epidemic. In a report to the Inspector of Colonial Hospitals, Sydney, he concluded that the disease was varicella and not smallpox. Busby's observations, however, were not as extensive as Mair's. He saw only two Wiradjuri from the Bathurst district showing clinical symptoms but could not determine the nature of their affliction. He based his diagnosis solely on the symptoms he monitored in a European male, Titman, who was confined in the Bathurst Hospital on August 6 1831 under Busby's care. Titman was 40 years old and had been living in the same house as an Aboriginal person who had caught the disease and later died. He informed Busby that he had suffered from smallpox as a boy, being one of a family of six, all of whom had the disease at the same time. Titman's symptoms were generally milder than those described by Mair in the Wiradjuri. He complained of headache, pains in the back and limbs, lassitude, loss of appetite, nausea, and fever.

Primary fever was, however, severe and lasted until the eruptive phase which was followed by secondary fever. The vesicles appeared earlier, achieved maturity quickly, covering most of the body, and were particularly numerous on the face, shoulders, back, and lateral surfaces of the legs. The symptoms lasted for fourteen days terminating in full recovery (Busby 1831).

Busby's diagnosis was that the disease he had observed was not small pox but varicella. His view was based firstly on the modified nature of the symptoms and secondly on Titman's previous infection with smallpox. In response to the prevailing epidemic among the Aboriginal population he concluded:

> I am, upon the whole, at present disposed to regard the eruptive disease lately prevalent among the black natives in this district as varicella, but possessing by no means a malignant character, nor likely, under ordinary circumstances of comfort and attention attainable in civilized society, to prove fatal in more than a few instances; the mortality it has occasioned among the blacks being sufficiently accounted for by the unfavourable circumstances in which they are placed (Busby 1831).

Mair (1831), on the other hand, saw it as a case of secondary modified smallpox because it passed through the regular stages, albeit quickly, of ordinary smallpox. He also examined the patient about six weeks after his discharge from hospital. He described the skin on Titman's face having a `mottled appearance of red and white, burning scales were separating from it, and numerous small pits or depressions could be discerned' some of which were recent and others older which further strengthened his diagnosis.

Busby's conclusion that the disease affecting the Wiradjuri was varicella was supported by the Inspector of Colonial Hospitals, Dr. James Bowman. Bowman (1831) disputed Mair's diagnosis of smallpox and cast doubts upon the reliability and integrity of

his witnesses. Writing to the Colonial Secretary he claimed that Mair and Imlay were incorrect in their diagnosis and had formed their opinions that the disease was smallpox from information they received from persons incapable of determining so important a point, despite Mair's assertions to the contrary . Bowman also suggested the disease among the Wiradjuri was not smallpox but varicella which, while common in the colony at that time was of little threat. He called it 'native pock' (incorrectly believed by some to be a local form of varicella).

He presented these arguments to the Governor and members of the Executive Council of the colony in December 1831 (Thompson 1831). The Council readily agreed with Bowman and passed over the whole matter of the epidemic apart from recommending a Government notice should be published stating that suspicion had arisen as to the current prevalence of smallpox. They recommended that voluntary vaccination could be obtained from any of the Colonial Surgeons. They also agreed that measures should be taken 'generally to induce the Aboriginal Natives also to submit to vaccination' (Thompson 1831).

Mair (1831) reported varied clinical symptoms among the Aboriginal populations. Among those in the Wellington Valley the vesicles began to coalesce on the face during the eruptive stage to be followed in a day or two by excessive salivation. An escaped convict who had lived among these people before, and during the epidemic, described 'water pouring from the mouth as they lay on the ground' (Mair 1831). About the tenth or twelfth day after the initial symptoms were noticed, many patients were seen to experience convulsions and the fluid discharge from the mouth became more bloody and viscid in appearance. The bloody discharge from the mouth and its late onset may be indicative of bleeding from the oral mucous membranes, a symptom of the more fatal

haemorrhagic type of variola major (Benenson 1976: 444, 1990: 395; Fenner *et al*. 1988: 38, 138-9).

Among the Wiradjuri living on the Lachlan River and Wellington Valley regions, death generally resulted after the third day of the eruptive stage. Mair (1831) reported that secondary fever was seldom observed amongst these groups, and when it did occur he suggested it was due to local low ambient temperatures. The rarity of secondary fever, which usually begins on the seventh or eighth days in severe cases (Fenner *et al*. 1988: 22), can be explained by the early fatality of most of the victims. Many were reported to have died at the very onset of the disease before the beginning of the eruptive stage.

Most of the eye-witnesses consulted by Mair remarked that the disease proved chiefly fatal to adults and elderly, and seldom to children even though their length of exposure to infected individuals was the same. It was, however, reported to Mair by several of the observers that many adults who bore pockmarks on their skin, evidence of a previous exposure to smallpox, escaped the disease altogether.

The usual duration of the disease was stated to be between fourteen to twenty-one days in cases where the patient survived and was restored to health. Many patients who did survive were unable to walk for a considerable time due to the separation of the epidermis from the sole of the feet. In other survivors more of the characteristic forms of smallpox sequelae were present. Severe keratitis and/or corneal ulcerations were reported in several victims who recovered from the infection. Brown stated:

> ... I found one middle aged woman who had lost her sight altogether by it, one who had lost an eye, and two children male and female who had each been deprived of an eye by it (Mair 1831).

In others secondary infections left the sufferer with ulcerations in different parts of the body where the smallpox lesions had occurred.

It had been noted by most of the eye witnesses of the epidemic that fatality occurred chiefly among adults and the aged, and seldom among children. In others who escaped infection it was observed that some had been exposed to smallpox before. Brown, who observed the disease first hand (Mair 1831), noted that it extended to most of the Aboriginal people he saw. He did, however, see three old men with 'evident marks' who informed him that they had been infected by the same disease when very young. Others who escaped infection had been vaccinated (or variolated) by European settlers, several years prior to the epidemic. During the epidemic Mair vaccinated many of the Aboriginal people he encountered. He met with little opposition among them, most of whom had come to realize the benefits that the simple procedure conferred in the face of the serious morbidity and mortality caused by the epidemic. After his return to Sydney, Mair sent supplies of recent and dried vaccine lymph to the Colonial Assistant Surgeon at Bathurst for future use (Mair 1831).

The government surveyor and explorer, Thomas Mitchell, also observed Aboriginal people suffering from the epidemic. On December 5, 1831 Mitchell and his expedition crossed the Liverpool Range, north of Bathurst, which at that time divided the settled from the unexplored districts of the colony. He recorded:

> We reached at length, a water-course called "Currungai" and encamped upon its bank, beside the natives from Dart Brook, who had crossed the range before us, apparently to join some of their tribe, who lay at this place extremely ill, being affected with a virulent kind of small-pox. We found the helpless creatures, stretched on their backs, beside the water, under the shade of the wattle or mimosa trees, to avoid the intense heat of the sun. We gave them from our stock some medicine; and the wretched sufferers seemed to place the utmost confidence in its efficacy (Mitchell 1838 vol I: 26).

According to Mitchell these people he saw were from another region as they had little knowledge of the countryside they were now in. Mitchell continued his journey to the north reaching the Gwyder River but made no further mention of smallpox among the Aboriginal groups he encountered.

From these districts the epidemic spread along the river systems far in front of the colonial frontiers. First hand observations of the epidemic by Europeans were now no longer possible. From the upper Darling it spread downstream. In 1835 Mitchell was again exploring beyond the frontier following the southerly course of the Darling River. On May 28 while at his base camp at Bourke he was visited by a group of Aboriginal people. They consisted of four adult males, seven females, and children. Mitchell remarked that most of them had visible smallpox scarring 'but the marks were not larger than pin heads' (Mitchell 1838 vol I: 218). From Bourke, Mitchell and his expedition continued along the Darling River as far as Menindee, 190 Km above its junction with the River Murray. He turned back on July 12 after an affray with the Barkindji. He had seen smallpox scarring on many of the Aboriginal people during the journey. At Menindee he recorded:

> These natives, as well as most others seen by us on the river, bore strong marks of the small-pox, or some such disease, which appeared to have been very destructive among them. The marks appeared chiefly on the nose, and did not exactly resemble those of the small-pox with us, inasmuch as the deep scars and grooves left the original surface and skin in isolated specks on these people, whereas the effects of small-pox with us appear in little isolated hollows, no parts of the higher surface being detached like islands, as they appeared on the noses of these natives (ibid: 261).

Mitchell may have observed the sequelae of confluent type smallpox. He was of the opinion that the disease had been severe and had caused a great mortality among the groups living along the Darling River.

The epidemic did not reach the Murray River populations until at least the second half of 1830. In January of that year Charles Sturt (1833 vol II), on his second expedition of exploration, began a journey down the Murrumbidgee and Murray rivers. He arrived at the termination of the river systems, at Lake Alexandrina in what was to be the colony of South Australia, on 9 February, passing the Darling River junction on his way. Sturt was an astute observer and documenter, describing the landscape, geology, the people and their state of health in some detail (Beale 1979). Along the Murray he noted that 'the most violent cutaneous eruptions' were affecting many of the Aboriginal people he encountered and remarked on their 'miserable state of disease and infirmity' (ibid: 148). Sturt was in two minds as to what the disease actually was, describing it in one instance as leprosy (ibid: 96) and in another as syphilis (ibid: 125). He made no suggestion, however, that it was due to smallpox, a disease he was surely familiar with among Europeans in Britain and had seen just one year before among the Aboriginal populations of the upper Darling. Nor did he mention seeing any Aboriginal people with smallpox-like sequelae that would have indicated a previous epidemic. In a later comment on smallpox (see below), however, Sturt makes a strong implication that the disease was smallpox. Sturt returned the way he had come, along the Murray and Murrumbidgee system and arrived back in Sydney in late May.

In 1830 the middle and lower Murray River corridor was well beyond the European frontier and the spread of smallpox went unobserved, and hence unrecorded by Europeans. There is, however, some indication that the epidemic had reached the populations of the lower Murray well before the first European settlement was established on the Adelaide plains in 1836. In February 1838 Joseph Hawdon (1838) overlanded the first herd of cattle along the Murray to the newly established settlement of Adelaide. On February 11 Hawdon set camp near the present town of Swan Hill on the Murray and met up with a group of Wadi Wadi:

> In the evening some of the Blacks came to Swan Hill, where we were encamped. After holding a little conversation with us across the river, they swam over to us. They were fine, well-made men about five feet eleven inches in height. Their faces were nearly all marked with smallpox, but otherwise their features were pleasing (ibid: 27)

The next day Hawdon saw 52 men, accompanied by their women and children and described them as 'not a good-looking set of men' and many of them 'blind in one eye' (ibid: 28). Hawdon makes no further mention of smallpox sequelae although he met several more large groups along his way.

Alone, Hawdon's observations of smallpox sequelae could not be accepted as being reliable. However, later the same year (1838) Charles Sturt followed Hawdon's trail along the Murray corridor, also bringing cattle to the colonists at Adelaide (Sturt 1839; Kenyon 1925: 178). His observations of smallpox sequelae support Hawdon's. Approximately 5 kilometres below the junction of the Murray and Murrumbidgee rivers he came across a large group of Aboriginal people:

> ... in the course of the forenoon we were joined by various parties from different quarters that when united formed a considerable body of athletic and well proportioned men. They came with the most peaceable intentions and several of them assisted us in our work. I observed many of them as if pitted by the Small Pox, or that would appear as the disease which was having such a fearful effect upon them when I was on the banks of the Darling in 1828 and the Hume [the Murray above the junction of the Darling] in 1829... It must have committed dreadful havoc amongst them, since on this journey I did not see hundreds to the thousands I saw on my former expeditions (Sturt 1838: 147).

Smallpox was present on the eastern coast by late 1831 (Mair & Imlay 1831). In October of that year two Aboriginal men brought an ill convict, Richard Scarme, to the European settlement at Port Macquarie. Scarme had escaped from the penal settlement at Moreton Bay some time before and had been living with a group of Dainggatti near the mouth of the Macleay River at Trial Bay, sixty kilometres north of Port Macquarie. He

stated that he had seen more than fifty ill with smallpox, a disease he had seen in England before he was transported. The military officer in command of Port Macquarie, Captain Smyth, stated that Scarme was recovering from a disease that bore a 'striking resemblance to the Small Pox' (Mair & Imlay 1831). Scarme was held in custody and later sent to Sydney where he was seen by Mair and Imlay. They examined him and reported that his skin bore unequivocal marks of the recent epidemic of smallpox. Scarme said he had not previously suffered from smallpox or cowpox and he bore no vaccination scar.

Six weeks after they had brought Scarme into Port Macquarie, the same two Aboriginal men returned to the settlement. Captain Smyth, described them as covered from `head to foot' with the disease. By late December the Dainggatti living around the Port Macquarie settlement were in turn infected. Many of the worst cases recovered but five died, all of whom were young and had previously been in good health. The disease did not affect the European population. Smyth isolated the Dainggatti by keeping them on the opposite bank of the river which flowed through the settlement (Mair 1831).

As the European frontiers expanded in later years many of the settlers noted smallpox scarring among Aboriginal people with whom they had frequent contact (Brough Smyth 1876: 253-257; Cumpston 1914: 147-162). Peter Beveridge, a squatter who held a property on the Murray River below Swan Hill for 23 years from 1845, and who acquired an extensive knowledge of the Aboriginal people, commented on the smallpox epidemic and its timing:

> All the very old aboriginals in the Colony [Victoria] show very distinct traces of small-pox, and in speaking of the scourge which has so indelibly left the marks of its foul presence, they say that it came with the waters, that is it followed down the rivers in the early flood season (about July or August), laying its death-clutch on every tribe in its progress...(Beveridge 1877)

Further evidence of the epidemic spreading along the Murray comes from accounts by Aboriginal survivors. In South Australia a survivor of the epidemic, a Ngarrindjeri woman named Clulwuwyrie (Jenny Pougie) died in 1911. She remembered the first visit by Sturt to the Murray in 1830 when she was a child and related that the disease, which left her with pock marks and carried off many of her people, came soon after (*Adelaide Observer* 1911). Another woman, Kontinyeri (Louisa Karpeny), possibly the niece of Clulwuwyrie, could also recount her experience of the epidemic (Stirling 1911). She lived most of her life on the southern shore of Lake Alexandrina and as a small child could remember her first sight of white Europeans, soldiers arriving at the lake at the time the colony of South Australia was established in 1836. She described to Stirling the coming of the sickness which was several years before the arrival of the soldiers:

> The old black spoke of the coming of a strong *West* wind which made the reeds all tremble, and this, she said, was taken as a sure sign that the sickness was coming... She described how the faces of those affected with the disease came out all over spots, and how that many died of it, including many children (Stirling 1911: 18-19).

Another survivor, an old male named Malo, who lived at Moorundie on the lower Murray, related that when he was a small boy there was a sickness amongst his people which killed many and left him with scarring on his face. Doctor De Lisle of the 96th Regiment examined the scarring on Malo many years later and concluded that he had undoubtedly suffered from smallpox (Hawker 1901). Three old Aboriginal men from nearby Swanport often spoke to Europeans about a great sickness which came along the Murray causing a great number of deaths. Such was the scale of mortality that the living could not cope with disposal of the dead. They describe that those who were inflicted by the disease broke out in spots all over their bodies (Stirling 1911: 17). Another account comes from a thirty-five year old male from Lake Alexandrina who bore smallpox

sequelae on his face. He related to Stirling that as a small boy a 'big one wind' came from the east carrying the sickness and caused the marks on his face (Stirling 1911: 19).

George Augustus Robinson noted the presence of smallpox sequelae during his journey through the southern highlands. In 1844 he spent three days among a group of 300 Aboriginal people near the present town of Yass. He noted, 'the virulent effects of Variola or Small Pox was apparent' (Robinson 1844; Mackaness 1941:26). Near Gundagai he reported that the Aboriginal populations in the Murrumbidgee valley and surrounding highlands were 'strongly marked with smallpox' (Robinson 1844; Mackaness 1941:28). Robinson does not give the ages of the individuals he saw bearing smallpox sequelae but as his sightings came a decade after the last epidemic as opposed to 55 years since the first, the former occurrence of the disease would most likely have been the cause.

As the European frontiers extended north into Queensland many of the colonists saw the tell-tale sequelae on the faces of the survivors. During the 1850s and 1860s pockmarked Aboriginal people were regularly seen and reported along the coastal regions as far as the Gulf of Carpentaria. Inland the extent of the pock marking was seen north of the Darling, along its tributaries and near Cloncurry (Butlin 1983; Campbell 1983 & 1985).

There is little evidence of the severity and extent of the epidemic south of the Murray River and its Victorian tributaries. There is no evidence at all that the 1830s epidemic reached the Kurnai people living in the Gippsland ranges in eastern Victoria, and indeed this region appears to have escaped altogether (Butlin 1983: 23; McBryde 1984: 277). Sharp social, political, linguistic, and economic boundaries between the Kurnai groups of Gippsland and surrounding populations, resulting in mutual hostility

prior to European contact, have been demonstrated by several researchers (Howitt 1889,1904; McBryde 1978, 1984; Smyth 1878). This disjunction of the Gippsland populations from their neighbours would most likely have acted as a deterrent to other Aboriginal groups fleeing the epidemic to enter their territories and so acted as a barrier to the transmission of smallpox.

The epidemic also seems to have missed the groups living in South Australia's mallee districts south of the River Murray valley. Unlike inland New South Wales, no active cases of smallpox were seen by the early settlers in Victoria. This is not surprising as the European settlement was only permanently established in 1834 at Port Phillip Bay (Melbourne) well after the epidemic was at its peak. A few of the settlers do, however, report seeing Aboriginal people bearing the characteristic sequelae which would be expected if there had been a previous severe epidemic of smallpox.

Before this settlement there had been a failed attempt to establish a southern outpost of the colony of New South Wales in September 1803 at Port Phillip Bay. William Buckley, a convict at this settlement, escaped in that same year and lived with the Wathaurong near the present city of Geelong for 32 years. After his return to European life he became known as the 'wild white man'. He recollected:

> I never observed any European contagious disease prevalent in the least degree; and this I thought strange. There was at one time however, I now recollect, a complaint which spread through the country, occasioning the loss of many lives, attacking generally the healthiest and strongest, whom it appeared to fix upon in preference to the more weakly. It was a dreadful swelling of the feet, so that they were unable to move about, being also afflicted with ulcers of a very painful kind (Morgan 1852: 94-95)

Buckley's description of the disease is vague. Although there is no other evidence for it he may be referring to an epidemic that had preceded his association with

the Wathaurong and one he had heard about through their recollections. Smallpox does affect the soles of the feet (Benenson 1990:395; Fenner *et al* 1988: 38-39) and if secondary bacterial infection occurs could cause the ulceration and swelling that Buckley describes. Such a symptom, one among the many of smallpox, may be all that Buckley was informed of, or all that he could recollect being told. There are other problems with Buckley's account. The record of his life among the Wathaurong was written for him after his return to the European colonists by Morgan, who had interviewed Buckley at length and it is impossible to discern just how much Morgan's impressions and conceptions misconstrue Buckley's reminiscences. Therefore, any diagnosis of smallpox made upon this evidence can only be uncertain at best; yet it is still intriguing that Buckley did not identify the disease as smallpox.

Several of the early colonists of Victoria do refer to smallpox south of the river systems. Dawson (1881), in describing the Aboriginal populations of western Victoria, briefly discussed smallpox and thought the epidemic had caused high mortality on the south coast, particularly on the coastal dunes to the east of Port Fairy. It was, however, a common misconception of the time that many of the extensive Aboriginal burial areas in the coastal and riverine dune systems, often with exposed skeletons on the surface, contained the victims of smallpox and were a direct consequence of the high mortality wrought by the epidemic (Moulden 1877; Dawson 1881; Stirling 1911; Cleland 1914). Radiocarbon dates from several of these burial sites have since shown an extended antiquity (Blackwood & Simpson 1973; Pretty 1976; Dowling 1990), in some cases dating back into the late Pleistocene (Pretty 1977), indicative of a long and consistent use for burial purposes (Pardoe 1988). Edward Curr (1886), a Victorian pastoralist, compiled two volumes on the Australian Aborigines. He discussed smallpox among the Aboriginal populations but cited no

strong evidence for the 1830s epidemic in Victoria and relied mainly on secondary evidence from New South Wales and the Murray River corridor for his information.

As well as leaving marks on the faces of the survivors the epidemic left its traces in the oral traditions of the Aboriginal people. Among the Aboriginal groups affected by the epidemic, smallpox was given many names (e.g. Curr 1886; Cleland 1928: 67-70). In the river districts of central and western New South Wales, where the epidemic was first recorded, it was called *Booert*, and on the Darling River where the first active cases were seen by Sturt it was called *Mungga*. North of the Darling among the Muruwari of the Narran River region it was called *Dunnerh-Dunnerh* (Langloh Parker 1905: 39). The Wiradjuri from the Wellington Valley referred to the disease as *Danna-Danna* and to the sequelae as *Gulgog-Gulgog.* In the western districts of Victoria the epidemic was called *Meen Warann* (Dawson 1881:60), and among the Dhiari of northern South Australia it was called *Moora Moora*. Years after the event a song or chant, known as *Mallae Mallae* in Victoria and *Nguyapalti* in South Australia, was performed by many groups in remembrance of the epidemic and the many who died from it throughout south eastern Australia (Dawson 1881: 60; Reynolds 1981:57).

The assignment of blame for the epidemic, by the Aboriginal people was attributed in some instances to the Europeans, in others to the malevolent magic of neighbouring tribes, and to mythical entities. Like the other major diseases of syphilis, gonorrhoea, influenza, and tuberculosis, that had severe effects on the Aboriginal people, they often associated the occurrence of smallpox as a direct result of the arrival of Europeans among them. Sturt was blamed by the Wiradjuri of the Wellington Valley (Mair 1831). He and his expedition passed through that region in 1828 just as the smallpox epidemic was advancing along the river systems. Others in the same region

placed the blame for the epidemic on *Darrawirgal*, a mythical entity who lived far to the west of Wellington. They believed that *Darrawirgal* was angry for the want of a tomahawk and so sent the disease among them. When the epidemic subsided they assumed that he had finally obtained one and the disease would therefore not return (Wilkes 1845: 197 *loc. cit.* Campbell 1985: 341). The malignant power of the Rainbow Serpent *Mindye*, was believed to be the source of the disease in Victoria (Reynolds 1981: 57). Ahead of the colonial frontiers, where Aboriginal groups had yet to have direct contact with Europeans, the blame was apportioned to neighbours. Among the Muruwari, traditional enemies were claimed to have sent the disease on the winds which hung it on trees over camps where it dropped on to its victims (Langloh Parker 1905: 39). Clulwuwyrie, the last Ngarrindjeri survivor from the lower Murray, often spoke of a peculiar wind-like noise immediately preceding the arrival of smallpox which came among her people from the people to the east (Stirling1911).

3.4.3 The third epidemic - 1866-67

Smallpox made its appearance again in epidemic form in late 1866. It was observed during a six month period among Aboriginal people living on the arid west coast of South Australia on the periphery of European settlement and spread into the inland regions (Figure 3.1).

The first reports of this third outbreak were made by police in the remote regions of Streaky Bay and Venus in late October and early November of 1866. They noted an epidemic disease among the Wirangu and Nawu which was causing damaging skin eruptions on many who regularly visited the settlements (Gething 1867).

When informed, the initial reaction of the medical authorities in Adelaide was that it was just another outbreak of the 'native pock' and therefore of little concern to the colonists. (Native pock was a contemporary euphemism given to impetigo contagiosa, a staphylococcal disease of the skin common amongst Aboriginal people, and while sometimes confused with smallpox, has no relationship with the latter). The Vaccination Board of South Australia was, however, more cautious. Its primary concern was to protect the colony from any outbreak of smallpox that may occur by a program of vaccination aimed at rendering the whole population immune. Any outbreak of smallpox in the colony whether among the European or Aboriginal communities, was therefore to be given immediate attention. The Board instructed a government medical officer, Dr. Robert Gething, to leave at the earliest opportunity to investigate the outbreak and carry out a vaccination program among the European and Aboriginal populations (Gething 1867).

Gething spent 21 days travelling the coastal regions between the two bays meeting Aboriginal groups and vaccinating as many as he could. On January 12 1867, approximately 30 miles (48km) from Streaky Bay, Gething met a group of Wirangu suffering from the disease:

> which had all the characteristics of having been pure variola; but which in these cases seemed to have been singularly mild, as there could be only three or four deaths distinctly traced to that cause

Although his report to the Vaccination Board did not describe clinical signs, Gething was in no doubt that the disease he saw in these people was smallpox and not 'native pock'. His initial diagnosis of smallpox was soon strengthened:

> The next day the natives I had seen the previous day came down to me at the Bay, bringing with them others who had had the disease somewhat more recently, thereby enabling me to see it in another phase. This strengthened

> my opinion as to the nature of the disease, which was confirmed on a after occasion [*sic*] (Gething 1867).

Gething vaccinated as many Wirangu as he could and then proceeded south to Venus Bay. There he met a group of Nawu suffering from the disease, one of whom was a young female in the stages of secondary fever 'suffering unmistakably from pure variola'. This particular case was probably exhibiting the earliest stages of smallpox signs that Gething had so far encountered.

The extent of the smallpox epidemic may have extended further than the bay regions visited by Gething. To the west of Streaky Bay, and at the limit of European occupation, a hut-keeper at Fowlers Bay, wrote to the *South Australian Register* informing them that the Wirangu there were 'very much diseased' and that many of them had died (*South Australian Register* 1867). He queried why Gething, who had attended at Streaky Bay, had not made his way further west.

More observations of the extent of the epidemic came from the Sub-Protector of Aborigines responsible for a large portion of central and northern South Australia. Sub-Protector Buttfield, a former Methodist minister, had only recently taken up his position and had travelled extensively, meeting many of the Aboriginal groups living in regions remote from European settlements. On the south east coast of Eyre Peninsula he reported that during the first six months of 1867 the Nawu in the region of Port Lincoln had been suffering from the same disease as that previously notified by Gething near Streaky Bay (Buttfield 1867a). In the Mount Remarkable region, near the present town of Port Augusta, Buttfield (1867a) reported to the Acting Protector that Aboriginal people (Nukunu) were suffering from an eruptive skin disease 'resembling in some of its characteristics, smallpox' (*ibid*). Buttfield reported no deaths from this disease and those infected were convalescent. Buttfield was not a medical practitioner and was not entirely

sure that the disease was indeed smallpox; he thought it may have been a skin disease caught from dogs 'covered with loathsome sores and vermin'. The timing and locality of Buttfield's observation, while not strongly supporting a presence of smallpox, do give some indication to the probable extent of the epidemic.

This epidemic was almost certainly associated with simultaneous outbreaks of smallpox among Aboriginal populations in the north and west of Australia (Fenner 1984: 733). Well established trading and ceremonial lines existed among the Aboriginal populations of central Australia prior to European contact linking the far north of the continent to the southern coast and extending into the west (McBryde 1987: 253-273). The movement of Aboriginal people along these routes may have facilitated the slow spread of smallpox (Fenner 1984: 733). Throughout central Australia and on the north coast periodic outbreaks of smallpox infected Aboriginal populations between 1860 and 1869 (Cleland 1914: 170). In 1984, Goodall (1994: 75, note 13), an anthropologist working among the Pitjantjatjara and Yanykunytjatjara in the central desert regions, noted that a number of people could recall their parents or grandparents talking about an illness of plague-like proportions which may have been smallpox. Goodall states, however, that it was extremely difficult to follow up these indirect statements.

The origin of these outbreaks have been loosely attributed to visiting Macassan fishing fleets that frequented the northern coastline during the monsoonal wet season, collecting trepang (sea-cucumber) (Fenner 1984:732; Cumpston 1914; Lewis 1989). Other sightings of active smallpox and facial scarring have been reported on the central west coast and in the central arid regions north and south of the present town of Alice Springs between 1865 and 1870 (Fenner 1984:732). During the same period Aboriginal people on the west coast of Australia between Geraldton and the Gascoyne

River were severely affected by smallpox along with several European settlers (Cumpston 1914; Lewis 1989).

The degree of morbidity and mortality from the epidemic is impossible to estimate. Gething's report suggests, however, that morbidity was high among the Nawu and Wirangu along the south west coast of South Australia. After the disease first came to the attention of the police at Streaky Bay in October and November 1867 it was estimated that at least 30 to 40 Aboriginal people could be seen at any one time showing active smallpox. By January of the next year, when Gething was investigating the disease, between 200 and 300 had been infected at Streaky Bay and between 60 and 70 at Venus Bay - a range of between 260 and 370 (Gething 1867). The number of Nawu and Wirangu living in the area at this time cannot be enumerated with any accuracy and so no estimates of relative population loss from the epidemic can be made. Given the previous history of smallpox epidemics and the resultant mortality, however, it would be reasonable to assume that the case-fatality was high.

A young male and female living on a property between the two bays were the only Europeans recorded with smallpox during this epidemic. The effect of the disease was mild and both survived, having been previously vaccinated. In each case the disease left only a few scars upon the neck and face of each (Gething 1867).

The mortality among the populations along the coast and those inland beyond the frontier of European occupation remains largely obscure. Gething had little idea of the mortality caused by the disease and could account for only six to eight deaths. For an epidemic of smallpox among a population who had not been vaccinated regularly, if at all, such a figure would appear to be a gross underestimation. In all likelihood it was. Gething had spent only twenty one days on the west coast and most of that in a restricted

region between Streaky Bay and Venus Bay. He had not gone beyond the borders of European occupation. Unless he had travelled further he would have had no knowledge of the extent and severity of the disease in the interior, nor would any of the European settlers he used as informants. Further, he had observed the epidemic only in its earliest stages, during January. According to Sub-Protector Buttfield the epidemic lasted for a further five months among the Nawu in the Port Lincoln region alone; and it was his opinion that the disease had 'threatened destruction to the native population' in that region (Scott 1867: 665). The mortality would almost certainly have been more than that reported by Gething.

3.5 Discussion

Despite the varied nature of the surviving historical documents there is strong evidence that the three epidemics were indeed smallpox. This conclusion has been challenged by both contemporary and modern writers particularly for the 1789 epidemic. The little evidence we have pertaining to the 1789 epidemic in the Sydney region has left some historians and medical writers (Crosby 1986; Cumpston 1914; Curson 1985; Hingston 1985: 278) with doubts as to whether it was smallpox. Chicken pox (varicella) has been proposed as the main alternative to smallpox (Hingston 1985:278), with others suggesting that it was cowpox, a form of 'native pox', or some other fatal disease, not specified.

All these alternatives can be eliminated from serious contention on several lines of evidence which lead to smallpox. Firstly the First Fleet recorders of 1789 who reported on the disease are unanimous in their opinions that it was smallpox. Some saw it first-hand, others did not. Those who did not were in all probability basing their comments on the general opinion of the settlement that smallpox was the disease

infecting the Aboriginal people. Most, if not all, the recorders would have had experiences with the disease, known of its effects and would have been familiar with its signs, as it was the most widespread and fatal disease throughout the British Isles in the eighteenth century (Howe 1972: 143).

Secondly, although none of the journals or diaries from the First Fleet surgeons survived (Gandevia & Cobley 1974: 111), we can surmise with confidence that they were involved with the epidemic and their opinions were known (Frost 1994: 191). This is indicated by accounts that describe Eora people being brought in to the settlement and being treated for the disease in the hospital by the medical staff. Indeed, one of the child survivors was later accepted into the household of Chief Surgeon White. There appears to be little doubt that the medical staff had other experiences with those suffering from the epidemic. When it was first brought to the attention of Governor Phillip in April 1789 that Aboriginal people were dying among the coves of Port Jackson he immediately left the settlement with a small party including a surgeon to investigate (Tench 1793: 146). They saw first-hand the effects of the disease in the living and the dead. We can then be equally confident, that when Phillip (1790a, 1790b) reported back to London that the disease affecting the Aboriginal people was smallpox, he had been advised so by his medical staff.

A further line of evidence that strongly links the epidemic to smallpox are the sightings of Aboriginal people bearing visible smallpox-like sequelae, both facial scarring and blindness, after the epidemic had passed. The epidermal damage done by smallpox infection can be severe and residual scarring is common; up to 85% in unvaccinated variola major cases (Fenner 1985: 278; Jezek *et al.* 1981: 801). In chickenpox the epidermal damage is more superficial and in most cases quickly repaired after infection and facial scarring is rare. In a study of 250 Somalia subjects who had recovered from

chickenpox Jezek *et al.* (1981) found 2.4% of cases who had five or more facial scars indistinguishable from those seen among variola victims. This figure, however, may be an overestimation as the recorders tended to include subjects with a severe varicella rash rather than those with a mild rash (*ibid*: 802).

The reporting of residual scarring attributed to smallpox among Aboriginal people was widespread after each the three epidemics. After the 1789 epidemic, sightings were seen to the north and inland of the Sydney settlement, and in the coastal regions to the south at Jervis Bay and Port Phillip Bay. The recorded sightings after the second epidemic are more profuse. Explorers travelling beyond the frontiers of European settlement and colonists establishing new grazing lands commented on seeing Aboriginal people bearing facial scarring which they attributed to a previous epidemic of smallpox. The sightings were spread over a large area of southeast Australia following the major river systems and their tributaries. With a low rate of residual scarring chickenpox would be an unlikely candidate for the epidemics. The observations by Mair (1831), a qualified medical practitioner (attached to the 39th Regiment), are more reliable. Mair was in no doubt that the scarring he saw on Aboriginal people 'of very advanced age' corresponded 'with the pits left by the Small Pox' and that it was a result of the 1789 epidemic.

The evidence for smallpox is more cogent for the second and third epidemics. Both outbreaks were investigated by qualified medical practitioners who would have been familiar with the disease if not from practical experience then most likely from their medical training. From the clinical descriptions and the behaviour of the 1830s disease provided by Mair (1831) there can be little doubt that it was an epidemic of smallpox and not varicella (despite the opposition of the Inspector of Colonial Hospitals, Dr. James Bowman), or another disease displaying similar symptoms. After his investigation Mair concluded that the 'eruptive febrile disease' that had so affected the Aboriginal people

'approached more nearly in its symptoms to the character of Small Pox, than any other disease'. The descriptions of the symptoms by those who observed the active disease, and later compiled by Mair, closely match the progress and clinical features of ordinary type confluent and semi confluent, variola major as outlined by Fenner *et al.* (1988: 4-22) and Benenson (1976: 443-446). The symptoms suffered by the European male, Titman, on the other hand are more indicative of a secondary infection of variola major in a person who had been either previously vaccinated or who had been infected with the variola minor. It would appear by his own account of a previous childhood infection and Mair's observation of old and recent sequelae resembling pockmarks, that the latter was the case.

Gething (1867), who saw active smallpox among South Australian Aboriginal people in 1866-67 leaves us with no clinical description of the disease. Nevertheless, he was in no doubt himself that what he observed was an outbreak of smallpox, and not chickenpox or some another non-specified exanthematous disease. According to his report he saw on three separate occasions Aboriginal people suffering with what he described as the unmistakable symptoms of the disease in its various clinical stages. The residual sequelae from this outbreak were still noticeable on surviving Aboriginal people some 15 years later. The Royal Society of South Australia was informed at its meeting in June 1882 that the outbreak had passed through the Wirangu living near Streaky Bay and Fowlers Bay leaving 'a great number of them dying', and that a few of the survivors were 'very much pitted, more especially an old lubra who was blind (*Adelaide Observer* 1882)'. Sightings of Aboriginal people pockmarked by this epidemic were common throughout the centre of the continent when the overland telegraph line was being constructed and further north on the coast during the settlement of Port Essington and Darwin (Fenner 1984: 732).

The three epidemics, although differing in their geographic and temporal aspects, had common characteristics each compatible with the epidemic behaviour of smallpox in a virgin-soil population (Benenson 1976: 435-440; Fenner 1984:729-733):

1. They were highly transmissible. Accounts of all three epidemics report that the disease was spreading rapidly from person to person. This was later confirmed when observers noted that exposure to a sufferer could result in infection (e.g. Arabanoo caught the same disease as those whom he attended and a European family suffered the same symptoms as an Aboriginal child after they cared for her within their home).

2. They were almost entirely confined to the Aboriginal communities. In all three epidemics the disease was first noted to be spreading among the Aboriginal communities either on the periphery of the European settlements or ahead of the frontier. Despite the spread of European settlements and their ever increasing population, particularly after the first epidemic, very few Europeans (or non-Aborigines) were infected during the epidemics. The reason can be seen in the separation of the two groups. From the beginning of the first settlement in 1788 the social and cultural restraints particular to the two groups largely ensured that European and Aboriginal people lived in separate domiciles with physical proximity restricted to a few individuals. Infection by smallpox is usually a result of close contact such as would be expected of individuals living in the same household who had regular contact with each other. The disease is rarely spread by fomites such as contaminated clothing or bedding; or by limited exposure to airborne agents (Fenner 1984: 729). Only in exceptional cases then did the epidemiological conditions enabling the spread of smallpox to move freely between the two societies exist. In most of the recorded cases of non-Aboriginal people acquiring the disease there had been close and sustained contact with infected Aboriginal people.

3. The extent of each of the epidemics followed a course through regions where Aboriginal population density was high and European settlement low or where there were well established exchange and ceremonial networks (Figure 3.1). The evidence surviving for the first epidemic indicates that it was largely confined to the coastal and hinterland regions on the eastern side of the continent and did not extend far inland. The second epidemic followed the courses of the inland river systems forming the vast catchment area (1,036,000 Km^2) of eastern Australia that terminates at the mouth of the Murray River on the southern coast of South Australia. It appears to have bypassed the region of Victoria below the Murray and most of the coastal populations. The regions affected by the epidemics were resource rich coastal and riverine environments and had carried high population densities ranging from 2.5 to 4.6 persons per km^2 (Kirk 1983: 40; Webb 1984a). The geographical extent of the third epidemic has been documented over a large extent of Australia from the northern and western coasts through the arid centre to the southern coast (Fenner 1984: 731). In Southeast Australia, however, it was confined to the coastal and central inland regions where, during the 1860s, the major remnant populations of Aboriginal people lived. In an 1861 census in South Australia, the Western, Northern, and North Eastern Pastoral Districts accounted for 2,761 Aboriginal people or 58.9% of the total population enumerated for the state. It was these groups who bore the major impact of the epidemic and the smaller population groups living on the fringes of the European settlements and along the River Murray who escaped.

4. The three epidemics caused severe mortality. A recurring observation by all recorders is the stark reference to high mortalities among the Aboriginal populations inflicted with smallpox. Just how high then was the mortality? The immediate answer, using the historical descriptions of each of the epidemics, is that the mortality was severe - but just how severe?

In his model of Aboriginal population decline, Butlin (1983: 65) suggested an overall death rate due to smallpox from 25% to considerably in excess of 50% of the total populations affected by the epidemics. Butlin based his estimates on the American Indian populations on the west coast of the United States during the 1837-39 epidemic (Stearn & Stearn 1945). Conservative estimates of 30% mortality or more in initial smallpox outbreaks have also been suggested for the Americas (Boyd 1990: 137; Crosby 1972: 44; Dixon 1962: 325). This estimate has often lead to the generalised acceptance of a minimal figure of one-third of the population dying from initial exposure to smallpox (Boyd 1990: 325). Can such rates be inferred for the three epidemics in Australia? The answer is yes, but with some caution.

Throughout its historic association with humans smallpox has had a high case-fatality, particularly among those who had not been protected from the disease either by previous infection or by inoculation. Case-fatality rates from smallpox among unprotected populations vary according to the clinical type, but in most cases are severe: ordinary-discrete type, <10%; ordinary semi confluent, 25-50%; ordinary-confluent, 50-75%; flat, >90%; and haemorrhagic type, almost 100% (Fenner *et al.* 1988: 5; Koplan & Foster 1979: 440). The young, the elderly, and pregnant women are the most likely to die from smallpox. The lethality of smallpox became graphically apparent when the disease was introduced from Europe into the New World at the end of 1518 or early 1519 (Crosby 1986: 196). The effect of variola major among the unprotected and virgin-soil populations in the Americas resulted in extraordinarily high mortality with estimates ranging from 25% through to 90% or more (Black 1975: 517; Boyd 1990: 144; Dobyns 1983; Fenner 1984: 730-733; Fenner *et al*. 1988: 1069-1102; Ramenofsky 1987; Snow & Lanphear 1988). By the end of the seventeenth century, nearly two hundred years after its introduction, smallpox was largely responsible for reducing the population of Central Mexico to an estimated 3% of its 1520 level (Snow & Lanphear 1988: 16). In just two

decades after the introduction of smallpox in the north east of United States the indigenous populations had been severely reduced by a factor estimated to be between 67 and 95%.

The clinical descriptions we have for the first and third smallpox epidemics do not allow us to discriminate precisely which types of variola major were present, although the discrete type would be favoured because of the high mortalities indicated by the reporters and diarists. Phillip estimated that the 1789 smallpox epidemic killed 50% of the Aboriginal people living around the Sydney settlement. The descriptions of smallpox by Mair (1831) during the second epidemic, and later by Mitchell describing the smallpox sequelae of the Barkindji on the Darling River, however, indicate symptoms which accord with confluent and semi-confluent clinical types. Mortality was estimated by Mair to vary from one in three (33%), to one in five or six (17-20%) of infected cases. He thought, however, that it could have been less if the victims had been able to have access to medical care and shelter. A case fatality-rate of 25% (the lower limit proposed by Butlin (1983: 65) is then plausible.

Although the general tone of the majority of reporting on the epidemics strongly implies high mortality, caution is required in accepting any mortality estimate categorically for all populations in the path of an epidemic. In the cases of all three smallpox epidemics there are several unknown factors relating to the prevalence of the disease and the mortality. In all three epidemics we would expect regional variation in the number of people infected by the disease. Some groups would have had a high proportion of their numbers infected and a resulting high mortality while other nearby groups managed to avoid contact with carriers and so escaped the epidemic. Within the infected groups there would have been a degree of differential resistance to infection. A number of individuals would have had an active immunity to the disease in which the

body reacts specifically to the pathogen and is able to survive the course of the symptoms. This would depend chiefly on the amount of exposure to the virus, the current health of the individual and the ability of the immune system to combat the spread of the virus. Also a genetically inherited nonspecific resistance to the virus by some groups cannot be discounted (Cockburn 1971: 51-52).

Finally, and as a word of caution, there is no way of determining the number of individuals who were infected by smallpox and the number who managed to escape infection. It is one thing to consider the various case-fatality rates of smallpox and another to equate those rates with mortality in one entire community or region. In all epidemics there are a number of individuals, families, groups, who manage to escape infection entirely. This can happen due to fortuitous circumstances such as living away from other groups who become infected during the epidemic, or active avoidance of the disease by gaining prior knowledge of its presence and fleeing its path. We simply do not know what this number may have been for any of the epidemics, nor in any of the regions they occurred. When using case-fatality rates to estimate population loss we must first know, or have a reasonable estimate of the number of cases of smallpox infection. For the three epidemics in Southeast Australia I can see no way of estimating a reasonably accurate number of infected cases. Therefore any estimation of Aboriginal population loss incurred by the three smallpox epidemics of Southeast Australia based on case-fatality must be treated with caution.

3.6 Conclusion

Three major epidemics of smallpox affected the Aboriginal population of Southeast Australia during the colonial period. Evidence for the first in 1789 suggests that it was confined mainly to the coastal regions of New South Wales and Victoria. The second epidemic occurred between 1828 and 1832 and was observed along the major

river systems. The third epidemic which was observed in the western coastal regions of South Australia was most likely a southern extension of a more extensive series of outbreaks that occurred in the north, west, and centre of the continent.

While smallpox had only a minor effect on the European population of Australia, the same cannot be said for the Aboriginal people The effects of the three epidemics in Southeast Australia caused excessive mortality and morbidity. The three epidemics hit hard at Aboriginal communities who were immunologically unprepared for the virus. By contrast, they had little effect on the European population. While no tangible estimate of population loss can be derived from the historical literature it is reasonable to expect that 30% or more population loss occurred in areas reached by each of the three epidemics.

CHAPTER FOUR

Sexually Transmitted Disease

4.1 Introduction

This chapter discusses the dissemination of syphilis, and to a lesser extent gonorrhoea, through the Aboriginal populations of Southeast Australia after colonial settlement. I will examine historical documents covering the region as well as skeletal evidence from the Murray River and will argue that these two diseases were repeatedly introduced into Aboriginal populations at the interface of contact with European colonists. I will show evidence for the introduction of syphilis initially from the founding settlements of each of the colonies of New South Wales, Victoria, and South Australia, then subsequent introductions following the course of colonial expansion. Finally I will discuss the evidence for syphilis along the Murray River in South Australia before the establishment of the colony.

In the historical accounts of treponematosis among Aboriginal populations in colonial Australia the problem arises as to which of the diseases in this group is being documented. Was it an endemic disease that existed before European contact which the colonist are confusing with the more familiar venereal form of syphilis, or were the colonists indeed describing just the introduced venereal form of the disease?

Previous authors have argued that two forms of treponemal disease existed in Australia prior to European colonisation; an endemic form of non-sexually transmitted syphilis (treponarid) and yaws. Hackett (1936a, 1936b, 1978) and Webb (1984a, 1984b, 1989, 1995) have reviewed oral, historical, and ethnohistorical evidence together with skeletal samples displaying typical treponemal lesions and claim that both forms existed

among Aboriginal populations of northern Australia and in some areas of the arid centre. The yaws spirochaete (*Treponema pertenue*) was endemic to the nearby tropical regions of New Guinea, and Oceania before European contact (Cahil 1975: 160; Cilento 1942; Kranendonk 1958). Its presence in nearby regions of Australia with similar climatic conditions is then no surprise (Garner *et al.* 1972).

In contrast, the evidence for any of the forms of treponematosis in pre-contact Southeast Australia is by no means certain (Dowling 1990; and see discussion below). The area is far removed from the equatorial and sub-equatorial regions of northern Australia that are suitable for the survival and non-venereal transmission of the spirochaete (Benenson 1990: 484). If there was indeed an endemic form of the disease in Southeast Australia it was most likely to have been of low prevalence and of little concern to the Aboriginal populations. The problem then of which form of syphilis, endemic or introduced, is being seen and documented by the early colonists is therefore much reduced.

A further problem arises in the historical literature regarding this disease through its confusion with other sexually transmitted diseases, particularly gonorrhoea (Spink 1978: 309). Historically, the spread of syphilis and gonorrhoea has been strongly linked with the increasing transcontinental travel and migration of human groups over the last five millennia (McNeill 1976: 202). Because of their similar modes of transmission these diseases have often accompanied each other during mass population movements, each in turn spreading in epidemic patterns among indigenous populations. In such cases the incidence of gonorrhoea has been up to ten times more than syphilis (Spink 1978: 312-315).

Although sexually transmitted diseases today are regarded as a diverse group of human infections with different aetiological and epidemiological features, this has not always been the medical understanding. Much confusion and controversy have surrounded the understanding of sexually transmitted diseases for several centuries, with medical scholarship being split regarding their aetiology; particularly gonorrhoea and syphilis. The influential British physicians Thomas Sydenham and John Hunter believed that gonorrhoea and syphilis were the same disease (Duin & Sutcliffe 1992). Hunter (cited by Duin & Sutcliffe 1992:100) argued that the difference between the two disease syndromes depended on the nature of the surface which was infected; it caused the characteristic ulceration or chancre on the cutaneous surfaces but only a purulent discharge when mucous membranes were infected. Hunter's influence on British medical scholarship was such that many of those trained in Britain held to his view of a single disease entity well into the nineteenth century. The frequent association of gonorrhoea with syphilis also added to the confusion between the two diseases and led many of the writers to regard the symptoms of gonorrhoea, the 'clap', as an early stage, and the symptoms of syphilis the more advanced stages, of a single disease. The controversy over the two diseases began to be cleared during the 1830s when the French venereologist Ricord distinguished the developmental stages of the diseases (Arrizabalaga 1993:1031-1032). During the second half of the nineteenth century Ricord's concepts were gradually being accepted by the medical scholarship of Europe, but it was not until 1879 when the gonococcus was isolated and identified as the pathogenic agent causing gonorrhoea, that the symptoms were seen as separate diseases. The identification of the treponeme causing syphilis then followed in 1905 (Arrizabalaga 1993:1031).

Although the two diseases were separately classified in the Victorian Nosological Index of 1863 (Morgan 1987), confusion surrounding the aetiology of syphilis and gonorrhoea persisted among Australian physicians well into the nineteenth

century (Farr 1854: 139). This was possibly because they were situated far from the mainstream of the development of medical knowledge in Europe. Further, those physicians dealing with Aboriginal people and reporting on their health were usually practising in rural regions further isolated from the medical centres of the cities. While the terms 'syphilis' and 'gonorrhoea' were used by some physicians and non-medical observers throughout much of the nineteenth century when referring to the symptoms they saw, others used the more general term of 'the venereal' revealing a persisting confusion between the diseases.

The result of this confusion is that it can be difficult (and sometimes impossible) when examining medical accounts from the eighteenth and nineteenth centuries to differentiate between the two diseases. In the source material used below I have used accounts where there appears little doubt that the observers are referring to a sexually transmitted disease, and in most cases it can be accepted as syphilis because of the more destructive and well recognized symptoms of this disease. But at the same time, the presence of gonorrhoea cannot be dismissed even though it is rarely referred to.

4.2 Biology of sexually transmitted diseases

4.2.1 Syphilis

Syphilis is a complex disease caused by the spirochaete *Treponema pallidum*. Other closely related spirochaetes which infect humans but through non-venereal means are *T. pertenue*, the causative agent of yaws, and *T. carateum* which causes pinta. Antigenic cross-reactivity between *T. pertenue* and *T. pallidum* is now well documented and the subtle molecular differences between these organisms are known (Baker-Zander & Lukehart 1981; Thornburg & Baseman 1983).

The development of syphilis has three stages. The primary stage presents as a characteristic lesion (chancre) at the site of entry of the treponeme. After four to six weeks even without effective treatment the chancre may spontaneously heal. A secondary disseminated macular-papular rash involving the skin particularly the palms and soles and mucous membranes follows two weeks to six months after the disappearance of the primary chancre. Lymph nodes, stomach, and liver may be affected. A latent period follows the secondary stage and can be succeeded by a more debilitating third and final stage (tertiary syphilis) in untreated victims (Benenson 1990: 420-421; Connor & Gibson 1988: 353-356).

The sequelae of syphilis if left untreated are multiple and unpredictable. Primary infections often heal spontaneously but can progress steadily to cause serious and debilitating symptoms which may lead to death. Syphilis can cause blindness, aortic impairment (cardiovascular syphilis), visceral disorders, loss of neuro-muscular control, and serious destruction of bone, skin and mucosal surfaces (Benenson 1990: 420-421; Connor & Gibson 1988: 353-356; Hart 1984:6; King & Nicol 1975: 13-127). A major debilitating effect of untreated syphilis is its involvement with the central nervous system. Neurosyphilis may occur during any stage of the disease and presents as acute syphilitic meningitis during the secondary or early latent phase, meningovasular syphilis later, and finally, fifteen to twenty years after initial infection, progressive degeneration of the dorsal roots and ganglia of the spinal cord (tabes dorsalis) characterised by impaired sense of joint position, ataxia, episodes of intense pain (tabetic crisis), impotence hyperflexia, secondary degenerative arthritis, and dementia (Benenson 1990: 421; Brown *et al.* 1970: 17; Connor & Gibson 1988: 353-356; King & Nicol 1975: 77-98). In addition chronic

untreated syphilis is often a predisposing factor in the appearance of other infections and illnesses.

Syphilis can be acquired *in utero.* Foetal infection occurs at a high frequency in pregnant women with untreated syphilis. It frequently results in abortion or stillbirth of the foetus and can be a factor in infant deaths due to premature delivery, low birth-weight, and generalized systemic infection and destruction of tissue and bones. Surviving infants may carry associated stigmata such as Hutchinson's teeth, sabre shins (boomerang shins) saddle nose, keratitis and deafness (Benenson 1990: 421; Connor & Gibson 1988: 356; King & Nicol 1975: 99-127).

4.2.2 Gonorrhoea

Gonorrhoea is caused by the gonococcal bacterium *Neisseria gonorrhoeae*. Infection is usually limited to the epithelial tissues of the urogenital tract, most commonly the urethra in males and the endocervix of females. In males symptoms present two to seven days after infection and are characterised by dysuria and a purulent discharge from the anterior urethra. The infection may be self-limiting or in some cases develop into a chronic carrier state. In such cases acute prostatitis, epididymitis, urethral stricture and sometimes male sterility can result (Benenson 1990: 185; Brown *et al.* 1970: 90; Connor & Gibson 1988: 391; Hart 1984: 3-4; King & Nicol 1975: 188-207). In females most infections are asymptomatic or mild enough to pass almost unnoticed. In cases of untreated gonorrhoea, pelvic inflammatory disease and chronic acute salpingitis may occur when the infection spreads from the cervical glands to the fallopian tubes (Cates *et al.* 1990). In such cases infertility occurs when inflammatory adhesions close the fallopian tubes blocking the descent of the ova and the ascent of the sperm.

While in most cases gonorrhoea is not life-threatening for either males or females, repeated infection and the lack of effective treatment may result in extra-urogenital involvement secondary infections, most commonly gonococcal septicaemia, arthritis, meningitis, and endocarditis. Neonatal infection can arise from infected amniotic fluid or an infected birth canal. Symptoms usually arise a few days after birth. The major form of neonatal infection is gonococcal conjunctivitis which may lead to permanent blindness. Other sites of gonococcal infection in neonates are the pharynx, respiratory tract, vagina, anus, joints, and bloodstream (Connor & Gibson 1988: 391; Brown *et al.* 1970: 90-91).

4.3 Syphilis and gonorrhoea among the European population of Southeast Australia.

Sexually transmitted diseases were common among the crews of ships exploring the Pacific in the eighteenth century. For example, Captain James Cook's crew together with other European voyagers left a trail of sexually transmitted diseases, particularly syphilis and gonorrhoea throughout the islands of the Pacific (Watt 1979: 148-151). Syphilis and/or gonorrhoea came with the First Fleet to Australia in 1788. Before sailing from Britain, Phillip had been concerned about the medical fitness of the future colony, realising that its success would largely depend on the health of all who sailed. He made a special request to the Home Office that convicts selected for transportation should be in a good state of health and free from sexually transmitted diseases (Watt 1989: 139). His request was not given due consideration. With the ever increasing number of convicts in the already overcrowded prisons the Home Office was under pressure to increase the numbers of convicts to be transported and their health was given little attention in the selective processes (Watt 1989: 139).

Sexually transmitted disease became an unwelcome passenger to Australia, not only among the convicts but among the military contingent as well. By the time the fleet had reached the Canary Islands, their first port of call after just three weeks at sea, ten cases of 'venereal' had been reported by the surgeons; eight of them were convicts, the other two were marines. Further cases were reported among both groups before the final destination of Botany Bay was reached (White 1787: 16).

Attempts to keep the sexes apart while at sea met with some success, but when landed their separation became impossible to ensure. Heterosexual liaisons began soon after the arrival in Port Jackson, particularly among the convicts (Tench 1793: 39). Within three months of the settlement's establishment the presence of sexually transmitted disease among the convicts began to concern Phillip and his medical staff. Not only was it becoming more prevalent but it was common for the sufferers to conceal their condition. In a vain effort to flush out those with the disease and prevent it spreading through the colony, Phillip ordered that any man or woman, found to be concealing the disease should receive corporal punishment and be put on a reduced allowance of provisions for six months (Collins 1798: 20). Marriage was also encouraged by both the medical and religious authorities as a further attempt at inhibiting the spread of the diseases (Wrogan 1978: 25). Neither method was successful in stopping the spread of sexually transmitted disease throughout the colony. In a five year period between November 1791 to September 1796 'Lues venerea' (another name for syphilis, see e.g. Arrizabalaga 1993: 1028; Farr 1854:165; Morgan 1987: 28) accounted for 5 of the recorded 137 deaths (3.6%) among the civilian, military, and convict contingents (Collins 1798: 426).

It continued to be a major cause of morbidity among the colonial population throughout the nineteenth century. Annual reports from the Sydney Dispensary and

Infirmary record that syphilis was the sixth most common disease in 1838-39, accounting for 4.8% of all diseases treated. By 1859 it was the third most common disease occurring at 7.0%, and by 1874 it had reached second at 6.5% behind the leading cause of morbidity, accidents and violence (Curson 1985: 19).

4.4 Syphilis and gonorrhoea among the Aboriginal populations - source material

The source material used below is examined in two contexts. Discussed first is the evidence of syphilis and gonorrhoea among Aboriginal people living in the vicinity of European settlements. Secondly I will examine historical and osteological evidence as to the spread of syphilis and gonorrhoea spread ahead of European frontiers.

4.4.1 At the frontiers

New South Wales

It remains unknown when sexually transmitted disease began to infect the Aboriginal people in the Sydney region after the arrival of the First Fleet in January 1788. Collins was the first to comment on its presence:

> The venereal disease also had got among them; but I fear our people have to answer for that; for though I believe none of our women had connection with them, yet there is no doubt but that several of the black women had not scrupled to connect themselves with white men (Collins 1798: 495-496).

Which disease Collins is referring to, and at what time after the arrival of the First Fleet he saw it, remains unclear. Collins served in New South Wales from January 1788 until August 1796 and published his account of the colony in 1798, so he is referring to the presence of one or more of the sexually transmitted diseases among Aboriginal people during that eight year period. He then goes on to say:

> It was by no means ascertained whether the lues venerea had been among them before they knew us, or whether our people had to answer for having introduced that devouring plague. Thus far is certain, however, that they gave it a name, Goo-bah-rong; a circumstance that seems rather to imply a pre-knowledge of its dreadful effects (*ibid*: 496).

By the term 'lues venerea' Collins, in this instance, most likely means venereal syphilis rather than gonorrhoea (Arrizabalaga 1993:1028; Farr 1854:165; Morgan 1987: 28).

How quickly the diseases spread throughout the Aboriginal population is unclear. It may not, however, have become a serious health problem among them until after at least the first six months of settlement at Port Jackson because of the infrequent contact between the two groups (Phillip 1788b). Governor Phillip's objectives towards the Aboriginal people were twofold (Blainey 1982c:25; Clark 1981: 116). On the one hand the function of the Port Jackson community was to establish a penal settlement in New South Wales. It was therefore necessary to control the movements of the convicts, who made up the bulk of the settlement's population, and to keep them from freely fraternizing with the Eora. Phillip had also been instructed to foster a friendly relationship with the Aboriginal people, to establish a system of trade with them, and to teach them the benefits of his own civilization. In order to do this he had to meet with them. The first contacts between the Eora and the colonists were, however, sporadic. When they did occur misunderstanding and mistrust between the two often resulted in the angry use of weapons. The Aboriginal response was to avoid the settlement and its occupants (Blainey 1982c: 25; Clark 1981: 116; Day 1996: 60-61; Phillip 1788b).

Keeping the convicts from meeting with Aboriginal people outside the confines of the settlement was however, a problem for the colonial authorities. Collins (1798: 29) records:

> In one of the adjoining coves resided a family of them, who were visited by large parties of the convicts of both sexes on those days in which they were not wanted for labour, where they danced and sung with apparent good humour, and received such presents as they could afford to make them; but none of them would venture back with the visitors

By the 1820s sexually transmitted disease was perceived by Europeans as a serious problem among the Aboriginal people in the Sydney region. Cunningham, a naval surgeon, who made five trips to New South Wales aboard convict transport ships between 1819 and 1828 commented on disease among the Aboriginal people. He made a distinction between gonorrhoea and syphilis although attributing one disease to females and the other to males:

> From their natural filthiness, the women soon become diseased with gonorrhoea, and propagate this infectious malady among the convict-servants who cohabit with them. I have often observed the men too, labouring under eruptions of the skin resembling syphilis, and open tumours [*sic*] also in their groins apparently of the same nature; but time with them cures all disorders (Cunningham 1827).

In all likelihood gonorrhoea and syphilis were affecting both sexes.

Cunningham's view that time cures all disorders, while reflecting the state of medical knowledge of the time, was correct in one point but incorrect in another. We have already seen that gonorrhoea in males can be self limiting, and if no further infection is acquired the individual may be entirely cured. On the other hand mild clinical symptoms may appear in females for a short period but lead to the more serious pelvic inflammatory disease. Cunningham also appears to be referring to the primary chancres of syphilis which can spontaneously heal within four to six weeks of infection even without effective treatment. The second and third stages of development of this disease often lead to more serious life-threatening complications if effective treatment is not provided.

Victoria (Port Phillip)

Sexually transmitted diseases among Aboriginal people soon began to be noticed by colonists after the settlement of Melbourne was established on the southern coast of Southeast Australia in 1835.

Within four years of the establishment of this settlement, syphilis was recorded by several observers to be widespread among Aboriginal people. During the late spring and winter months of 1839 an increasing group had been living on the banks of the Yarra River, on the fringe of the new European settlement. Many of these people were in a poor state of health and were suffering from a variety of symptoms including dysentery and fevers (Cussen 1839a). After six had died Dr. Cussen was instructed by Chief Protector of Aborigines, G.A. Robinson, to visit the group and offer what assistance he could. He reported back to the Chief Protector that most of those who were ill were suffering from syphilis (Cussen 1839b).

Syphilis had been present among the Aboriginal people of the colony (principally the Woiworung, Boonwurrung, Wathaurong, and Kurnai) before this incident according to Cussen - probably as early as 1837, two years after the settlement. In the same report he noted:

> From an experience of more than a year and a half of the tribes which frequent this neighbourhood, I can assert that syphilis is (generally speaking) committing the most extensive ravages amongst them, and shall most shortly, if unchecked, render them extinct in a very few years. I have had several of them from time to time under my care and they appear to receive with avidity and gratitude any measures that are adopted for their advantage (*ibid* 1839a)

In May 1839 Assistant Protector Sievewright wrote to the Chief Protector reporting that 'nine-tenths' of the Wathaurong under his administration around the present city of Geelong on the western side of Port Phillip Bay were victims of syphilis. The symptoms, he claimed, appeared in a more 'violent' form than even the most 'extraordinary' European cases (Sievewright 1839). Sievewright went on to report that neither sex was exempt from the disease and those infected ranged from infants to the aged.

His observations were later supported by Fyans who had moved to the region as the police magistrate in 1837. He stated that 'large families of natives - husband, wife, boys, and girls - were eaten up with venereal disease' and estimated that 'two thirds of the natives' of the colony had died from this infection (Fyans (c1853) in Bride 1898: 181).

The disease in this district could have two sources. It may have been carried along from the Melbourne settlement 60 km east with the movement of colonists and Aboriginal people between the two settlements. Another potential source may have been from the sealing and whaling ships that had been frequenting the southern waters of Southeast Australia since the early 1800s (Critchett 1990: 133). If the latter, then syphilis and possibly gonorrhoea may have been present among the Aboriginal groups in this area for thirty years before the settlement of Victoria.

In 1840 syphilis was reported by Assistant Protector Parker among the Wadi Wadi, Wemba Wemba, and Wergaia in the northern district of the Port Phillip colony. From early December of that year an increasing group of up to 170 had been gathering at the Loddon River Aboriginal Station. Parker (1840a) reported that he had found '...five cases of syphilis of a very severe character.' The rest of the people appeared to be free from syphilis. Parker was concerned about the frequent sexual fraternization between

Aboriginal women and European men. He saw the Aboriginal people '...sinking to a lower degree of moral degradation by the pernicious intercourse which they have with the vitiated portion of the lower classes in the colony' (Parker 1840b). He was concerned enough to write to Chief Protector Robinson and request that it would be 'highly desirable' for a medical officer to visit the station as soon as possible to instruct him and his overseer on ways to administer to the disease. Medical help was not immediately forthcoming and his predictions about the increasing problem of syphilis were realised ten months later. In an urgent disposition he wrote again to Robinson:

> The call for medical assistance is however rendered especially urgent at the present time by the rapid and extensive spread of syphilitic affections among the aborigines... At the present time I fear more than half the women are infected, and the disease is rapidly spreading (Parker 1841).

Similar reports were made by a third Assistant Protector of Aborigines, James Dredge, on the Goulburn River among the Taungurong. Dredge reported to the Chief Protector:

> ...it is extremely difficult to convey a correct report of the awful amount of disease amongst both sexes of every age, resulting from illicit and promiscuous intercourse with the Whites, and with each other. This disease amongst the natives originating, in every instance - as my observation goes - in their intercourse with white men. For the cure of this loathsome and deadly disorder, no medicines adapted for their use have been placed at my disposal (Dredge 1840).

While neither the Assistant Protectors nor the Police Magistrate were medically qualified, their reports of what would appear to have been an epidemic of syphilis can be taken with some confidence. All three Assistant Protectors had close contact with the Aboriginal people in their regions and were required to investigate and

report any disease among them. Moreover, there is a strong similarity between all their reports and with the observations of Dr. Cussen who was convinced of a highly virulent epidemic of syphilis present among the Aboriginal people of the colony.

The reports of Dr. Cussen and the three Assistant Protectors reveal several features of the disease they observed:

- The disease was almost certainly syphilis. All but Dredge reported that the symptoms they saw were due to syphilis and referred to it by that name. Dredge, while not specifically naming the disease as syphilis, states that he is certain that it was spread by sexual intercourse, particularly when Aboriginal females and European males were involved.

- By 1840 it was widespread throughout the colony. Cussen reports it among Aboriginal people at the main settlement of Melbourne, many of whom had come from areas further inland to camp on the fringe. The Assistant Protectors observed the symptoms in the vicinity of the European pastoral settlements in the southeast and the north of the colony.

- All report that there was a rapid dissemination of the disease among the Aboriginal groups.

- Morbidity and mortality were excessive. Both sexes and a wide spread of age groups were observed to be affected. While the claims of ninety percent morbidity and two-thirds of the Aboriginal population dying

from the epidemic may be exaggerations on the part of Sievewright and Fyans, they lend support to Cussen's observations that syphilis was at a high level among the Aboriginal people of the colony at the time. What the actual mortality from the epidemic was is of course unknown, but the general tone of the reports demonstrates that it was severe.

Further evidence of the extent, persistence, and severity of the epidemic is available from the papers of Chief Protector Robinson. Between April and October 1844 Robinson travelled throughout the eastern interior of the colony. His purpose was to examine at first hand the state and welfare of the Aboriginal people living near European settlements and those in the regions not settled by Europeans. Table 4.1 lists Robinson's journal comments on syphilis among the Aboriginal populations he encountered.

Table 4.1 Comments regarding the observation of syphilis among Aboriginal populations made by Chief Protector Robinson during his journey through the eastern interior in 1844 (Robinson 1844; Mackaness 1941).

Area	Comment
Lake Omeo	A loathsome disease (Syphilis) among the Natives imparted by Europeans is making ravages.
Southern Monaro/Upper Snowy River	Syphilitic and other European Disease among the Natives is prevalent, and their numbers are rapidly decreasing.
Twofold Bay	Two men died from syphilis
Gundagai	...numbers are suffering from syphilis among whom are several bad cases.
Albury	A large number of Natives had congregated at the Hume [Murray River] ... they were greatly suffering from Syphilitic disease.

On his return Robinson reported that syphilis was 'almost general throughout the land' and had 'extended [its] baneful influences to the remotest parts of the interior' (Robinson 1844).

In the same year concerned settlers on the Murray River in the northern region of the colony wrote to Robinson calling his attention to the state of the Aboriginal people in their region:

> At the present moment there are few stations in the District at which there are not from one to three Aborigines suffering under the various stages of Venereal; and besides the mortality it causes amongst the natives themselves it is also the means of propagating the disease among the labouring men of the District to a very great extent. There is, and we fear will be, until religion gradually works a change, a great deal of promiscuous intercourse between the Europeans and the Aboriginal native, the consequence of which is the rapid increase of the disease in the District (Huon *et al.* 1844).

In 1848 while Robinson was visiting the Aboriginal Protectorate settlements of the colony he reported that along the main road between Sydney and Melbourne there were 'as usual a great number suffering from Syphilis' (Robinson 1848).

By 1860 reports on the health of Aboriginal people living in the Murray River corridor began to indicate that the cases of sexually transmitted disease had lessened. In the first report the Central Board for the Protection of the Aborigines, Thomas Goodwin reported from the Yelta settlement near the junction of the Murray and Darling Rivers in the north west of the colony that:

> Venereal is not so frequent amongst the men as is generally supposed, I have seen very few cases, but I believe many of the young women, and even girls are afflicted with it (Goodwin 1861-62).

In 1864 and again in 1866 the reports of the Board indicate an improvement in the general health of the Aboriginal people along the Murray and a decrease in the numbers of sexually transmitted disease being observed (B.P.A.1864-65, 1869). Dr Campbell (1867) who had been employed as a medical practitioner among the Aboriginal

groups on the Lodden River reported in 1867 that he had not seen sexually transmitted disease among those residing or visiting the station during the past four years.

Nineteenth century medical descriptions of sexually transmitted disease among Aboriginal people are not well represented in the historical records. Probably the earliest surviving description of a sexually transmitted disease comes from Dr. W.B. Wilmot, medical officer and coroner, at Port Phillip. He attended the sick at Nerra Nerra Warren Aboriginal Station, forty kilometres south east of Melbourne. His diagnosis of a 'syphilitic disease', which he saw among Aboriginal people was based on his own observations of the clinical signs of the disease in what he took to be its various stages of development, from a few weeks through to six months after the suspected time of initial infection:

> It generally originates with a small cluster of irritable papulae on the inside of the thigh or scrotum and perineum, which rapidly coalesce, and degenerate into foul ulcers, terminating ultimately in a warty excrescence; in a few weeks it spreads very widely, and the skin becomes at length affected with a scaly eruption of a circular form, with a well-defined margin, giving it much the same appearance as *lepra vulgare*.
>
> The action of the virus [*sic*] is singularly modified by the habits and constitution of the native as it does not appear to pass through the ordinary channel of the lymphatic system into the constitution, but directly through the general circulation or capillary vessels as only in one instance and of a dubious kind did I meet with any appearance of specific glandular affection.
>
> In no case did I find any other than the inguinal glands affected; in no case was there any ulceration of the fauces [mouth and throat], nor did I meet with a single instance of a venereal node (Wilmot 1842: 257).

The signs Wilmot describes are not compatible with the clinical signs of syphilis or gonorrhoea. Primary syphilis typically presents as a single painless ulcer or chancre at the site of inoculation usually on the shaft of the penis, vulva, labia or cervix. The secondary stage of syphilis is characterised by a spreading maculopapular rash in a variety of organs, most commonly the skin of the palms and soles, mucosa, lymph nodes,

stomach and liver. The rash is apparent in the moist genital surfaces as well as the mouth, although in Wilmot's description the latter was not the case. Other secondary symptoms include the broad flat plaques of condylomata lata located in warm moist areas, and lesions around hair follicules, neither of which can be reconciled to Wilmot's description. Gonorrhoea infections are commonly localised to the urogenital tract in both sexes with males displaying the characteristic purulent discharge from the anterior urethra. No cutaneous lesions are associated with the infection (Beneson 1990: 421; Connor & Gibson 1988: 354-355).

The small clustering of 'irritable papulae on the inside of the thigh' is more suggestive of genital herpes. Herpes simplex virus type 2 produces a spectrum of vesicular and necrotizing lesions localised to the genitalia and immediate surrounding regions. The principal sites of primary infection are the glans penis, cervix, and vulva with recurrent disease involving the perineal skin, legs and buttocks. The degeneration of the primary rash into 'foul ulcers' described by Wilmot may represent localised secondary bacterial infection particularly if the Aboriginal people were living under unhygienic conditions as was possibly the case. The 'warty excrescence' may represent genital warts (*Condylomata acuminatum*). They commence as small swellings in the peri-genital and peri-anal regions and rapidly grow into grouped pedunculated lesions. A number of lesions may fuse into a cauliflower-like mass (King & Nicol 1975).

A final possibility is that the pustular lesions may be due to chancroid infection caused by *Haemophilus ducreyi*. This bacillus is highly infectious and invades on contact through the skin or mucosa of the genitalia. A papule develops up to fourteen days after inoculation, becomes pustular and ulcerates. The ulcers are usually small but large mutilating ulcers can occur. Multiple ulcers are common. The lesions may spread locally to the perineum, anus, scrotum, thighs or lower abdomen (Benenson 1990: 81-82).

From examining Wilmot's description it is not possible to come to any firm conclusion in identifying the disease, or indeed diseases, which he saw. I think, however, that two of the most common sexually transmitted diseases of that time, syphilis and gonorrhoea can be ruled out as contenders in this case due to the incompatibility of the symptoms. This then leaves the other possibilities, herpes simplex, genital warts, or chancroid. The latter is unlikely because the disease is mostly confined to the tropical and subtropical regions of the world (Benenson 1990:81); although introduction by infected sailors recently arrived via a tropical port is a possibility. An outbreak of herpes simplex possibly accompanied by cases of genital warts seems the most likely .

South Australia

The initial introduction and dissemination of sexually transmitted disease among the Aboriginal groups of South Australia differs from the two other colonies discussed above. As I will discuss below a disease resembling syphilis was present among the Meru and Ngaridjeri living along the Murray River and southern coast before the arrival of permanent European settlers and the establishment of Adelaide in 1836. Before doing so I will continue with the discussion of sexually transmitted disease spreading into the Aboriginal populations at the edges of the European settlements.

There is little surviving evidence of syphilis among the Kaurna living around Adelaide and the surrounding coastal plains during the early years of the settlement. Dr Wyatt was the first Aboriginal Protector appointed in South Australia and held the post for two years from 1836. In an inquiry into the state of the Aboriginal people of the colony Wyatt remarked that he first became aware of sexually transmitted disease, although 'manifested as very considerable difference in appearance and symptoms' after

his first year as Protector (SAPP 1860). It is not clear, however, whether Wyatt was referring to syphilis, gonorrhoea or some other disease. Nor is it clear that he was referring specifically to the Kaurna or to the Ngarrindjeri and Meru with whom he also had contact. Protector, Dr. M. Moorhouse, appointed in June 1839, in a brief report on the health of the Aboriginal people, indicated that syphilis was present among Aboriginal people in Adelaide (Moorhouse 1844). Again there is some doubt as to whether he was referring to the Kaurna or a visiting group. In 1843 Moorhouse estimated that there were 300 Aboriginal people in the Adelaide district in regular contact with the settlers but many of these were Meru and Ngarindjeri who regularly visited the region following European settlement (Moorhouse 1843a).

The diffusion of sexually transmitted disease into the Kaurna and its subsequent morbidity and mortality among them may have been less than it had been among the other Aboriginal groups living near the settlements of Sydney and Melbourne. Summers (1986: 288) has argued that the more liberal, humanitarian background of the first settlers to South Australia, and the more orderly nature of colonization had the effect of creating amicable relations with the Kaurna. Also, the absence of convicts in the colony may have had some effect in reducing sexual relations between the two groups and consequently the prevalence of sexually transmitted disease. On the other hand, it would seem highly unlikely that sexual relations between free European males and Aboriginal women did not regularly occur. It was a common practice for Aboriginal women to be prostituted in exchange for tobacco, alcohol, and food from the early days of the settlement (Summers 1986: 299). The timing of sexually transmitted disease into the Kaurna population of the Adelaide Plains is unknown.

Its timing and distribution north and west of Adelaide is more readily substantiated. During the 1850s and 1860s European pastoral leases and settlements

became established on Eyre Peninsula and in the recently surveyed regions north of Spencer Gulf (Williams 1969: 16-20). As early as 1852 sexually transmitted disease was being reported as a serious health problem among the Banggarla and Nawu who had contact with the pastoralists. Moorhouse reported to the South Australian Government that there had been a 'considerable mortality' among the adults. Thirteen men and one woman had died chiefly of 'secondary symptoms of venereal' (Moorhouse 1852b). Moorhouse wrote that he could do little about the problem other than send twenty blankets to be given to those suffering.

The process of settlement and Aboriginal dispossession north and west of Adelaide continued into the final decades of the nineteenth century with recurrent releases of pastoral land tracts at the head of Spencers Gulf and into the Flinders Ranges (Brock 1995: 220). Sexually transmitted disease among the Nukunu and Ngadjuri spread at a comparable pace. In 1867 the newly appointed Sub-protector Scott travelled 2,000 miles (3,200 Km) along the spine of the Flinders Ranges and between Lakes Frome and Torrens. He met with the Banggarla, Adnyamathanha, and Kuyani and reported to Protector Buttfield, with some ambiguity, that their general health was good but there had been a great amount of 'sickness' among them (Buttfield 1867). Subsequent reports by Protector Buttfield to the Commissioner for Crown Lands (now the responsible body for Aboriginal affairs in South Australia) allude to a satisfactory state of health and make no reference to any sexually transmitted disease present among them (Buttfield 1871a,b, 1872).

By the mid 1870s further pastoral development and an increase of the European population altered the Aboriginal health situation. Buttfield (1874) now reported that there was 'a great deal of sickness' and syphilis was one of the principal diseases. Once established, syphilis was maintained among the Aboriginal people who

had frequent contact with Europeans. In his annual report on the state of Aboriginal people in the northern district of the colony for 1887 Sub-protector Beasley recounted that:

> ...there are a great many suffering badly from venereal disease, caused principally by contact with Shearers and those from Town, this is a sad thing for these unfortunate creatures, but what can we do. The evil cannot be stopped, nor the disease cured in most cases, as they cannot be induced to use or submit to the necessary Medicinal remedies... Consequently this terrible disease is spreading (Beasley 1888).

By 1879 the pastoral frontier had moved north of the Flinders Ranges and was now into the arid regions beyond Lake Eyre. Syphilis had spread with the northern movement and was infecting the Dieri whose lands centred around Cooper Creek on the edge of the Strezlecki Desert. The missionary, C.A. Meyer, at Kopperamana Mission reported to the Sub-protector that:

> The general health of the Aborigines has not been very good during the past year as they all - with only a few exceptions - suffer from Syphilis [emphasis in the original] it would therefore be very advisable to have medicines for them (Meyer, C.A. 1880).

Syphilis developed into a major health problem among the Dieri with little or nor treatment or help available to them. In some circumstances help for the inflicted was actively denied. It was the policy of the Lutheran missionaries at the Killapaninna Mission settlement to expel those suffering the disease because of their perceived 'immoral behaviour' (Stevens 1994:191).

The effects of chronic sexually transmitted disease on the fertility of the Aboriginal groups cannot be assessed with any degree of precision from the historical records since there is not enough quantitative demographic data. It is reasonable to

assume, however, that where there is a high prevalence of sexually transmitted disease, particularly where it is likely to go untreated and the chances of reinfection are high, there is a strong likelihood of associated infertility (Cates *et al*. 1990: 214). A hint of such a situation is given by Meyer in 1885 among Aboriginal people at Kopperamana mission settlement. Although Meyer kept no written record of births and deaths he observed that there had been no increase among the sixty-five living on the mission settlement nor among the forty or fifty living nearby (Meyer 1885). Whether this was an effect of a reduction in the birth rate, mortality, or migration was not specified by Meyer. If it was due to reduced births then gonorrhoea may have been involved. Tubal destruction from pelvic inflammatory disease in females and epdidymo-orchitis in males are complications of untreated gonococcal infection leading to sterility.

Several years earlier Taplin, in a report on the Ngarrindjeri living on the Point McLeay settlement, stated that:

> I am well aware that the temptations held out by white men to the native girls are constant and attractive, especially to those who can earn so little money... I notice that decent, chaste women among the natives almost invariably have large families. I know of three who have had three children in five years (Taplin 1868).

Taplin is referring mainly to those who were living on the Point McLeay settlement and its outstation. Like most missionaries Taplin strongly opposed pre-marital sexual relationships and encouraged monogamous marriages. Such a practice, if maintained, would reduce the incidence of sexually transmitted infection. The implication of 'decent and chaste' women having large families may then be an indicator of the reduction of sexually transmitted disease, particularly gonorrhoea, and a reduction of associated tubal infertility.

4.4.2 Beyond the frontiers

So far discussion has focussed on the introduction and spread of sexually transmitted disease as a consequence of European frontier movements. Below I wish to examine the evidence for the spread of what was most likely venereal syphilis ahead of the European frontiers and before the settlement of South Australia. I will again use historical literature as the main form of evidence but support it with the use of osteological evidence from the Murray River diagnosed with treponemal lesions.

Historical evidence

During his exploratory expedition along the inland river systems in 1830 Charles Sturt (1833) reported the presence of a disease severely affecting many of the Aboriginal people he encountered. Sturt began his exploration at the limits of European settlement; first by tracing the Murrumbidgee River from its upper reaches westwards into the interior, then entering the Murray River and following it to its termination at Lake Alexandrina on the south coast of South Australia. The day following his entry to the Murray, Sturt and his party were met by a group of eighty-three men, women and children (either Latje Latje or Kureinji) whose appearance he described as being 'extremely picturesque and singular' (*ibid*: 91). Two days later, after setting camp, Sturt was approached by a group of thirty-five:

> The most loathsome diseases prevailed among them. Several were disabled by leprosy, or some similar disorder and two or three had entirely lost their sight (*ibid*: 96).

Further downstream Sturt was again struck by the appearance of a disease, this time among the Barkindji. He saw many more affected by it:

> The most loathsome diseases prevailed throughout the tribes, nor were the youngest infants exempt from them. Indeed, so young were some, whose condition was truly disgusting, that I cannot but suppose they must have been born in a state of disease; but I am uncertain whether it is fatal or not in its result, though, most probably it hurries many to a premature grave. How these diseases originated it is impossible to say. Certainly not from the colony, since the midland tribes alone were infected. Syphilis raged amongst them with fearful violence; many had lost their noses, and all glandular parts were considerably affected. I distributed some Turner's cerate to the women, but left Fraser to superintend its application. It could do no good, of course, but it convinced the natives we intended well towards them, and, on that account, it was politic to give it, setting aside any humane feeling (*ibid*: 125).

Crossing what is now the border of South Australia and into the territory of the Meru, Sturt and his party were in daily contact with larger groups, now numbering up to 200 or more:

> Their sameness of appearance, the disgusting diseases that raged among them, their abominable filth... combined to estrange us from these people, and to make their presence disagreeable (*ibid*: 131)

Sturt and his party continued to observe the presence of this same state of disease among Aboriginal people during their progress downstream towards Lake Alexandrina. Finally he comments on the Ngarrindjeri he met on the lower Murray:

> It would disgust my readers were I to describe the miserable state of disease and infirmity to which these tribes were reduced. Leprosy of the most loathsome description, the most violent cutaneous eruptions, and glandular affections, absolutely raged through the whole of them (*ibid*: 148).

From Sturt's comments there can be little doubt that the Aboriginal people he met were indeed suffering from some form of debilitating disease; but what disease (or diseases) was it? Sturt alternately called the symptoms he saw leprosy and syphilis. His confusion is understandable when the regions of the body displaying leprosy lesions are compared to those affected by syphilis. Leprosy affects the cooler parts of the body. Infection involving the nasal mucosa, upper respiratory tract, and anterior segments of the eyes leave the victim with nodular-like distortions of the face which can be confused with

syphilitic destruction in the same areas (Benenson 1990: 243-244; Connor & Gibson 1988:397-400: von Lichtenberg 1989: 380-382). The testes can also be affected giving the misleading presentation of a sexually transmitted disease to the inexperienced observer. Because of their similar clinical features leprosy and syphilis have often been confused by early writers.

I think that we can rule leprosy out. While this disease was a problem among Aboriginal people in the northern Australia where it became endemic, no clinical or palaeopathological evidence for this disease has come from Aboriginal people in the southern half of Australia (Basedow 1932: 13; Cleland 1928: 57; Cumpston 1989: 208; Elphinstone 1971: 300; Webb 1989: 90). The disease described by Sturt appears to be of very high prevalence, affecting all ages. Leprosy, however, has a low attack rate and a long incubation period ranging on the average from three to six years making its communicability low with clinical consequences presenting in just a few individuals (Arnold 1966; Steinbock 1976) Leprosy then, does not fit the description put by Sturt.

What then of syphilis? Sturt was an astute and methodical observer and recorder during his expeditions but his descriptions of the disease he saw are those of a lay person. Sturt's education was not in medicine but in classics, mathematics and the natural sciences, from which he developed a 'superior dilettante acquaintance with botany, ornithology, zoology and geology' (Beale 1979:11). We have little way of knowing how experienced he was in identifying the clinical signs of diseases, although like others of his time he would have had at least a passing knowledge of syphilis and its symptoms having lived in England where it was a common disease of adults (Arrizabalaga 1993: 1029; McNeill 1976: 203).

Nevertheless, Sturt's use of the term 'syphilis' cannot be seen as a firm indicator that the disease existed among the people he saw along the Murray River. But if we look more closely at his comments in an aetiological and epidemiological context, several points emerge which indicate an epidemic disease, most likely syphilis, was spreading along the Murray River populations at the time Sturt and his party were exploring the river:

- The infected Aboriginal people Sturt saw were living in a region far removed from any colonial settlements in a land that was largely unexplored by the Europeans.
- The disease he saw was active between a point upstream of the Murray-Darling junction (between the present towns of Robinvale and Mildura) and the mouth of the Murray River at Lake Alexandrina.
- By Sturt's implication the prevalence of the infection was high among the groups he saw.
- Both sexes and all age groups (infants, adolescents, adults, and elderly) were infected.
- Infection was severe among all ages.
- Infection may have been congenital in some cases.
- Areas of the body involved were: integument tissue over much of the body, soft tissue and bone of the nasal region.
- Blindness resulted in some cases.

The diseased state described by Sturt of the Aboriginal groups along the middle and lower Murray River is indicative of an active epidemic of treponematosis with a high prevalence among the populations. Destruction of the peri-nasal soft tissue and bone would indicate that the disease is in its tertiary stage in some of the individuals. The

blindness seen by Sturt may be the result of tertiary syphilis of the central nervous system (neurosyphilis) or interstitial keratitis, a condition of late congenital treponeme infection affecting mainly the 4 to 30 years age group (King & Nicol 1975: 85, 133). The primary and secondary stages, particularly the dissemination of skin lesions were most likely to be the main representation of the disease, but this is not clear from Sturt's descriptions. Foetal involvement also seems to be represented with infants displaying lesions to the surface of the body similar to adults.

Osteological evidence

Further evidence linking the clinical signs seen by Sturt to an epidemic of treponemal infection comes from examinations of Aboriginal crania from the Murray River in South Australia. From a total of 94 (6.8% of sample) Murray River crania diagnosed with treponemal lesions (see Webb 1984a, 1989, 1995 & below) two specimens bearing severe osteological lesions of tertiary syphilis are described below. The first is from the eastern limits of the Meru and was found in sand deposits bordering the Murray River in the Riverland region. The second was found near the town of Manum in similar deposits. Its location is at the southern limits of the Meru bordering the Ngarrindjeri. In terms of river distance they are separated by 340 kilometres (Engineering and Water Supply Department 1975). The extracts from Sturt's narrative presented above indicated that he was constantly seeing active lesions among the Meru during his transit of this section of the Murray River. The two specimens showed clear evidence of gummatous osteoperiostitis and were diagnosed as treponemal according to Hackett's (1976) diagnostic criteria for syphilis, yaws and treponarid in dry bone. It is worthwhile presenting and describing these two crania with a view of showing the pathogenic pattern and extent of destruction; tying it in with Sturt's descriptions, and to eliminate other forms of treponemia. Neither cranium has been dated. It should be noted

however, that these two specimens represent probably the severest form of treponemal disease and are not indicative all the crania diagnosed with the disease.

Skull A911 434, (South Australian Museum) an adult male showed a severe pattern of lesions. Infection involved nasal, frontal and right parietal bones (Plates 4.1, 4.2, 4.3). The nasal bone has been completely destroyed and erosion has proceeded below the anterior nasal spine to the superior edge of the maxilla. Located above the lateral margin of the right orbits is an irregular centre of serpiginous cavitation (caries sicca sequence 6, Hackett 1976: 42) involving the rim of the orbit and extending obliquely towards the mid-line for a distance of four centimetres. The lower margin of the cavity has a sharp edge and the upper margins are slightly raised and rounded. The walls of the cavity are concave and the diploe and inner table of the skull have been perforated. No bone remodelling indicative of healing was observed. Further up the frontal two additional centres of cavitation (caries sicca sequence 6) have united to form a crescent shaped region approximately four centimetres long and one centimetre wide extending from the mid-line down towards the anterior zygomatic arch. The crescent lesion has concave walls ending in sharp edges at the rim. Three shallow circular depressions (caries sicca sequence 4 to 5) form a line parallel to the coronal suture. The central one of the three has a sharp, ridged margin and is deeper with radial scarring beginning. The other two depressions have less defined, flattened margins. No thickening of the inner surface of the frontal is obvious below the depression nor were there any other changes observed on this surface. The right parietal is more heavily involved. Two shallow depressions, similar to those on the frontal, and at the same stage of development (caries sicca sequence 4 to 5), are located on the superior-anterior aspect of the parietal between bregma and vertex forming a line at right angles to the sagittal suture. The more medial depression borders, but does not cross, the suture line. Both depressions have flattened rims. No radial scarring or inner table involvement was

observed. On the posterior half of the same parietal a confluence of clustered pits leading into serpiginous lesions has resulted in bone destruction and perforation of the diploe and inner cranial surface in four distinct regions (caries sicca sequence 6). The cavitations roughly follow the temporal lines. The ectocranial edges of the cavities are sharp with sloping sides leading down to the perforations of the endocranial surface, the edges of which are also sharp. Another similar sequence of five lesions has penetrated the dorsal half of the parietal resulting in considerable bone destruction. Clustered pits (caries sicca sequence 1) surround the areas of penetration. The nature and distribution of these lesions is indicative of gummatous destruction beginning on the external surface of the calvarium caused by and infection extending from the soft tissues of the pericranium.

Skull A351 (South Australian Museum), an adult male, shows a similar pattern of bone destruction (Plate 4.4). Nasal destruction in this individual has occurred and the frontal bone displays several regions of radial scarring (caries sicca sequence 5) with bone healing evident on the floor of the lesions and flattening rim margins. One of these lesions is visible on the right mid region of the frontal bone. The posterior region of the right parietal was the most effected at time of death. Active bone destruction is evident in two areas. A series of four foci of destruction forms a row along the inferior temporal line ending at the lamboidal suture. The lesions have not progressed to the occipital bone. A second area of two destructive foci is located inferior and follows the temporal line at the junction of the lamboidal and squamous sutures. The occipital and temporal bones are not involved. All six cavitations are irregularly rounded with sharp edges on the ectocranial surface and sloping walls penetrating to the cancellous tissue of the diploe. The most inferior of the lesions is circumscribed by newly regenerative tissue. The destruction of bone tissue in this individual is progressing from right lateral inferior region of the parietal progressing along the temporal lines.

The cause of the lesions in both individuals is from gummatous infection eroding the ectocranial bone followed by a regenerative process laying down new bone tissue. Gummatous osteoperiostitis, as displayed in these two skulls is a characteristic lesion of tertiary syphilis (Hackett 1976; Ortner & Putschar 1981; Steinbock 1976). Gumma forms as a combined result of the restriction of blood supply to the bone by thrombosed vessels entering the cranium from the pericranial tissue, and the toxic product of the treponeme (Steinbock 1976: 123). A diagnosis of chronic tertiary syphilis can be supported.

Several important questions now arise. What type of treponemal infection was affecting these skulls? Was it venereal syphilis, or a form of non-venereal syphilis, or yaws? Was it introduced, or was it occurring naturally among Aboriginal populations? How long had it been present and how long did it last among the Murray people? Why was the disease restricted to the middle and lower Murray populations and absent from those further east as Sturt stated?

Webb (1984a, 1989, 1995) carried out a comprehensive investigation of bone pathology using several Aboriginal skeletal collections from most of Australia's geographic and climatic regions. A substantial part of the investigation involved collections from the upper, middle, and lower Murray River. Webb found that these populations had the highest prevalence of treponemal bone lesions for the whole continent (Table 4.2).

Table 4.2 Treponemal infection in undated Australian Aboriginal crania (after Webb 1984a)

Area	n	Treponema	Percent
River Murray	1374	94	6.8
South Coast	261	7	2.7
Desert	183	9	4.9
Tropical	154	6	3.9
Coast	219	4	1.8

He concluded that the disease Sturt observed was indeed a form of treponemal disease but could not determine whether it was introduced venereal syphilis or an endemic form of the disease transmitted by non-venereal means. His inability to resolve the nature of the disease stemmed from two main considerations. Firstly, while the diagnostic criteria for treponemal lesions in bone have been determined (Hackett 1976) the discrimination between venereal syphilis, non venereal or endemic syphilis, and yaws has not. The second problem is related to the skeletal samples themselves. Most of the skeletal collections are poorly dated. Webb had little or no temporal control on the collections of individuals he diagnosed with treponemal disease, and in many cases had difficulty in determining whether they were pre-European contact or post-European contact. This is particularly the case with much of the Murray River collections which were collected without detailed recording of their spatial or temporal integrity. He concluded that the 'extent and prevalence of treponemal disease in pre-contact Australia cannot be determined from skeletal material held in collections around Australia until some of it is dated' (Webb 1989: 98).

In order to establish which form of treponemal disease was affecting the Murray River groups we must now consider yaws, non-venereal syphilis (endemic syphilis), and venereal syphilis. All can display clinical symptoms similar to those seen by Sturt.

Yaws certainly appears to have been common until recent times in some Aboriginal populations but its distribution is restricted to the tropical regions of central and northern Australia (Elphinstone 1971: 297). Long standing endemicity of the disease was noted by Cleland (1928: 144) in tropical northern Australia; by Hackett (1936) among the Aranda of northern and central Australia; and by Basedow (1932) in the Kimberley region of Western Australia, but no trace of active yaws has ever been found among the Aboriginal groups of southern Australia (Cleland 1928: 141).

The temperate climate along the Murray River corridor does not favour yaws. The yaws spirochaete is a fragile organism outside its host, highly intolerant of seasonal changes, particularly seasonal cold and low humidity such as are characteristic of the Murray River and southern inland regions of Australia. The optimum environment for the organism and for the disease to become endemic in human populations is the humid equatorial, lowland rainforests where all months have a mean temperature exceeding 18^0C and a rainfall exceeding 65 mm per month (Pirie 1972: 189). The contemporary world-wide distribution of yaws in tropical and sub-tropical regions reflects this optimum environment. On the basis of this geographical restriction it is unlikely that yaws was the disease that Sturt saw or that is represented by the cranial pathology.

Yaws can then be excluded on a further number of points. It is primarily a disease of children and adolescents (Cleland 1928: 144; Benenson 1990: 484) with males being more frequently affected than females. It is usually contracted in infancy or early childhood through surface contact with infected children or adults. The condition may last for several years into adulthood, often in latent form (Steinbock 1976: 142). The initial lesion commences as a large, rounded papule (mother yaw) at the point of inoculation usually on the leg. It may also occur on the mouth of a suckling infant or on

the breasts of a nursing mother (King & Nicol 1975: 302). Within three to six weeks after initial infection secondary eruptions occur and can extend over much of the body's surface. In many cases the disease is self-limiting and does not progress into the tertiary stage to involve skeletal and soft tissue organs, or the central nervous system. In later life all that may remain of an infection is scarcely perceptible scarring in the areas affected.

Bone lesions in yaws occur in about 1% of cases (Ortner & Putschar 1981: 180) and are commonly confined to the limbs. The most frequent bone is the tibia, followed by the fibula, and the distal third of the femur. Cranial involvement in yaws is even less common and much less destructive than from syphilis (Ortner & Putschar 1981: 180). Steinbock (1976: 145) reports that of 119 cases of osseous yaws, the cranial vault was affected twenty times (16.8%), and of these, only one approached the severity of bone destruction that is found in syphilis. The main involvement in yaws was destruction around the nasal region. The frontal bone may be involved, but only in rare cases of tertiary yaws. When affected, the frontal bone usually displays shallow pitted depressions which do not perforate the ectocranial surface and are not characteristic of the carries sicca common in tertiary syphilis (Ortner & Putschar 1981: 180; Steinbock 1976: 145).

With yaws then seeming unlikely, the two remaining forms of treponema - endemic non-venereal syphilis and venereal syphilis - remain. Could the disease Sturt saw be an endemic form of syphilis that had flared up into an epidemic just as he was proceeding down the Murray River? This is possible but again unlikely. Endemic syphilis, like yaws, is a disease of childhood, usually contracted before the age of fifteen years (Garner *et al.* 1972: 287; Steinbock 1976: 138) with self-limiting symptoms and a low case-fatality rate (Benenson 1990: 425-6).

There is no definitive evidence of treponemia in Murray River skeletal material dated before European contact. Table 4.3 displays the collections from Southeast Australia that have been dated. All are from the Murray River apart from Broadbeach which is an east coast sample.

Table 4.3 Prevalence (%) of pathology in dated skeletal collections of Southeast Australia (Blackwood & Simpson 1973; Haglund 1976; Pretty 1977; Pretty and Kircun 1989; Prokopec 1979; Sandison 1973: 173; Thorne 1971, 1975).

Sample	Cranial Treponema	Post-cranial infection	Porotic Hyperostosis	Trauma
Roonka (n=144) 7000-50 BP	1	5	1	14
Broadbeach (n=139) 1390-50 BP	0	3	23	15
Chowilla (n=72) 6000-170 BP	0	0	0	2
Murray River Pleistocene (n=44) >9500 BP	0	_	17	14

The first of these collections to be excavated under controlled conditions was the Chowilla collection. It came from a series of burial places located in the Murray Valley between the present towns of Mildura and Renmark and Lake Victoria (Blackwood & Simpson 1973). Radiocarbon dates of organic content from bone of one burial and on charcoal intimately associated with another at Chowilla Station (near the border of South Australia and Victoria) put the antiquity of the burials between 4,000 and 6,000 BP. Pathological examination of this material showed no changes attributable to treponemal disease (Sandison 1973: 173). The skeletal collections excavated from Broadbeach (Haglund 1976) and the Murray River Pleistocene collection (Thorne 1971, 1975) have also shown no evidence of treponemal infection (Webb 1989).

A large burial site at Roonka Flat on the lower Murray was excavated by Pretty (1977). Radiocarbon analysis of sample from its stratigraphic horizons established a range of dates from 18,000 BP with skeletal material coming from horizons dated 7,000 -50 BP (Pretty 1977; Brown 1996). Prokopec (1979) carried out a biological analysis of this collection examining 132 full and partial skeletons from excavated graves and surface exposures. Although there were four cases of bone changes suspected to be from treponemal infection he could not confirm the presence of the disease.

In a more recent re-examination of skeletons (*n* = 144) from Roonka, including those examined by Prokopec, Pretty and Kircun (1989) observed periostitis, osteitis, osteoperiostitis and chronic osteomyelitis in six post-cranial bones and cranial lesions in one individual from the Roonka III horizon radiocarbon dated between 4, 000 and 220 BP (Table 4.3). They ascribed the lesions to either yaws or endemic non-venereal syphilis. All the postcranial osseous changes however, can be grouped together as nonspecific infections caused by various kinds of pathogens (e.g. *Staphylococcus* sp.) reacting with the bone (Steinbock 1976: 60) and are not peculiarities attributed only to treponemal infection. The main bones infected were the longbones of the arms and legs all of which are susceptible to nonspecific osteomyelitis caused by primary and/or secondary infection. While treponema is possible as one cause of the postcranial osseous lesions seen in the Roonka individuals it is by no means the only possible cause (Dowling 1990: 59). The single case of cranial lesions attributed to yaws is in an adult male cranium coming from Tomb No. 18. It has not been described by the authors nor has this individual been firmly dated. A photograph of the cranium appears in Prokopec (1979) and Pretty & Kircun (1989) papers but again is not accompanied with any written description. In the latter paper the lytic lesions visible on the frontal bone are diagnosed with some reservation as being due to 'treponarid infection yaws?'. The skull is intact

with very little sign of post-depositional deterioration and may well be of post-contact origin coming from the later range of radiocarbon dating of the site. If so, then the suspected treponemal infection may well be venereal syphilis acquired from Europeans.

It is realised that lack of evidence for pre-contact treponemia on the Murray River does not prove that it had never existed there. This can only be resolved by a series of reliable radiocarbon age determinations for existing skeletal material held by museums displaying unequivocal diagnosed treponemia sequelae or by newly excavated samples from stratigraphically sequenced deposits with reliable radiocarbon dates. There is at present little prospect of the former as this is seen by Aboriginal groups and museum keepers as culturally sensitive and inappropriate. They are therefore reluctant to sanction such tests. Time may tell for the latter.

There are still problems with accepting endemic non-venereal syphilis as the disease Sturt observed. Was it just by chance that it was, for some reason, in a severe form of an epidemic phase, when Sturt passed through the region? How often an endemic disease flares up into a severe epidemic is dependent on the number of susceptible individuals in the community. In most cases of infectious endemic disease, endemic syphilis and yaws included, those susceptible are the young who have not been exposed to the pathogen and who have not developed immunity to the disease. Such a scenario does not sound plausible for the Murray River populations. The disease Sturt saw was affecting all the ages, both young and old and the symptoms were severe. This is consistent with the two crania described above, both were adult and the treponemal symptoms had reached a severe tertiary stage characterised by bone destruction.

A further problem in accepting the existence of endemic syphilis along the Murray is that a considerable degree of cross-immunity to venereal syphilis is acquired by

infection from non-venereal syphilis. Schell *et al.* (1982) have shown that hamsters infected with endemic syphilis (*T. pallidum* Bosnia A) were resistant to challenge from other virulent treponemes causing yaws and venereal syphilis symptoms. A similar indication of treponeme cross-immunity in humans has been observed in regions where other forms of treponemia are endemic (Garruto 1981: 561). An example of this comes from the Pacific Islands. Until yaws was largely eradicated in many Pacific Island populations as a result of concentrated medical campaigns, venereal syphilis was virtually unknown even after close contact between the indigenous populations and European colonists had been well established (Pirie 1972: 192). If endemic treponemal infection had been present along the Murray before the European contact we would expect to see a low prevalence and attenuated clinical symptoms of venereal syphilis when it was introduced. History shows us that this is not the case.

Venereal syphilis was just as common and severe among the Ngarrindjeri and Meru as it was elsewhere in the colony. George Taplin, who spent from 1859 to 1879 among the Narrindjeri of the lower Murray and coastal regions as founder and superintendent of the Point McLeay mission settlement, writes:

> I have seen cases, even bad cases of syphilis amongst the natives. I am sure the disease was imported among them; they knew nothing of it before the advent of whites - this is the testimony of the natives. I have known fatal cases, also cases where the tibia was affected, and bony excrescences on the skin, with atrocious neuralgic pain (Taplin 1879: 46)

During his residency at Moorundie Aboriginal station from 1841 to 1843 as Protector, Eyre observed many Meru and Ngarrindjeri inflicted with venereal syphilis. His account (originally written in Latin) reveals more about the disease:

> Of the diseases from which they suffer since the arrival of the Europeans by far the most frequent and the most deadly is the venereal stain... Among the natives this disease manifests itself in the same way as in many Europeans, yet for various causes it is even more hateful, especially because round pustules, commonly the size of an ounce weight, rise at the same time from the skin. The centre of these is gradually filled with flowing pus, then as they grow larger and larger and disperse, the surface of the whole body is affected with wasting and scab which cause horror and disgust to those near them. These ulcers sometimes may persist for six or eight months; but generally when irritants or caustics are applied locally they are cured within three weeks... After the first or second year the disease disappears, but sometimes causes death (Eyre 1845: 379, translation in Cleland 1928: 142)

Eyre appears to be in no doubt that the disease was venereal syphilis and that it followed a similar symptomatic course among the European population as it did among the Aboriginal population. His description of 'round pustules' rising from the skin and dispersing over the body corresponds to the secondary stage maculopapular rash which disseminates in a variety of organs, most strikingly the skin over much of the body surface. Secondary symptoms of syphilis often spontaneously disappear within weeks and up to twelve months after formation so it is doubtful whether Eyre's 'irritants or caustics' were effective. The disappearance of the disease symptoms after the first or second year as noted by Eyre would equate to the latent period between the secondary and tertiary stages of the disease. This observation is interesting because it suggests that Eyre was seeing only the primary and secondary stages of the disease and not the third and final stage. Symptoms of tertiary syphilis would indicate that the disease had been present among them for some time and many would have been initially infected much earlier than when Eyre was seeing the disease.

The most parsimonious explanation for the disease Sturt observed is in terms of an epidemic of introduced venereal syphilis spreading along the Murray corridor. This is supported by osteological evidence of treponemia and by further historical

documentation of venereal syphilis in the Murray population, elsewhere in South Australia , and in the other eastern colonies.

An early introduction into South Australia

A final set of questions must now be addressed. If venereal syphilis was the disease seen by Sturt it was present before the establishment of the colony of South Australia. When was it introduced, by whom, and where? While these questions cannot be answered unequivocally, a strong contender for the introduction of venereal disease is the sealing and whaling industry that began around the islands in Bass Strait and off the South Australian coast in 1803 (Fig 4.1). Settlements were established on Kangaroo Island around this time (Moore 1923) by the sealers, and ships often visited the mainland coast to kidnap Ngarrindjeri women for wives and slaves (Summers 1986: 285). In 1819 Captain Sutherland of the brig *Governor Macquarie* recorded in his diary while at Kangaroo Island:

> They have carried their daring acts to extremes, venturing on the mainland in their boats, and seizing on the natives, particularly the women, and keeping them in a state of slavery, cruelly beating them on every trifling occasion, and when at last some of the marauders were taken off the island by an expedition from New South Wales, these women were landed on the main with their children and dogs, to procure a subsistence, not knowing how their own people would treat them after a long absence (Moore 1923: 121).

There is no direct evidence for the entry of sexually transmitted disease at this time, but a final point coming from Sturt's observations supports a southern coastal entry (Cleland 1928:141). Sturt first recorded the symptoms of syphilis at a point upstream of

the Darling River junction, then continued to make similar observations among the Aboriginal people he met along the remainder of the river to its mouth. He makes no mention of the disease among the Aboriginal communities before this point. An origin from the east then seems unlikely. Discussing the origin of syphilis along the Murray Cleland (1928:141) concluded that 'It is possible that from such a source syphilis may have been introduced first amongst the Encounter Bay natives [the Ngarrindjeri] and then have spread to those of the lakes [L. Alexandrina and L. Edward] and up the Murray'. Sturt had observed the disease in 1830, twenty-seven years after the first European contact with the Ngarrindjeri. This is an acceptable time frame, I would think, for venereal syphilis to spread from the coast to the point from where Sturt began to observe it.

4.5 Conclusions

The observations by the colonial writers document the spread of sexually transmitted disease, particularly venereal syphilis, among the Aboriginal populations throughout the European settled regions of Southeast Australia and beyond their frontiers. Considered together they reveal several epidemiological factors.

Sexually transmitted disease, particularly syphilis, spread among the Aboriginal populations in epidemic waves; in one instance together with the expansion of European settlements, and in the other ahead of the frontier. The major foci of introduction and spread of syphilis were from the settlements of Sydney (1788), Melbourne (1835), and Adelaide (1836). Once these centres were established the colonial settlement pushed inland and along the coast with a concurrent introduction and dissemination of syphilis among the Aboriginal populations. A possible secondary point of introduction was from the coast of South Australia in the vicinity of the Murray River mouth and Lakes Alexandrina and Edward sometime after 1803 and prior to the

establishment of the colony. The most likely cause of this introduction was the establishment of the sealing and whaling industry. By 1830 the disease had spread from the coastal Aboriginal communities into the communities along the Murray River corridor to a point just below the junction of the Darling river.

Both sexes were infected by sexually transmitted disease. While the most common introduction of syphilis into the Aboriginal communities would have been through sexual contact between European males and Aboriginal females it readily spread within the Aboriginal communities by further sexual relationships. A high rate of early and late congenital syphilis symptoms among pre-pubescents would be expected to have occurred.

The fulminating signs that syphilis exhibited among the Aboriginal people of Southeast Australia are similar to those recorded when introduced into other immunologically unprotected communities in the past. Initially the disease appears to have been highly infective with severe and life-threatening symptoms. An unusual degree of severity of clinical symptoms was commonly noted among the Aboriginal groups compared with its pathogenesis among the Europeans. This suggests that it was a new disease in a population whose individuals had little effective immunological resistance to its effects. A similar pattern of infectivity and severity of symptoms was documented in the early 16th century when syphilis reached epidemic proportions in Europe (McNeill 1976: 63); in the post-contact period in America (Crosby 1972, 1986), and the Pacific Islands (Marshall 1993: 490).

Although not always detectable from the historical literature, gonorrhoea most likely accompanied the transmission of syphilis and quite likely was of higher morbidity. Its effects on the fertility of males and females can be assumed to have been serious.

The arrival of syphilis to Australia with the First Fleet in 1788 and its spread into the indigenous population of Australia represents the final continental introduction and establishment of this disease.

CHAPTER FIVE

Tuberculosis

5.1 Introduction

Tuberculosis and humans have had an association stretching back at least 8,000 years (Merbs 1992: 17). It is not known, however, just how much longer this association has been. One school of thought presents the view that tuberculosis and the genus *Homo* have evolved together - the chronic nature of the disease enabling it to be supported and transmitted within small populations. A second view is that the disease evolved from a bovid zoonosis to a human disease during the early agricultural periods in Europe and Asia when people began to domesticate and live closely with cattle (Dubos & Dubos 1953; Johnston 1993: 1062; McKeown 1991: 38, 49). Whatever its origin, tuberculosis was present among most people worldwide by the end of the prehistoric period. Notable exceptions to this were the Maori of New Zealand (Bryder 1991: 80; Johnston 1993: 1062; Wells 1991: 97) and the Aboriginal populations of Australia (Basedow 1932: 16; Thomson 1991: 62; Webb 1984a, 1989, 1995; and below).

Using eighteenth and nineteenth century literature pertaining to tuberculosis, this chapter will examine the effect this disease had on Aboriginal communities. The examination will consider both stage II (early contact and diffusion) and stage III (settlement and acculturation) of the medical model (Table 2.2). A further examination of tuberculosis and its epidemiological nature is presented in Chapter Eight which deals exclusively with stage III of the model. In this chapter the timing and dissemination of tuberculosis throughout the Aboriginal communities and its morbidity and mortality among them will be discussed.

5.2 Biology of tuberculosis

Tuberculosis is a chronic communicable disease caused by infection from various species of *Mycobacterium.* The disease usually infects the lungs causing chronic pulmonary tuberculosis in approximately 75% of cases (Bannister 1983: 166) but may cause lesions in any other organ or tissue of the human body (Table 5.1) notably in cervical and axillary lymph nodes; liver, spleen, and intestines; in the skeleton and in the genitourinary system.

Table 5.1 International Classification of Tuberculosis (ICD.9.CM 1991).

Classification Code	Clinical Manifestation
010	Primary tuberculosis infection
011	Pulmonary tuberculosis
012	Other respiratory tuberculosis
013	Tuberculosis of meninges and central nervous system
014	Tuberculosis of intestines, peritoneum & mesenteric glands
015	Tuberculosis of bones and joints
016	Tuberculosis of genitourinary system
017	Tuberculosis of other organs
018	Miliary tuberculosis
771.2	Congenital tuberculosis

Disseminated or miliary tuberculosis, where the disease is spread throughout the body by the lymphatic system and the bloodstream, and tuberculous meningitis, where the meninges surrounding the brain and the spinal cord are infected, are the two most rapidly lethal forms (Sutherland 1977:175).

In humans the main species causing tuberculosis is *M. tuberculosis* but the disease can be caused by infection from other species particularly *M. bovis*. The main

route of infection is from person to person by aerobic droplets transmitted in the air by sneezing and coughing. In this case the portal of entry of the bacillus into the body is usually the respiratory tract and initial infection begins in the lungs. Infection can also take place through the digestive tract and the skin. The digestive tract is the main route for *M. bovis* which can be ingested by humans through the milk of infected cows. Ingestion by humans of infected milk may result in extensive tuberculous disease (Youmans 1980: 371). Infection by the bovine tubercle in humans has now been virtually eliminated in Western countries by the pasteurization of milk. Infection through the skin occurs rarely under natural conditions and is usually a result of handling infectious material (Youmans 1980: 371)

Two main types of tuberculous infection are recognized, primary and reinfection or reactivated tuberculosis. Primary tuberculosis is the result of infection by the organism in a person who has had no previous contact with *M. tuberculosis* and has no developed cellular immune response to tubercular protein. The disease is characterised by rapid multiplication of the tubercle bacilli followed by a wide dissemination of the organism by the lymphatics or the bloodstream with no extensive necrosis. Reinfection tuberculosis, on the other hand, is the disease occurring in a person who has previously developed an immune reaction to tubercular protein because of a previous exposure to *M. tuberculosis*. In this case the disease usually remains localised in the lungs causing early and extensive necrosis. Dissemination to other parts of the body is usually absent unless a bronchus or blood vessel is broken, in which case the bacilli can be transported to other parts of the body (Bannister 1983; Cotran *et al.* 1989: 374-380; Youmans 1980: 374-375).

Mycobacteria are among the oldest taxonomic groups on earth. *M. tuberculosis* has become well adapted to living in humans who are its major reservoir

(Proust 1991a: 1; Youmans 1980: 371). Most people who are infected by *M. tuberculosis* remain asymptomatic and show few or no clinical signs of the disease throughout their life. In such cases the most common result is a minor pulmonary lesion which heals spontaneously, leaving no serious sequelae but giving an increased resistance to further infection. Only a fraction (around 5%) of people infected develop the clinical symptoms of the disease (Cotran *et al.* 1989: 374). All infected asymptomatic persons, however, remain indefinitely at risk of developing the disease throughout their life.

Progression of the disease into serious and life-threatening lesions depends on several factors relating to the general health, socioeconomic status, nutrition, age of the host and the amount and time of exposure, and the virulence of the bacillus (Table 5.2).

Table 5.2 Factors relating to host/pathogen and the manifestation of tuberculosis symptoms.

Factors favouring disease	Factors favouring host
Large dose of pathogen	Low dose of pathogen
High virulence of pathogen	Low virulence of pathogen
Poor nutritional state	Good nutritional state
Poor health	Good health
Low socio-economic status	High socio-economic status
Inefficient immune system	Efficient immune system
Young and old age groups	Mid-age groups

The importance of social changes cannot be underestimated in the epidemiology of tuberculosis. During periods of acute socioeconomic decline, as in widespread warfare and colonization, outbreaks of tuberculosis commonly occur in those communities most disadvantaged. For example, in Europe during the two World Wars, epidemics of tuberculosis became rampant among refugee groups and prisoners of war (Dubos & Dubos 1953; McElroy & Townsend 1989:243) even though the disease had

probably been endemic for thousands of years. A similar picture can be seen on the American continent. Although it is now widely accepted that tuberculosis was present throughout most of the Americas prior to 1492 (e.g. Allison *et al.* 1973; Armelagos 1990: 132; Arriaza *et al.* 1995; Buikstra 1981; Lucas Powell 1992: 41-53; Merbs 1992:17; Stodder & Martin 1992: 63) skeletal collections and historical documents have shown a high susceptibility of Amerindians to tuberculosis following contact with Europeans during the processes of colonization and forced resettlement (Arriaza *et al.* 1995; Carlson *et al.* 1992:143; McElroy & Townsend 1989: 308; Walker & Johnson 1992: 132). Clark *et al.* (1987: 45) have argued that the high susceptibility of Amerindians to tuberculosis following colonization was largely due to the socioeconomic changes enforced upon them rather than an innate genetic susceptibility to *M. tuberculosis* (Cotran *et al.*1989: 374).

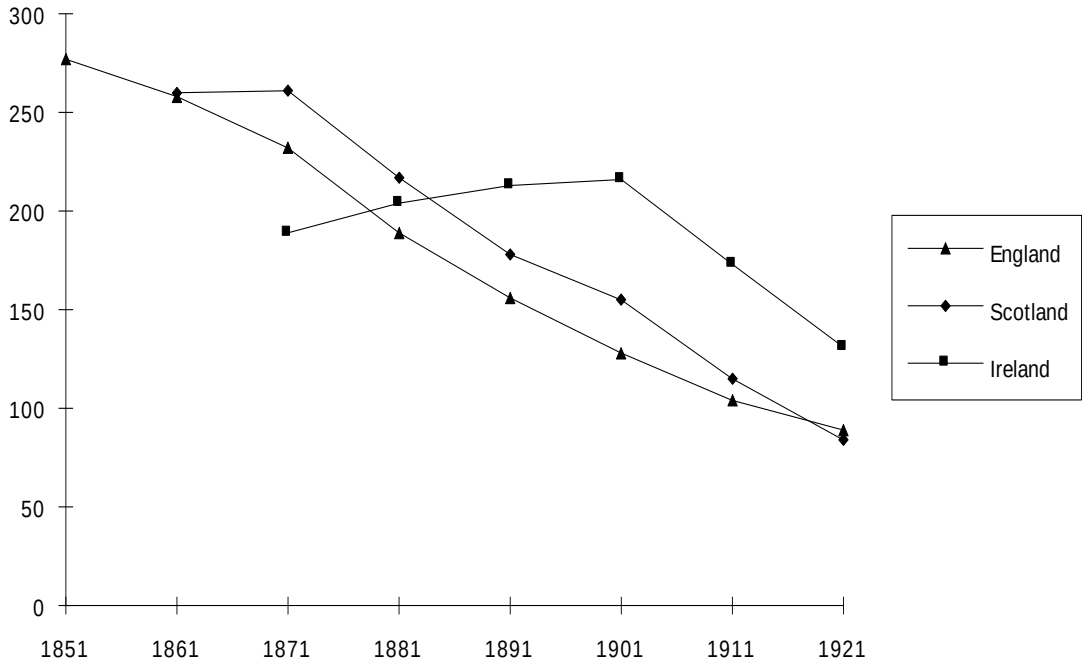
Tuberculosis infection has fluctuated during the last three hundred years. In Europe, tuberculosis was the paramount cause of morbidity and mortality in the seventeenth and eighteenth centuries (McElroy & Townsend 1989: 150) and was probably responsible for 20% of all deaths (Smith, F.B. 1988:4). In an extensive review of the disease in 1815 Thomas Young (cited in Dubos & Dubos 1953: 9) stated:

> Of all hectic affections, by far the most important is pulmonary consumption, a disease so frequent as to carry off prematurely about one-fourth part of the inhabitants of Europe, and so fatal as often to deter the practitioner even from attempting a cure.

In London alone during the middle and late seventeenth century the death rate from all forms of tuberculosis was estimated at 1,300 per 100,000 (Smith 1988:4). By 1850 the mortality rate of respiratory tuberculosis had reached a peak in England and Scotland and began to decline, but still remained one of the primary causes of death throughout the rest of the century (Figure 5.1).

The fall in the mortality rate in Europe is not fully understood, but the gradual improvement of working conditions, the development of social measures aimed towards the relief of poverty and distress, the generalised betterment of living conditions (Long, E.R. 1940; McKeown 1991), and the decline in family size (Smith 1988:8) are considered to have had an effect.

Figure 5.1 Mean annual death rate from respiratory tuberculosis per 100,000 in England and Scotland (after Smith 1988:7).



The last decade of this century has seen an increase in mortality from tuberculosis surpassing any other period in history (WHO 1996). On a global scale deaths have risen from 2.5 million in 1990, to an estimate of 2.9 million for 1995, and the

trend is expected to last to the end of the century when 3.5 million deaths annually are predicted (WHO 1994a, 1996). The major cause for the increase is the association of tuberculosis with HIV infection. The World Health Organization has estimated that a person with TB/HIV infection is nearly thirty times more likely, in any given year, to become sick from tuberculosis than a person who is only infected with the tuberculosis bacillus (WHO 1994a). In Asia, where nearly half of the people are infected with the mycobacterium, the deaths from co-infection of TB/HIV are doubling every three years. A second cause for the increase of tuberculosis globally is the impact of recent political, social and economic changes in Eastern Europe (WHO 1994b). After a steady decline of the disease for the last forty years tuberculosis has began to increase in Eastern European and former Soviet Union countries. Large cities are the most affected. Annual mortality in Moscow has increased from 27 cases to 50 cases per 100,000 population in two years. In the Siberian city of Tomsk the rate is 200 cases per 100,000 population, a level of incidence usually only found in the most severely affected populations in the world (WHO 1994b).

In Australia tuberculosis in all its forms is a minor cause of mortality. For the years 1979 and 1990 the mortality from tuberculosis was 0.5 and 0.4 per 100,000 of population respectively (Bennett *et al.* 1992).

Because of its long association with humans and its ability to attack a variety of organs and tissues, tuberculosis infection has been known by several names in the English speaking world (Dubos & Dubos 1953: 3-10; Farr 1854: 141; Morgan 1987).

Table 5.3 Terms given by medical practitioners in the nineteenth century that may be attributed to tuberculosis infection (Murray 1830; Registrar General 1863; Ruddock 1873; Smith 1988:3-5;).

Abscess in the pelvis or abscessus
Cachexia
Consumption
Consumption of the bowels
Decay
Decline
Dropsy of the brain
Enlargement of the glands (abdomen or throat)
Enlargement of the ometum
Gathering (abscess)
Haemoptysis
Abscess in pelvis or abscessus
Hives of the bowels
Hydrocephalus or water in the brain
Inflammation of the glands
King's evil
Phthisis
Pneumo-thorax
Psoas abscess
Scrofula
Scrofulosa
Struma
Swelling of the glands
Tabes
Tabes mesenterica
Tubercular disease
Tubercular peritonitis

Table 5.3 shows a variety of terms given by medical practitioners in the nineteenth century that can be attributed to tuberculosis.

'Consumption' was the most common term used in the nineteenth century referring to the most common form of the disease, pulmonary tuberculosis. Other common terms were 'Phthisis' referring to the slow wasting away of the body; 'scrofula' (tuberculous cervical lymphadenitis) referring to the direct extension of tuberculosis into the skin from underlying lymph nodes (scrofuladerma). Tuberculosis has also been

known under other names, such as 'tabes' or 'tabes mesenterica' (tuberculosis in the mesenteric glands of children resulting in progressive atrophy of the body or a part of it) and 'lupus' (scrofuladerma) (Registrar General 1863:8; Waksman 1965: 7). Many of these terms have largely been abandoned by Western medicine today. The actual name 'tuberculosis' was introduced during the first half of the nineteenth century to designate a group of symptoms characterised by the presence of tubercles in different body organs. More recently, it has come to mean any infection caused by tubercle bacilli, whether or not tubercles could be found in the infected organs (Waksman 1965: 7).

5.3 Tuberculosis among the European population of Southeast Australia

The first recorded case of tuberculosis infection in Australia was before European settlement. Forby Sutherland, a crew member of Captain James Cook's *Endeavour*, was severely ill of pulmonary tuberculosis ('consumption') when the ship entered Botany Bay on 29 April 1770. He died of the disease and was buried ashore the next day (Cumpston 1989: 276; Proust 1991b: 5; Watt 1979: 138). Cook records in his private log:

> Last night departed this life Forby Sutherland, seaman, who died of a consumption and in the a.m. his body was entard [sic] at the watering place. The circumstance occasioned my calling the south point of this bay Sutherland's Point (cited in Cleland 1938:257; Cumpston 1989: 276)

Sutherland was almost certainly not the only crew member to be suffering from the disease when the *Endeavour* reached Australia (Watt 1979: 138).

When the First Fleet left its home port of Portsmouth for Australia in 1787, tuberculosis was near its peak in the industrial cities of Britain. It is quite likely that it

was present among members of the First Fleet, and survived the long trip to Australia particularly when considering the crowded and squalid living conditions of the convicts (Bateson 1959: 60; Watt 1989: 138).

Although there are no surviving official medical statistics on tuberculosis, the first reports of death among European settlers that could be attributed to tuberculosis come several months after the arrival. John Easty, a marine private of the First Fleet, recorded several deaths in his personal diary among which are cases that he considered to be tuberculosis. He records for October 10th, 1788:

> This Day John Jones A marine Departed this Life of a concumtion [*sic*] and fever (Waksman 1965:106)

and for February 2nd, 1789:

> This Night att [*sic*] 10 oclock Cap^{tn} John Shea of Marines Departed this Life after a Long illness of a Concumtion [*sic*] and was Buried the next Day in Miliantary [*sic*] form very Neat and handsom^{e} (Waksman 1965: 108)

Easty also records four other deaths from long illness and fevers between June 1788 and June 1791 that may have been from tuberculosis. Little is known of Easty, apart from his service in the marines, and he almost certainly had no medical education, so his descriptions must be taken as a lay opinion only. Nevertheless, he was probably recording the general opinions of the medical officers' diagnoses.

A sounder confirmation of tuberculosis in the new settlement comes from records of the newly established Sydney General Hospital which listed in their 1803 returns of death, two males and two females dying of consumption during that year (Jamison 1803). Tuberculosis, however, was responsible for just 4.2% of deaths recorded

in the hospital's register. The major cause of mortality in the settlement at this time was due to dysentery which accounted for 52.1% of deaths recorded at the hospital.

The first recorded non-European death from tuberculosis was a Tahitian who was visiting the new colony. 'Mowie', who had returned from a visit to England on HMS Glatton, arrived in Port Jackson on the 11th of March, 1803, and died on the 6th August before there was a ship available for his return home. The cause of death given was 'inveterate Scrophuli and decay' (King 1803: 370).

From the beginnings of European settlement in Australia tuberculosis was constantly present among the first colonists. Governor Phillip considered that, up to February 1790, about one-third of deceased convicts under his command 'died from disorders of long standing, and which it is more than probable would have carried them off much sooner in England'. Gandevia and Cobley (1974: 123) consider this comment by Phillip to be based upon the observations of his medical officers and most likely to be made in regard to pulmonary tuberculosis.

The disease was maintained in the population of the colony, being supported by the new arrivals in the Second Fleet in June 1790 and in subsequent fleet arrivals (Cumpston 1989:276; Proust 1991b: 5). Of the Second Fleet complement of 1026, 267 died enroute and 488 arrived at Port Jackson ill many requiring medical treatment (Clarke 1981: 123) McLeod (cited in Cumpston 1989: 276), writing in the fourth decade of settlement says:

> Consumption of the lungs (phthisis pulmonalis) is much more frequent than from the mildness of the climate might be expected, and more in advanced life suffer from this disease than in England. It is remarked that in people who arrive in this Colony labouring under this complaint, it runs a much more rapid course than it is observed to do in colder climates.

In 1860, when regular statistics began to be collected on the disease, tuberculosis accounted for 146.6 deaths per 100,000 population in Victoria and ranked as the third most common cause of death among the non-Aboriginal population. For the rest of the century the disease maintained a high level of between 145.8 and 116.2 deaths per 100,000 of population (Government Statist 1904)

5.4 Tuberculosis among the Aboriginal populations - source material

5.4.1 Early contact and diffusion (Stage II)

Despite a long period of human occupation there is yet no evidence for tuberculosis in Australia before European settlement (Basedow 1932:16; Cumpston 1989: 276; Thomson 1991: 62). Webb (1984a, 1989, 1995) conducted an extensive palaeopathological study of Aboriginal skeletal collections covering most of Australia and found no evidence of the disease. Other palaeopathological studies done on more localised samples along the Murray River corridor in South Australia (Dowling 1990; Pretty & Kricun 1989; Prokopec 1979; Sandison 1973) have had the same result.

This should not be seen as conclusive evidence for the absence of the disease in Australia before 1788. Skeletal involvement in tuberculosis infection is uncommon. With rare exceptions, skeletal tuberculosis results from secondary infection by the bacilli spreading from either the lungs or the lymph nodes and just 5 to 7% of cases will show discernible bone lesions (Steinbock 1976: 175). This substantially reduces the probability of identifying tuberculosis infected individuals in skeletal collections. The probability is further reduced when one considers the sites of skeletal involvement and the preservation of the collections examined. The most frequent sites of infection are the lower vertebral

column, ribs and sternum, the proximal and distal articulations of the femur and the proximal articulations of the tibia (Lucas Powell 1992: 42; Steinbock 1976: 178). Other less involved sites of infection are the articulate regions of the shoulders, elbows, hands, ankles, and feet (Steinbock 1976: 178). The skull is rarely involved. Many of the skeletal remains examined in Australia for pathology consist of only the skull and/or incomplete post-cranial bones and many are fragmentary and poorly preserved (Webb 1989: 10-14; Dowling 1990: 12). It is not therefore surprising that palaeopathological examinations have revealed no evidence of tuberculosis in Aboriginal Australia before European colonization even if it had been present.

The first reference to tuberculosis among the Aboriginal people comes from a visiting French expedition. R.P. Lesson, the second surgeon on *La Cocquille* which visited the Sydney settlement from January 17th until March 20th 1824, noted that:

> These natives are susceptible to a large number of illnesses, which their miserable state makes them bear more readily than would the more robust European. Most have chronic catarrh; some women are consumptive... (cited in Royle 1973:950).

Although we cannot accept this observation at face value it is possible that some of the Aboriginal people (both male and female) seen by Lesson were suffering from tuberculosis. As discussed above the disease appears to have accompanied the First Fleet colonists in 1788 and became established in the colony at that time, or soon thereafter. The observation by Lesson was then some 36 years later, which is ample time for the disease to have spread from the Europeans into the Aboriginal populations.

The first record of tuberculosis becoming a major health problem among Aborigines is among those interned at Wybalenna settlement on Flinders Island in Bass

Strait (Plomley 1987: 917-947). In November 1831 the Tasmanian (Van Diemen's Land) government moved one hundred and fifty Tasmanians from the main island to temporary settlements on Flinders Island and later in 1833 the settlement of Wybalenna was established. The mortality of the Aboriginal people was high and by 1847 when the establishment was closed and the inmates were moved back to the main island, just forty-seven had survived (Plomley 1987: 172; Ryan 1996: 203).

Before 1835 the causes of deaths on the main settlement and in the transit camps went largely unrecorded. From October 1835 to February 1839, however, when G.A. Robinson was in charge of Wybalenna, causes of death were recorded and thirty-two autopsies were undertaken. After February 1839 the reporting of mortality again lapsed. During 1837 and 1838 the autopsy reports show at least thirteen, or 35% of all autopsy cases, involved extensive tuberculosis infection (Austin 1837; Walsh 1838; Plomley 1987: appendix II).

The number of deaths from tuberculosis in the settlement may have been higher. Surgeon Allen stated he had been informed that most of the old and infirm Aborigines had died of consumption and scrofula before he took up his position as medical officer of the station (Allen 1837). The records do not give the cause of these deaths, but in 1838 alone, the autopsy reports show that 9 (64.3%) deaths of the 14 recorded can be diagnosed as tuberculosis. After 1838 tuberculosis seemingly loses its prominence in the records and influenza, pneumonia, catarrh, and 'pleuritis' were recorded as the most common causes of death on the station (Plomley 1987: appendix II). This does not, however, indicate that tuberculosis was becoming less of a problem. Mistaken diagnosis of the cause of death and the underlying immuno-suppressant nature of chronic tuberculosis could well have predisposed many of the Aboriginal people to life

threatening infections of influenza and pneumonia that were then recorded as causes of death.

On the Australian mainland, for the first sixty years of European colonization, tuberculosis appears to have had little impact on the Aboriginal population as there are few references to it. Where it did occur, the morbidity was minor. With the instigation of Aboriginal Protectorates in Port Phillip in 1835 and South Australia in 1836, reports on the health status of Aborigines were sent by the Assistant Protectors from their various stations in country and urban regions. In many cases the these reports allude in general terms to severe and chronic diseases among the Aborigines. Few, however, make reference to consumption, phthisis, tabes, or scrofula, the usual terms for tubercular infection.

The disease is not mentioned as a major health problem among the large groups that regularly left their homelands and congregated on the fringes of the major settlements of Sydney, Melbourne, and Adelaide. One large group which collected during April 1839 on the banks of the Yarra River flowing through the settlement of Melbourne was described as being in a state of 'wretchedness and disease' by the Assistant Protector of Aborigines for that area (Sievewright 1839). Doctor P. E. Cussen was instructed by the Chief Protector of Aborigines to examine them and administer treatment. He reported that they were suffering from dysentery, typhus, syphilis, rheumatism, catarrh, and starvation (Cussen 1839a). He made no mention of any tuberculosis infections

The disease also appears to have been uncommon among the Aborigines living among the pastoral settlements of Victoria. In 1840, Watton, the medical officer of the Mount Rouse Aboriginal Station, listed one case out of 142 presenting to him for

treatment that could be ascribed to tubercular infection. This was 'a very old woman' who died while suffering from 'tabes' (Watton 1840). Of a further eighty-seven Aboriginal people treated by Watton at Mount Rouse during the last four months of 1842, just two (2.3%) were diagnosed as suffering from tuberculosis. One young female, aged eight years was diagnosed with 'tabes mesenterica' and another female six years of age was diagnosed with 'tabes' (Watton 1843). Doctors William Baylie and Neil Campbell regularly examined Aboriginal people in the north east and north west districts of Victoria between 1841 and 1845 while acting as medical officers for the Aboriginal Protectorate. Neither diagnosed any as suffering from consumption or phthisis (Baylie 1841, 1841-42; Campbell 1841, 1843, 1845) but Baylie (1841-42) described one case, a nine year old male, suffering from 'inflammation of the mesenteric glands' which could possibly be miliary infection

E.M. Curr, who compiled one of the most extensive accounts on Aborigines last century, recorded the absence of tuberculosis among the tribes of the Murray River in northern Victoria during the early period of European settlement:

> During the first eight years of my residence amongst several large tribes near Echuca, in 1841, I can recollect no instance of consumption; nor, though I have made inquires on the subject of a few old residents who were good observers, have I heard of more than one death of that disease at that period (Curr 1886: 227)

In South Australia the situation was similar. Edward Eyre spent a decade in close contact with Aboriginal people, during his residence in Australia, initially as a traveller and explorer, but in particular during three years from 1841 to 1844 he spent as Resident Magistrate and Protector of Aborigines at Moorundie, a police post and Aboriginal depot on the Murray River . During this time Eyre made regular journeys

along the Murray as far as the Darling River and had frequent contact with many large groups living along the river corridor. In regard to tuberculosis he records:

> Phthisis occasionally occurs ... Scrofula has been met with, but rarely (Eyre 1845: 379).

Tuberculosis reporting among Aboriginal populations began to increase in the second half of the nineteenth century when it became the major cause of death. William Thomas (1861-62), a Victorian medical practitioner, reported that 'eight-tenths' of the mortality he had observed among the Aboriginal people of the colony arose through pulmonary disorders, mainly pleurisy, pneumonia and consumption. Thomas, like most medical practitioners of that time, kept few or no health statistics on Aboriginal people he examined so his statement must be seen as an impression based on his experiences. Nevertheless, it suggests that tuberculosis was becoming a major cause of morbidity and mortality among the Aboriginal people. He related these deaths to the Aboriginal people's addiction to alcohol which, when coupled with infection, led, he believed, to their deaths within days of showing the first symptoms (Thomas 1861-62).

The Ngarrindjeri who had their homelands extending along the lower reaches of the Murray River and Coorong district of South Australia also became victims of tuberculosis. This group had been in contact with Europeans since 1803 when sealing enterprises began to be established on Kangaroo Island and the southern mainland coast of South Australia (Moore 1923; Summers 1986: 285). They were soon infected with syphilis and gonorrhoea spread by the sealers (Dowling 1990: 87-103; and Chapter 4) but their early experience with tuberculosis is unknown. After eighteen years of close association with the Ngarrindjeri, as well as other groups from further upstream on the

Murray, however, George Taplin (1876a) noted the importance of the disease amongst them:

> My observations have led me to the following view of the principal disease from which the natives suffer with is evidently tuberculosis in its different forms. I think that a large number of deaths arise from this cause. Of 50 deaths of Adults which occurred here [Point McLeay Settlement] between 1859 and 1869 twenty five were caused by tubercular consumption.

5.4.2 Settlement and acculturation (Stage III)

The extent of tuberculosis among the Aboriginal people in the latter half of the nineteenth century is best seen in the records from the Aboriginal settlements (Table 5.4 and Figure 5.2). It should be noted, however, that tuberculosis was not confined to Aboriginal people living on established settlements but affected those who were able to live away from European settlements. In many instances migration of Aboriginal people to and from the settlements would have facilitated the spread of the disease to those who lived most of their life on their traditional homeland.

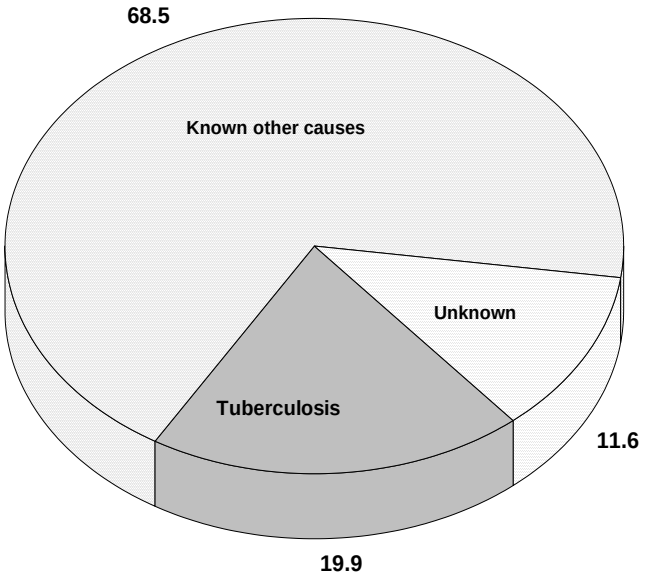
Table 5.4 Extent of tuberculosis as the recorded cause of death on Aboriginal settlements in the nineteenth century for which there are reliable data..

Station	Period	Cause of death TB	Other	Unknown	Total	% TB
Framlingham	1876-1883	4	14		18	22.2
Lake Condah	1876-1900	30	66	1	97	30.9
Lake Wellington	1876-1900	20	68	1	89	22.5
Lake Tyers	1876-1900	8	85		93	8.6
Coranderrk	1878-1900	22	49		71	31.0
Lake Hindmarsh	1876-1900	18	64		82	22.0
Point McLeay	1859-1900	60	226	94	380	15.8
Point Pearce	1880-1900	12	25	5	42	28.6
Total	**1859-1900**	**174**			**872**	**19.9**

Data have been taken from the registers of births, deaths and marriages compiled by each of the settlements during the latter half of the nineteenth century. Those causes of death recorded in the registers as 'consumption', 'phthisis', 'phthisis pulmonatis', 'tabes mesenterica', 'tubercular peritonitis', and 'tuberculosis', have been extracted. Table 5.4 lists recorded deaths from tuberculosis on eight Aboriginal settlements for varying periods and is compared with deaths from all other recorded causes.

Fig 5.2 combines the data from the settlement records and expresses the importance of tuberculosis as a cause of death among Aboriginal people living in institutions throughout Victoria and South Australia between 1859 and 1900.

Figure 5.2 Tuberculosis as a percentage of the recorded causes of death on Aboriginal settlements in Victoria and South Australia 1859 - 1900.



It can be seen from Table 5.4 and Figure 5.2 that tuberculosis was a major cause of mortality among Aboriginal people living on the settlements accounting for 19.9% of all the recorded deaths. The records from Point McLeay, however, contain a substantial number of deaths, (94 out of 380, or 24.7%) for which no cause was given. This may have occurred because the cause was not known by the recorder or that it was simply a failure to record the cause at the time of death. This has most probably distorted the data for both tuberculosis as well as all other causes of death on this settlement and for the combined total. In this case the extent of tuberculosis as a cause of death at 19.9% of all deaths should be seen as an approximate figure only. I feel it may be an underestimation because of the chronic nature of the disease making the sufferer more susceptible to secondary infections, particularly respiratory, whose symptoms would be more readily recognized by the recorders at time of death. In such cases tuberculosis would most likely have been the underlying cause of death. An indication that this figure may indeed be an underestimation comes from George Taplin (1876b), the superintendent

of Point McLeay settlement, who estimated that 50% of the deaths he observed among the Ngarrindjeri of the lower Murray River between 1859 and 1869 were of those suffering from tuberculosis. While this estimate should, of course, be taken with caution Taplin's observations do intimate that tuberculosis was inflicting an excessive level of mortality and by implication a high morbidity.

The prevalence of tuberculosis on Aboriginal settlements in South Australia and Victoria had regularly been a point of concern for those in charge (e.g. Taplin 1876b; Curr & LeSouef 1879). In 1878-79, however, it was causing high mortality among the Aborigines on the Victorian missions. In 1878 Reverend Hagenauer of Lake Wellington mission reported to the Board that the state of health of the residents had been poorer than usual and the doctor who attended them had given up on several who had been suffering from consumption (Hagenauer 1878). For the same period the superintendent of Lake Tyers Aboriginal station reported that the past season had been a very trying one and there had been more sickness during the year than at any other time (Bulmer 1879). The superintendent at the Government's own settlement, Coranderrk, referred to the morbidity and mortality caused by tuberculosis. There were approximately 80 Aboriginal people living on the station and twelve deaths were recorded in the two year period. Of these six were attributed to consumption, another one was attributed to 'softening of the brain' and an infant died from 'low fever'. In July he reported to the Board that:

> The health of the Aborigines is, I fear, much affected by the cold and damp climate; scarcely any of them are sound, as they are the subjects of phthisis, which is much promoted by their innate indifference to exposure to all kinds of weather (Strickland 1879)

The Aborigines Protection Board responded by sending two members to visit the stations at Lake Wellington and Lake Tyers. They reported that 'consumption or lung

disease' was widely prevalent with many Aborigines dying and consequently recommended to the government that the 'best medical talent procurable' be appointed to investigate the disease and if possible determine how it could be mitigated (Curr & LeSouef 1879).

The number of deaths from tuberculosis was lowest at Lake Tyers settlement (9% of total) (Table 5.4). Only one death from tuberculosis, and very little sickness was reported between 1876-85 (Bulmer 1880-81) on this settlement. After 1885 there were a further 7 deaths reported from tuberculosis. The other settlements were not so fortunate. At Lake Wellington, Hagenauer (1880-81) reported to the Board in 1880 that the state of health among the Aborigines seemed to be good 'although one could never rely on it'. His fears were realised because tuberculosis was to remain the principal cause of death on that settlement despite the care provided by two medical practitioners from the nearby town of Sale. The situation at the remaining stations in Victoria and South Australia was similar with tuberculosis consistently being a major contributor to mortality.

5.4.3 Clinical Features

There are a handful of surviving reports that give reliable descriptions of the clinical features of tuberculosis. During the period that Robinson was commandant of the Flinders Island Aboriginal settlement, several autopsies were conducted by the medical officers on the Aboriginal residents (Austin 1837; Walsh 1838). One report describing progressive miliary tuberculosis with tuberculous ascites and body emaciation in an adult male is given below: Table 5.5 summarises the remainder of the autopsies displaying tuberculosis involvement, Appendix A contains the autopsy reports in full.

27 February 1838

The body was quite emaciated prior to his dissolution and was in reality a living skeleton. On opening the cavity of the thorax the lungs presented chronic adhesions to the ribs, pericardium and sternum were for the greater part solid and hepatised, particularly the upper lobe of the left lung, which contained two or three large patches of a white caseous consistency. Both lungs were thickly interspersed with small hard white lumps of a tuberculated nature, although none of them had assumed a puriform state. The pericardium was much enlarged and contained about six ounces of fluid.

The liver was of extraordinary size, was hard and adhered to the diaphragm. On separating this connection a large ulcer was discovered on its upper surface of a dirty gangrenous appearance; it contained a large quantity of blood in a fluid state, was thickly covered with white hard small lumps similar to those found in the lungs, which pervaded its internal structure also. The whole peritoneal lining of the intestines exhibited the same tuberculated appearance but much larger in size and containing a thick yellowish purulent matter. The spleen was of natural size but hard and solid and on cutting into exhibited the same tuberculous formation with the aforesaid viscera. The peritoneal sack contained about four pints of water. The kidneys appeared healthy.

(Walsh, M., in Robinson n.d.; Plomley 1987: 933)

Table 5.5 Summary of autopsy reports 1837-1838 from Wybalenna Aboriginal settlement, Flinders Island, involving tuberculosis (Robinson n.d; Plomley 1987: 927-937)

Date	Sex	Age	Symptoms	Diagnosis*
20- 8-37	Female	Adult	Adhesion of lungs to surrounding membrane and ribs; extensive diffusion of tubercles externally and internally of both lungs; liver enlarged with small caseous foci; possible meningeal involvement	Miliary tuberculosis
30-12-37	Male	Adult	Adhesion of lungs to surrounding membrane; both lungs indurated; diffusion of tubercles on external and internal surface of lungs; extensive dissemination of tubercles to liver and intestines.	Miliary tuberculosis
12-5-38	Male	Adult	Extensive adhesion of lungs to surrounding membrane and ribs; both lungs indurated and dispersed with small tubercles; large purulent abscess on right lung; small intestines thickly coated with tubercles extending into peritoneum.	Miliary tuberculosis with possible cavitation of right lung.
2-6-38	Female	Adult	Right lung extensively adhered to pleura; cavity of thorax containing effusion of colourless serum	Pulmonary tuberculosis
21-6-38	Female	2	Adhesions of lungs; both lungs hepatised and interspersed externally and internally with small tubercles; left lung with three caseous cavitations; liver and spleen indurated and interspersed with tubercles; abscessing of pancreas.	Miliary tuberculosis; cavitating tuberculosis
2-7-38	Female	7	Adhesion of lungs to surrounding tissue; 2 to 3 small suppurated lesions on lungs; liver enlarged with extensive miliary involvement; spleen and pancreas indurated, abscessed and covered with small tubercles; intestines inflamed and interspersed with tubercles	Miliary tuberculosis
6-8-38	Male	Adult	Extensive hepatisation of lungs with a number of caseated cysts.	Cavitating tuberculosis
3-9-98	Male	3	Extensive lung adhesion to surrounding tissue; both lungs hepatised and dispersed with tubercles; left lung abscessed; spleen thickly dotted with tubercles; liver enlarged.	Miliary tuberculosis
29-10-38	Female	60	Adhesion of lungs to ribs, sternum, and surrounding soft tissue; both lungs extensively hepatised with tubercles externally and internally; large purulent cysts on both lungs; liver and spleen extensively disseminated with tubercles; cause of death given as phthisis.	Miliary tuberculosis, cavitating tuberculosis of lungs.
9-11-38	Female	Adult	Chronic adhesion of lungs to surrounding bone and soft tissue; right lung tuberculous; left lung severely necrotised and purulent; cause of death given as phthisis.	Pulmonary tuberculosis.

17-12-38	Female	Adult	Adhesion of lungs to surrounding tissue; both lungs indurated and covered externally and internally with small tubercles; internal tubercles larger; adhesion of liver, external tubercles; spleen enlarged and tuberculated externally and internally; extensive dissemination of tubercles along intestinal canal; cause of death given as phthisis.	Miliary tuberculosis

*Diagnoses taken from Plomley 1987: 927-937

In all cases but one the disease began as primary pulmonary tuberculosis, exhibiting adhesions and well-developed external and internal granuloma of the lungs. The exception was a seven year old female, whose lungs were described as 'healthy except [for] a few adhesions of the left of a chronic nature with two or three small specks on its posterior surface in an incipient state of suppuration' (Walsh 1838). Systemic dissemination of the disease by haematogenous and/or lymphatic distribution to the abdominal organs followed the primary infection in ten of the twelve cases. One of the cases where this may not have happened was that of an 'aged' female who was suffering concurrently from pneumonia and the another, a female, whose abdominal viscera were described as 'natural' although the kidneys were large, but appeared healthy (Walsh 1838). The organs infected were those favoured by miliary localisation, namely the liver and spleen. Renal involvement, was, however, uncommon. Abnormal kidneys were noted in only one case, that of a female whose kidneys were described as being 'quite flabby' (Walsh 1838). Miliary dissemination of tubercle bacilli involved the intestines in four cases and the pancreas in three.

Among the Ngarrindjeri in South Australia Taplin described the symptoms and the course of the disease:

> It also very often manifests itself in the form of tabes mesenterica about the third or fourth year or even later. I have even known of a very bad case occurring in a man of 25. This constitutional tendency often appears in the form of induration and ulceration of the glands of the neck. Where it comes out [like] this it is generally cured and the person becomes healthy afterwards. But its most usual and most fatal form is that of tubercular consumption. Any

> accident to the chest seems to lead to the deposition of tubercle (Taplin 1876a).

5.4.4 Aboriginal Treatment

Little has been recorded on how traditional Aboriginal medical practices perceived and coped with tuberculosis although they certainly were familiar with it. In Victoria the Assistant Protector William Thomas reported that:

> The blacks study much the colour of the spittle in those affected in the lungs, and know well its stages. When the patient begins to expectorate blood, much attention is paid him; should this increase, which is generally the case, the doctors hold a consultation, and when once a consultation is held the doctors will not allow the patient to take any more medicine from the whites. The invalid is laid on his back and held firm by three or four blacks, whilst the native doctor keeps continually pressing with his feet, and even jumping on his belly. I need scarcely state that this cruel practice brings on premature death (Thomas 1861-62).

Also in Victoria, Beveridge (1883) records Aboriginal people suffering from pulmonary tuberculosis undergoing heat treatment:

> The bath is constructed in a very similar manner to their cooking ovens, the only real difference being simply that the hole for the bath is made sufficiently large to contain the body of patient, and the glowing bottom of the hole is covered to the depth of a foot and a half with boughs which had previously been made damp, instead of a thin sprinkling of moist, grass, as is the case when cooking. When the hole has been sufficiently heated the ashes, etc., are scraped out and the damp green boughs nicely spread, upon which the patient is carefully placed. He is then covered all over by an opossum cloak, with the exception of his face, which is left bare. Then all over the cloak earth is spread of a thickness capable of retaining the steam without weighing too heavily upon the patient. To attain the former and obviate the latter the finest earth that can be procured is used - that is, in the absence of sand, sand in all cases being preferred when obtainable. During the progress of the bath that perspiration exudes from the face in great globules, and the hair becomes quite wet from the same cause.

5.5 Discussion

The first recorded indication of tuberculosis among Aboriginal people comes in 1824, after thirty-six years of European colonisation. Tuberculosis may not have been able to spread into the Aboriginal populations away from main European settlements in an extensive way during this period because of their mobility and infrequent contact with the colonists. Humans are the major reservoir of the disease and infection is primarily from human to human often requiring prolonged exposure to the bacilli before symptoms are noticed. It has been described as a 'family' disease (Meyer, E.A. 1974: 126) often acquired after long and close contact with an active case.

Although there was contact between Europeans and Aboriginal populations during this time (Blainey 1982c: 3-48, 1987:413-443: Clark 1981) it is doubtful whether it would have been sufficiently close to allow for the infection of many individuals. We have no record that it did. Other predisposing factors in the development of the disease in a community are a nutritionally poor diet, unsanitary living conditions, other acute and chronic respiratory infections, and the reduced efficiency of the immune system in individuals. While these factors were certainly present in the Aboriginal populations who lived in the vicinity of the first European settlements (see Chapter Six) and received the initial biological impact of colonisation, they were not factors affecting the majority of the Aboriginal population in Southeast Australia at this time.

It was not until 1837, at Wybalenna on Flinders Island, and after 1850 on mainland Southeast Australia, that tuberculosis began to be recorded as a major cause of death among Aboriginal populations. Its increase was due mainly to interacting political, social, and demographic factors affecting both the European and Aboriginal populations.

The major factor in the increased prevalence of tuberculosis began when the Aboriginal communities in Southeast Australia became increasingly more sedentary and centralised. Government and missionary policies encouraged and sometimes forced Aborigines to live in established settlements. In the early decades of the nineteenth century the influence of humanitarians reached its peak and the British Government was obliged to take steps to protect the rights and welfare of the indigenous peoples of its colonies. In Australia this caused a dilemma for the Colonial Governments. The 1830s and 1840s were periods of rapid European expansion across the southern part of the continent, often resulting in conflicts with Aboriginal people over land occupation. The official good intentions of the colonial governments and the concerns of the settlers were often in acute opposition (Foxcroft 1941: 22-28). The answer found to this dilemma was to establish government funded stations and missionary settlements where the Aboriginal people could be confined and controlled.

By the mid-1870s, 18 Aboriginal settlements had been established in Victoria and South Australia; most were administered and largely financed by missionary societies, and two in Victoria were exclusively under the control of the Aboriginal Protection Board, established in 1869 to provide for the protection and management of the Aboriginal natives of Victoria (Barwick 1971; Summers 1986: 304-307). A census of Aboriginal people both 'black' and 'mixed blood' taken in 1877 for the Aboriginal Protection Board revealed that almost half (46%) of the estimated Aboriginal population of 1,067 in Victoria were living on government stations and missions (BPA 1878; Barwick 1971). In 1876 South Australia enumerated an Aboriginal population of 3,953 (many of whom lived in the more arid regions of the colony) of whom 5.3% were living in the environs of Adelaide, on the four mission settlements, and in the settled counties

near to government aid distribution points (South Australian Government Gazette 1879: 797).

The Wybalenna Aboriginal settlement on Flinders Island was among the first of several of its kind in Australia established for the purpose of confining the Aboriginal people. The primary objective of the Government in establishing the settlement was the removal of the Aborigines from the mainland of Tasmania where their presence clashed with the interests of the European settlers. A secondary objective, which was to become commonplace in future settlements, was the Europeanization of the Aborigines once they were confined. It was the intention of the Government to replace the traditional culture and society of the Tasmanian Aborigines with that of a peasant society, as self-sufficient as could be managed by the European commandants. Descriptions of the living conditions endured by the Aboriginal inmates of the settlement (Ryan 1836) reveal they were required to live a style of life totally which was totally foreign to them, and which caused nutritional, medical, and psychological problems. They resided in cottages and huts, inadequately maintained and frequently cold, damp and poorly ventilated; they occupied a permanent settlement with inadequate and polluted water supply; far from their homelands and often living side by side with traditional enemies.

Their diet was varied. The colonial government made attempts to supply the settlement with rations but these were often late in arriving (Reynolds 1995: 175). Reynolds (1995: 175-6), however, notes that the Aborigines on the settlement were better provided for than the colony's other welfare recipients. The expenditure on government rations to the Aborigines was higher than that going to the colony's paupers, convicts, and destitute (Reynolds 1995: 175). In the early days of the settlement mutton birds (*Puffinus*

tenuirostris), which migrated annually to the island, were a valuable source of fresh meat for the Aborigines but these were seasonal (Ryan 1996: 186). Although there is no comparative study of the other nineteenth century Aboriginal settlements on the mainland, it seems likely that Wybalenna was the best equipped and most heavily funded of all. But given the degree of poverty on the mainland settlements this may be an overstatement (Reynolds 1995: 176). Other respiratory infections were common (Plomley 1987:915-937; and see Chapter Six). The lifestyle led on the island was highly conducive to the development of tuberculosis as a major cause of morbidity and mortality.

From the descriptions of the post-mortem examinations conducted by the settlement's medical officers between 1835 and 1838 (Austin 1837; Walsh 1838; Plomley 1987: appendix II), miliary dissemination developed in most cases of tuberculosis leading to death. The presence of small caseous tubercles, of effusion in the pleural, pericardial and peritoneal cavities, together with adhesions described in the reports is characteristic of this form of the condition. Extensive necrosis indicative of a long standing infection was not common. There is no description of brain or meningeal pathology. This may be because the skull was not opened. One might have expected evidence of tubercular meningitis, which may have been the immediate cause of death (von Lichtenberg 1989: 377-378) in some of the subjects examined and others who were not subjected to post-mortem examination. In these cases a reactivation of pre-existing tuberculosis seems unlikely. Chronic pulmonary tuberculosis leads to the formation of cavities in the lungs, which often become calcified. There is no reference to hard calcification in the autopsy reports, or to cavities with thickened walls, which one would have expected in cases of chronic pulmonary tuberculosis.

A second factor contributing to the increase of tuberculosis among the Aboriginal populations of mainland southeast Australia was the discovery of gold in 1851 in New South Wales. In the following ten years, 'the golden decade' (Moony 1987: 104), Victoria's population increased from 77,000 to 539,000 and that of New South Wales from 179,000 to 351,000 (Caldwell 1987). They comprised people from all strata of life, and all economic circumstances. They often became wanderers, crowding on to the gold fields while there were payable seams, then moving on to the next after each worked out. Most of the immigrants were in the 25 to 35 year age-group.

While it may have been a 'golden decade' for the economy of Victoria and New South Wales, it was a bleak period for those on the goldfields in regard to infectious diseases. Dysentery and typhoid fever were the major diseases on the gold fields, claiming many lives (Gandevia 1960: 757), but the demographic change brought with it acute forms of tuberculosis at a growing incidence. In the period from 1 July 1855 to 30 June 1856 phthisis and scrofula accounted for 8.1% of all deaths in the European population; by the end of 1856, the proportion of deaths from these diseases had more than doubled, accounting for 18.0% of the total deaths in Victoria (Registrar General 1861, 1856). Doctor W.L. Richardson (1869), who was a physician practising at the Ballarat District Hospital near to the largest gold fields in Australia during the gold rush period commented that:

> Tubercular pulmonary consumption presents frequently; it certainly has become more frequent within the memory of the writer.

A third factor following the gold rush was the arrival of convalescent tuberculosis sufferers to Australia. During the first half of the nineteenth century it was generally believed by the English medical profession that a long sea voyage in the clear

air of the oceans to a place with a mild and unchanging climate was of great therapeutic value to sufferers of pulmonary tuberculosis (Dubos & Dubos 1953: 26; Ruddock 1873: 93-100). This supposition was also partly based on the belief that vomiting as a result of sea-sickness helped the consumptive patient (Charlwood 1981:175). There was also little or no suspicion that tuberculosis was communicable (Johnston 1993: 1061-1062; Ruddock 1873: 26-44) and quite often the tubercular emigrants were not separated from other healthy passengers. Many, therefore, would have passed on their infection to others within the crowded confines of below-deck accommodation. Shipping companies catered for those sufferers of tuberculosis wishing to migrate to Australia, offering them special facilities on ships specially fitted out for convalescents. The Atlantic great circle route, passing well south of the Cape of Good Hope and then east at latitudes 40^{o} or 50^{o} across the Indian Ocean to Australia was especially recommended by medical practitioners and favoured by the sufferers (Dubos & Dubos 1953: 26-27; Proust 1991b: 8). Doctor S. Dugan Bird, who came to Australia himself suffering from pulmonary tuberculosis, was a great proponent of the custom and wrote a book encouraging suffers in Britain to emigrate (Bird 1863). He advised that the best time to leave England was in October and November, leaving the northern winter behind and to arrive in Melbourne, as the preferred destination, where the sufferer could soon look forward to complete recovery (*ibid*: 83-89).

The hypothesis behind this conviction was, however, unsupported by any scientific evidence. It arose from the cognizance that the disease was common in England, which had a large and growing population living in densely packed industrial cities and towns. Australia was known for its comparatively lower prevalence of tuberculosis, its smaller population, less industrial pollution, and warmer climate (Dubos & Dubos 1953: 26-27; Proust 1991b: 8). All these were thought by some medical authorities to be factors leading to the cure of the disease. The opposite opinion was also

held by other medical practitioners (Thomas 1870; Proust 1991c: 234) and as early as 1830 it was noted that the change to a warmer climate was not necessarily a guaranteed cure. Writing from Australia, McLeod observed:

> Consumption of the lung/Phthisis Pulmonalis is much more frequent than from the mildness of the climate might be expected, and more in advanced life suffer from this disease than in England. It is remarked that in people who arrive in this Colony labouring under this Complaint, it runs a much more rapid course, than it is observed to do, in colder climates (McLeod 1830).

Nevertheless, many 'consumptives' fled England choosing to emigrate to Australia to effect a last resort cure. By 1860 a growing number were making the trip; no doubt the decision was made more enticing after 1851 by the prospect of finding gold (Proust 1991b: 8). In a five and a half year period from 1865 to 1870, within the city of Melbourne and its environs, over two thousand deaths from phthisis were recorded; 86.3% of these deaths were those of immigrants. Not only did the influx of tuberculous immigrants increase the population of Victoria, they added substantially to the increased mortality from the disease in the colony, and were the vectors for the disease which then enabled it to become more firmly established in Australia and produce indigenous cases (Cleland 1938: 257). As a result the mortality rate from pulmonary tuberculosis in Victoria increased to 138 per 100,000 in 1880 from a rate of 115 per 100,000 population for the period between 1864 and 1871 (Proust 1991b: 10).

The change in lifestyle and living conditions of the Aboriginal people, the increasing European population, and the influx of infectious sufferers, were favourable to the spread of tuberculosis among the Aboriginal populations. A pathogenic process leading to active tuberculosis in individuals would have been further enhanced by poor host resistance to infection. Many of the Aboriginal people living on settlements and in pastoral regions had, for many years, been living in poor nutritional and contaminated

environments, suffering from a range of chronic and acute infections. Together with alcohol and tobacco addiction, the inexorable result in many would be an impaired immunological system resulting in less resistance to the pathogenic impact of tubercle bacilli infection.

Once established, tuberculosis became, and continued to be, the leading cause of death among Aboriginal people. Records of Aboriginal mortality from government and mission settlements in Victoria and South Australia between 1876 and 1900 show tuberculosis as the cause of 20% of all recorded deaths (Chapter 8). While this figure indicates tuberculosis as a major cause of death among the Aborigines it possibly understates the true impact of the disease on the Aboriginal populations and that the mortality rate of infected individuals was higher. Misdiagnosis of the symptoms presenting at time of death and secondary symptoms, particularly of respiratory disturbance, would disguise and underlying chronic state of tuberculosis. For example, many of the causes of death listed in the registers are given as 'lung disease', 'congestion of the lungs', or 'inflammation of the lungs'. These symptomatic states may have been misdiagnosed or may in fact be secondary conditions (pneumonia, bronchitis, influenza) in individuals suffering from chronic tuberculosis.

Another factor in the underestimation of the deaths from tuberculosis is that the Aboriginal people who lived permanently or temporarily off the settlements, or who died away from the settlements, are largely unrecorded. An unknown number of these latter cases would have had periods of exposure to other tuberculosis sufferers when they spent time living on the settlements (Taplin 1876b). Others, who knew the seriousness of their symptoms and were aware of approaching death, left the establishments in order to die in their own home lands. Their numbers remain unknown.

Taplin (1876a) considered that fifty per cent of Aboriginal deaths among the Ngarrindjeri of the Lower Murray River district of South Australia, between 1859 and 1869, were from tuberculosis. A similar situation was observed on settlements in Gippsland region of Victoria in 1879. After visiting Ramahyuck and Lake Tyers settlements in May of that year, Curr and Le Souef, members of the Aboriginal Protection Board, reported:

> We observed, however, with deep regret, that consumption, or lung disease, here as elsewhere, was very prevalent, and the natives are rapidly decreasing in numbers; few of those born on the stations seem likely to attain the age of thirty years. The disease is always, sooner or later, fatal, and it has been for many years a terrible scourge among the blacks, thousands having no doubt died from its effects in the last thirty years. The complaint was most likely engendered in the first instance by an entire change of life, consequent on coming into contact with the whites (Curr & Le Souef 1879).

The first of these factors resulted in a general increase in the European population and a subsequent increase in the prevalence and mortality from tuberculosis among them; the third resulted in changes in social and living conditions of the Aboriginal population leading to more exposure to the pathogen and lowered immunological protection.

5.6 Conclusion

Tuberculosis probably arrived in Australia in 1788 with the members of the First Fleet. Deaths from the disease were recorded among the colonists before the subsequent arrival of further fleets. It is uncertain, however, how soon after the arrival of Europeans the disease spread to the Aboriginal population.

Little is known about tuberculosis among Aborigines in the early years following the colonisation of Australia. The first medically identified cases of the disease were diagnosed as a result of autopsies on Aboriginal people interned at Wybalenna settlement on Flinders Island in 1837 and 1838. These cases displayed extensive miliary dissemination and were most likely the primary cause of death among the residents.

In the second half of the 19th century tuberculosis became well established and was a major cause of mortality among the Aboriginal communities of southeast Australia. Mortality records on Aboriginal settlements between 1859 and 1900 reveal 19.9% of all deaths attributable to tuberculosis in its several forms. A higher mortality from the disease should be expected due to misdiagnosis of symptoms at death and unrecorded Aboriginal deaths.

The change of lifestyle and living conditions predisposed the Aboriginal people of mainland southeast Australia to increasing infection from tuberculosis. Adding further to the increase of the disease was the rising European population and the increasing incidence of the disease among the European population.

CHAPTER SIX

Acute Respiratory Diseases

6.1 Introduction

This chapter is concerned with respiratory diseases during the early contact and diffusion stage (stage II) of the medical model. It focuses particularly on influenza and pneumonia, as the most common respiratory diseases that have at one time or another caused severe morbidity and/or mortality among indigenous populations. Other respiratory diseases such as bronchitis, pertussis (whooping cough), and the common cold are covered to a lesser degree. A further examination of acute respiratory diseases among Aboriginal communities during the settlement stage (stage III) is covered in Chapter Eight.

Although respiratory diseases were readily recognized as such by nineteenth century medical practitioners, diagnosis was often not specific. Several disorders were, however recognized. For example, influenza, pneumonia, and bronchitis were known to be separate but often related disorders recognized by their differing clinical signs and epidemiological circumstances. But in many cases, death or illness was ascribed in a general way to 'congestion of the lungs', 'inflammation of the lungs' or 'lung disease'. Given this ambiguity in diagnosis I have combined the numerous diseases and signs affecting the respiratory system that were seen among the Aboriginal populations under the ICD classification of diseases of the respiratory system. Because influenza and its companion pneumonia were common in the nineteenth century and often reported in the medical literature I have paid special attention to these diseases.

The most common respiratory infection carried from Europe to the New World and Pacific was influenza. Influenza may have been the first epidemic disease

causing high mortality to be introduced into the New World by European colonists. An outbreak of what was probably swine influenza emanating from the Spanish settlement of La Isabela on Santo Domingo in 1493-94 has been blamed for the disappearance of the indigenous populations of the Antilles (Guerra 1988: 305). Accounts of the outbreak describe an acute infectious disease that was extremely contagious, with a very short incubation period, affecting simultaneously a large population, and having the signs of a high fever, prostration, and inflicting excessive mortality (Guerra 1988:316). A Spanish observer recorded that 'so many Indians died that they could not be counted ... all through the land the Indians lay dead everywhere (cited in Stannard 1992: 69). Influenza epidemics and associated respiratory complications among the American Indians became common during New World colonial expansion. Such outbreaks were responsible for many deaths in the Southwest of North America (Dobyns 1983: 264; Thornton *et al.* 1992: 191; Upham 1992: 227); the Northeast (Cook 1973; Carlson *et al.* 1992: 149) the St. Lawrence Valley (Dobyns 1992: 218), and New Spain during the seventeenth century (Reff 1992: 268); on the Northwest coast in the early nineteenth century (Boyd 1992: 249); among the Chumash in the eighteenth and nineteenth century (Walker & Johnson 1992: 130-33); and among the Aleut of Alaska from the early days of contact (Thornton *et al.* 1992: 128). Following the first epidemic, a second outbreak in 1539 of what was possibly influenza spread among the northern Andes and into populations further inland (Cook, S.F. 1992: 211). Further epidemics of influenza and other introduced maladies caused severe population loss among the Inca (estimated to be around nine million) in the sixteenth century (Thornton *et al.* 1992: 87-91).

Epidemics of influenza and secondary pneumonia occurred in the Pacific Islands following European contact. Influenza was first reported in Tahiti in 1772 followed by Fiji in 1792, Samoa in 1830, the Cook Islands in 1837 and New Zealand in 1838 (Lange 1984: 325, 1988; Marshall 1993: 486; McArthur 1967). Severe epidemics

resulting in high mortality were recorded throughout the Pacific from the 1830s to the end of the nineteenth century and after (Marshall 1993: 486).

6.2 Biology of influenza and pneumonia

Influenza is one of the most common diseases that infect humans. It is a highly contagious and acute viral disease which has had a long history of association with humans and appears regularly throughout the world in epidemic forms (Stuart-Harris & Schild 1976:112; Crosby 1993b: 807). The clinical manifestation of symptoms differ. Several host factors such as age, immunological status (i.e. prior exposure to a similar viral subtype) and physiological state may influence the severity of the diseases syndrome.

In its mildest form infection by the virus may result in an asymptomatic condition or may produce mild symptoms such as fever or cold. More serious forms, often occurring among the very young and the aged, may cause an acute, prostrating febrile illness with a sudden onset of a range of symptoms including a sore throat, cough, coryza, chills, headache, weakness, and myalgia. Infection may be accompanied by undifferentiated acute respiratory complications, viral pneumonia, bronchitis and the common cold. Gastrointestinal manifestations presenting as nausea, vomiting, diarrhoea, prolonged fever, and convulsions may occur in children (Benenson 1990:224).

During epidemics the disease is often severe in very young or elderly victims, or in victims with pre-existing underlying conditions. The latter include victims with immunocompromised conditions caused by underlying chronic diseases such as tuberculosis, cardiovascular disorders, other respiratory disorders such as chronic bronchitis, asthma, emphysema, renal or metabolic disorders, anaemia, nutritional

deficiencies (starvation and alcoholism), age, a history of inflammation, inadequate therapy, and iatrogenic insult (Beneson 1990: 224; Crosby 1993b: 807; Stuart-Harris & Schild 1976: 97). Many of these medical states were common among post-contact Aboriginal populations in various degrees of chronicity last century.

The causative agents for influenza are three myxoviruses, influenza viruses A, B, and C. The most common, and the one that is the cause of the modern epidemics and pandemics, is the A virus which exists in a number of sub-types or strains which usually do not induce cross-immunity to each other. Type B has been associated with wide-spread epidemics and type C with sporadic and localised minor outbreaks (Beneson 1990: 224). The antigenic shift of the influenza A virus leads to the emergence of new sub-types that cause the regular outbreaks of new epidemics and pandemics of influenza. Airborne spread of the virus is the predominant mode of transmission but the virus can spread through direct contact, usually hands or fingers, by droplet dissemination. The spread of the disease often occurs in the winter months and people living in crowded and enclosed spaces are more susceptible to invasion by the virus (Beneson 1990: 224; Stuart-Harris & Schild 1976: 29-35).

6.3 Influenza and pneumonia in the European population of Southeast Australia

In the examination of historical documents, single cases of influenza are often difficult to resolve and differentiate from other respiratory complications like the common cold, particularly when the symptoms are mild. But when a sudden outbreak of the symptoms occurs within and/or between different communities it is almost always an influenza virus (Crosby 1993b: 807). In many such cases, influenza virus infection often leads to secondary, and more life threatening, complications of pneumonia. In documenting the history of influenza and pneumonia in Australia Cumpston (1989: 319-

20) showed that the incidence of pneumonia among the European population was closely associated with the epidemics of influenza. Secondary pneumonia complications involving inflammation and destruction of alveolar tissue are often the major cause of death during influenza epidemics.

There are no extant records referring to cases of influenza, an influenza-like disease, or pneumonia in the historical literature during the first thirty-two years of colonial settlement in Southeast Australia. The first recorded incident was an epidemic of influenza which occurred in New South Wales in 1820 and may have persisted within the colony until 1826 (Cumpston 1989:313). After the first recorded appearance, the nineteenth century epidemic events of influenza in Australia have in the main paralleled the pandemics of the northern hemisphere (Cumpston 1919: 2; 1989: 313). Table 6.1 shows the relationship between nineteenth century world pandemics of influenza and subsequent epidemics and localised events of excessive morbidity and mortality in Southeast Australia.

Table 6.1 Pandemics and periods of epidemic and unusually high morbidity from influenza in Australia (after Beveridge 1977; Crosby 1993b; Cumpston 1989; Lancaster 1953; Warburton 1973).

Pandemics	Australian Epidemics			
	N.S.W.	Vic.	S.A.	Tas.
1800-02				
	1820			
	1826			
1830-33				
1836-39	1836-38	1837-38	1838	1838
		1844-45	1844-45	
1847-48	1847-48	1847-48	1847-48	1847-48
1850-52	1852	1852		
1857-60	1860	1860	1860	1860
1873-75	1875	1875	1875	
	1885	1885	1885	1885
1889-91	1890-91	1890-91	1890-91	1890-91
	1894-95	1894-95	1894-95	
	1898-99	1898-99	1898-99	1898-99

Apart from localised epidemics in 1820 and 1826 in New South Wales, in Victoria and South Australia in 1845-46, and the non-appearance of the disease during the pandemic of 1830-33, the epidemics of influenza in Southeast Australia have paralleled that of the pandemics of the northern hemisphere (Cumpston 1919: 2; 1989: 313).

There are just a handful of extant references that document the first influenza epidemic in 1820 and these were written at various times after the event. The Deputy Inspector of Military Hospitals, Dr. D. McLeod, referred to the epidemic when he replied to a set of questions posed by the Royal College of Physicians:

> In July and August, 1820, an Epidemic Catarrh prevailed throughout the Colony; occasional cases of it had been appearing from the beginning of the Year, but most Families began at this time to suffer severely, there being hardly a House that had not cases of more or less danger. It proved chiefly fatal to infancy and old age; the symptoms of this Complaint were common Cold, Cough, pains in the forehead, sneezing, Soreness and Spasms in the Chest, copious discharge of mucous [*sic*] of various Colours from consistencies from the Lungs (McLeod 1830)

The epidemic was considered serious enough to be recorded in the 'Chronological Table of Remarkable Events' of the *Australian Almanac of Sydney*:

> An epidemical catarrh prevailed throughout the Colony about this period Many families suffered severely from this strange complaint. The symptoms of this disease were unaccountably varied. In many instances it appeared in a hectic cough, accompanied by violent spasms. In others, it produced, independent of the violent cough, a severe affection in the ear, with pus occasioned by internal inflammation. Some cases were visited with external eruptions, accompanied with extreme debility, and intense pain. The complaint was general. Many of the inhabitants were consigned to the grave in a few days, from the violence and fury of the attack; and some few, to this day, have the remains of this visitation still as a painful companion. Great numbers of the poor Aborigines fell victims to this novel and severe distemper (*Australian Almanac of Sydney* 1832: 245).

Writing some six or seven years after the event Cunningham made a brief reference to the epidemic and listed some of the more prominent symptoms:

> An epidemic influenza carried off a number of the old Europeans some years ago [1820], and also not a few of the aborigines, while many of our younger individuals occasionally feel the effects of it to this day. It appeared at the time, or immediately in the rear, of a hot northern wind, the symptoms being violent headaches, cough, sneezing, and inflamed eyes; with a quick pulse, and other general febrile concomitants (Cunningham 1827).

After the first epidemic subsided the infection may have persisted in the colony at a low level of morbidity. Although there is little evidence for this in the records of the colonists the second surgeon aboard the French ship *La Coquille,* which visited Port Jackson between January and March 1824, recorded that:

> The sailors of our crew, during this long stay presented only a numerous series of catarrhal complaints, ephemeral inflammatory fevers, colds in the head, illnesses neither interesting nor serious (Royle 1973: 951).

A second epidemic of influenza struck the colony in November of 1825 and lasted into the next year (Cumpston 1989: 313). There is little evidence of the morbidity and mortality upon the European populations from the subsequent epidemics up to 1890

other than to say that they were substantially higher than normal (Cumpston 1989: 314). The 1860 and 1885 epidemics were general throughout the Southeast Australian colonies and beyond (Table 6.1). The 1860 epidemic was called 'Fog Fever' in Victoria where 'the cases were very numerous, some very severe in nature - whole families being affected' (Jay cited in Cumpston 1989: 313).

The epidemic for which there are estimates of mortality is the 1890-91 event. Table 6.2 shows the mortality rate per 100,000 population in the Southeast colonies from influenza and pneumonia in 1890-91.

Table 6.2 Mortality rates from 1890-91 influenza epidemic (deaths from pneumonia included) (after Cumpston 1989:3i5-317).

Colony	Mortality Rate (per 100,000)
New South Wales	87
Victoria	98
South Australia	84
Tasmania	80

This epidemic was severe in terms of the mortality and morbidity it inflicted upon the European population. Curson (1985: 125) has estimated that in Sydney there were 100,000 cases of influenza making approximately 25 to 26% of the city's population infected by the disease. The mortality rate from influenza alone was 61 per 100,000 population for 1891.

6.4 Influenza and pneumonia among the Aboriginal populations - source material

Evidence on influenza among the Aboriginal people begins with the first epidemic of 1820 in New South Wales. This epidemic was a local event in New South Wales and was not connected with a pandemic. Observations made by a 'Medical Gentleman' in a letter to the Sydney Gazette describe the effects of the epidemic. Brief as it is, this description of the 1820 epidemic among the Aboriginal people reveals the severity of the symptoms and the Aboriginal response to the disease which was new to them. It is worth quoting at length. After giving an account of his own experience with the disease and that of his family he goes on to state that the Aboriginal populations of the interior of the colony suffered excessively from the same infection:

> which had produced a great mortality; and the *many young stout* [emphasis in the original] and robust people among them had become its victims, during the winter. In one severe instance a father, a very stout man, not exceeding forty years of age, with the mother and two daughters, and the infant of one of them, had all been carried off within the space of a month, leaving but one alive, a male about three years old, very distressed, until taken into protection by a European inhabitant of the settlement. Some cases ... appeared to ... have terminated in inflammation of the lungs; and that they had for the most part quitted the thinly wooded and more open tracts of the interior, and be taken themselves to the sea-coast, and brush and broken country, where were quantities of honey, and where they would undoubtedly remain until the return of summer. That these people should suffer intensely under every such contagion is not to be wondered at, when their state of privation from all comforts of life is considered; and that when prevented by bodily ailment from seeking their precarious means of sustenance, they are likely to become victims to famine, as unhappily from distemper ('Medical Gentleman' 1820).

There are no further records of this epidemic among the Aboriginal communities of New South Wales and we are left wondering about the severity of the diseases and its demographic and social effects upon them.

The second epidemic of influenza spread through the European population of the colony in 1826 (Cumpston 1989: 313). There is little known of its effects upon the European population and it went largely unrecorded among the Aboriginal population.

Influenza and whooping cough associated with an outbreak of measles was reported among Aboriginal populations in the Port Macquarie region in 1835 (Threlkeld 1835).

After these early reports the records on subsequent influenza epidemics and accounts of acute respiratory disease allow a better insight into the effects influenza and pneumonia in particular had among the Aboriginal people.

6.4.1 Influenza and acute respiratory disease among Tasmanian Aboriginal people 1831-67.

In May 1831 an epidemic of influenza, or a disease resembling influenza, broke out among Aboriginal people who were taken from the mainland of Tasmania to the small settlement on Gun Carriage Island. Gun Carriage Island is part of the Furneaux Group of islands in Bass Strait and was the last temporary settlement before the establishment of the major Aboriginal settlement on Flinders Island (Plomley 1987). On March 20 of that year, prior to their arrival on the island, a young female died. Archibald Maclachlan, a convict surgeon, submitted the following report on her death:

> Died at eleven o'clock am, Mary, a native child aged six years, having been afflicted with catarrh for some length of time, and from these last few days past, from fresh exposure to cold, she was seized with acute inflammation of the lungs which terminated her existence (Plomley 1966: 328).

By the end of March the epidemic had spread. Several were reported to be ill and by late April two more had died, an adult male and adult female. On May 2 Robinson reported that fifteen Aboriginal people at the settlement were in 'a sickly state' and later that month Maclachlan reported that 'catarrhal fever' was taking its course among all the Aboriginal people (Plomley 1966: 329; 1987: 35, 938). The population on Gun Carriage Island at this time was about fifty, including several women who had been

living with European sealers until the settlement was established. Maclachlan blamed the outbreak of the disease on the 'bleakness of this place and the want of proper habitation' (Plomley 1966: 456).

It is quite likely that this outbreak was associated with the 1830-33 world pandemic (Table 6.1) although there are no records of major outbreaks among the European population (Cumpston 1989: 313). If so, the virus may have been introduced by one of the sealing ships which frequented Bass Strait although by then large scale commercial sealing had ceased and only local sealers remained (Ryan 1996: 66-67). The epidemic ran its course among the Aboriginal people and by November when the settlement was eventually moved to Wybalenna on Flinders Island, 8 had died (Plomley 1987: 938).

Wybalenna functioned as a place of incarceration of the Tasmanian Aborigines from 1833 to 1847. The colonial government sought to solve the problem it had with the Aboriginal population (Reynolds 1995) by removing them from the main island of Tasmania and isolated them on Flinders Island. From the beginning of the settlement on the island the, commandant (Darling) impressed on the Aboriginal people the need to eat European food in the European manner. The change from their traditional diet was drastic. Their daily diet consisted largely of the colonial rations of salt meat and flour substituted by local food sources collected from the land or obtained by hunting. They had to learn new methods of cooking, boiling and baking, the new food with unfamiliar European utensils (Plomley 1987: 72-73). Dampers made from the flour, stews made from the salted meat with cabbage and turnips, and tea were the staple diet (Ryan 1996: 186) Arrangements for supplying the settlement were often unsatisfactory, with food running short, largely due to the irregular arrival of shipping at the island and the colonial administration (Plomley 1987: 79). The Aboriginal people often had to leave

the settlement and live on what ever they could obtain in the bush. Game animals quickly became scarce and on several occasions the annual arrival of mutton birds (*Puffinus tenuirostris*) to the island averted a more serious food crisis (Plomley 1987: 66, 79; Ryan 1996:186).

Water for the Wybalenna had to be obtained from brackish lagoons and brought to the settlement in containers. A fresh water creek near to the settlement ran only in the rainy season. No attempts were made by Robinson or the other commandants of the settlement to dam the creek to create an annual source of fresh water (Ryan 1996: 186). In 1835 the combined European and Aboriginal population was more than 150, putting a serious strain on water resources (Plomley 1987: 92-94).

Prior to Robinson's arrival at Wybalenna in 1835 the houses or huts offered to the Aborigines were neither warm nor comfortable (Plomley 1987: 91). In the early period of the settlements on Flinders Island huts were made from bush materials collected by the Aboriginal people. They were simple A-frame constructions expected to house six persons but were unsuitable as a permanent structure against the cold westerly prevailing winds of Bass Strait. Robinson attempted to improve the living conditions first by repositioning the doorways so the winds did not blow directly into the huts. Later he built brick houses with grass-thatched roofs in an L-shaped block of 20 dwellings which were to be the permanent accommodation for the Aboriginal residents until their removal from Flinders Island in 1847 (Plomley 1987: 92). Each dwelling had one room with two beds for four people (Ryan 1996: 191). The dwellings later became infested by vermin (Plomley 1966: 932; Ryan 1996: 186). In the last years of the settlement the living conditions experienced by the Aboriginal residents had seriously deteriorated largely due to neglect by Dr Jeanneret, commandant of the settlement from June 1842 to early 1844

and again in 1846, and a severe reduction in financial commitment by the colonial government (Ryan 1996: 196).

An indication of the poor conditions at Wybalenna comes from a petition written in February 1846. Signed by eight Aboriginal residents, it was forwarded to Queen Victoria through the Secretary of State for the Colonies:

> Dr Jeanneret kept plenty of pigs in our village which used to run into our houses and eat up our bread from the fires and take away our flour bags in their mouths also to break into our gardens and destroy our potatoes and cabbage...Our houses were let fall down and they were never cleaned but were covered with vermin and not white-washed. We were often without clothes except a very little one and Dr Jeanneret did not care to mind us when we were sick until we were very bad. Eleven of us died when he was here. He put many of us into jail for talking to him because we would not be his slaves. He kept from us our rations when he pleased and sometimes gave us bad rations of tea and tobacco (Plomley 1987: 148-9; Reynolds 1995: 7-9).

In 1847 Wybalenna was finally closed and the Aboriginal survivors moved to Oyster Cove on the main island of Tasmania.

Although the medical and mortality records at Wybalenna are inconsistent (Plomley 1987: 916) they reveal a high level of acute respiratory diseases among the residents. . Table 6.3 displays the number of deaths at Wybalenna and their recorded causes with special attention to acute respiratory diseases.

Acute respiratory diseases (n = 33) accounted for 26% of the total recorded deaths. This figure should, however, be seen as a minimum because of the high number of deaths for which no cause was given (n = 64). The 18 deaths recorded as pneumonia all occurred within the first 6 years of the settlement. This may indicate the decline of

this disease as a major cause of morbidity and mortality. Within the first 6 years of the settlement 111 or 87.4% deaths occurred causing a severe population loss from an estimated original population of 150 in 1833.

A number of individual cases of acute pneumonia leading to death were documented by Dr Allen in 1837 (Allen 1837; Plomley 1987: 923-924). Two have been selected in order to indicate the severity of symptoms suffered. Both show severe inflammation, adhesion and destruction of lung tissue, oedema in the alveolar spaces, and hepatization. A diagnosis of severe pneumococcal pneumonia is consistent with the recorded signs.

Table 6.3 Total recorded deaths and causes from acute respiratory diseases at Wybalenna Aboriginal settlement 1833 to 1847 (after Plomley 1987: 939-944).

Year	No. of deaths	Pneumonia	Influenza	Other ARD	Other Causes	No cause recorded
1833	31	-	-	-	-	31
1834	9	-	-	-	-	9
1835	14	11	-	-	3	-
1836	4	-	-	-	1	3
1837	29	3	-	-	8	18
1838	14	4	-	-	10	-
1839	8	-	8	-	-	-
1840	3	-	-	1	1	1
1841	3	-	-	1	-	2
1842	3	-	-	-	3	-
1843	1	-	-	-	1	-
1844	1	-	-	-	1	-
1845	2	-	-	-	2	-
1846	1	-	-	1	-	-
1847	4	-	-	4	-	-
Total	127	18	8	7	30	64

Case 1 is a 3 year old child seen by Allen and described as 'of spare habit of body with a large abdomen large joints small unshapely limbs head disproportionably large, with a pustular eruption over the scalp.

> 2nd day - Febrile symptoms increased with hurried respiration, and small quick pulse.
> 3rd day - Pulse small and quick, dry hot skin, tongue furred, cough accompanied with a glary [*sic*] expectoration which formed thick dark crusts on the lips, a wheezing noise in the chest as if the breath was passing through a fluid, a peculiar motion in the abdomen on inspiration and expiration as if respiration was carried on in and not in the chest in which latter [*sic*] there was very little motion. Comatose.
> 4th Day - Pulse small and quick. Thirst, will take nothing but water. Skin dry and shrivelled, difficult respiration, troublesome cough.
> 5th Day - Great difficulty of breathing; towards evening convulsions set in accompanied with dilation of the pupils; greatly emaciated; lingered until the morning of the seventh day when the respiration having become very laborious died.
> Post mortem examination - The lungs and pleura were highly inflamed, extensive adhesions had formed between them in several places, the parts were joined together by thick layers of coagulable lymph, there was a considerable quantity of a serous fluid in the cavity of the thorax. The air cells [*sic*] were filled with a glary serum. The diaphragm was also inflamed. The abdominal viscera were healthy.

Case 2 was a 26 year old male who initially complained of a pain behind the ear which impaired his hearing. Allen noted an accelerated pulse, disinclination to move about, a tendency to sleep, a clean tongue, and hot dry skin.

> 2nd day - Skin cool but dry, pulse not so full but rather quicker than yesterday, inclination to sleep continues, the pain has shifted half way down his neck, loss of appetite, tongue furred.
> 3rd day - Skin dry and hot, pulse small and quick, dry cough, difficult respiration; the pain has shifted from his neck, it is now down in his chest.
> 4th day - Much the same as yesterday, cough increased with expectoration of blood and mucus . Skin hot and rough, continually drinking cold water.
> 5th day - He seems very much relieved, skin moist, cough much easier, no blood expectorated, the tendency to sleep has gone off, pulse soft and nearly natural.

> 6th day - Improving fast. Pulses soft, skin moist, has been smoking this morning which is a sign of favourable termination.
> 7th day - Improving rapidly.
> 8th day - Still improving.
> 9th day - He got up this morning and walked about and during the course of the day he eat [*sic*] a large quantity of kangaroo meat and in the night was taken ill again.
> 10th day - Complains of a pain in his left side, cough. Pulse small and quick, skin dry and hot, considerable difficulty of breathing, prostration of strength.
> 11th day - The pain in his side still continues, febrile symptoms rather increased, cough accompanied with a glary expectoration, difficult respiration.
> 12th day - The difficulty of breathing much increased, he is now very much emaciated, feeble quick pulse, dilation of the pupils, coma.
> 13th day - Greatly agitated, extreme difficulty of breathing, hot dry shrivelled skin, lips and mouth thickly coated with the expectoration which flows from his mouth and nose, in the evening he became convulsed and towards morning he expired.
> Post mortem examination - On cutting into the peritoneum a quantity of serum escaped, the diaphragm and liver were inflamed and adhering firmly together, on attempting to remove the latter which was indurated an of a dark colour, it was easily torn. The spleen was indurated and thickly studded with strumous tubercles. In the thorax - the pleura and pericardium contained a large quantity of serum, the lungs and pleura were highly inflamed and several extensive adhesions had formed, a layer of coagulable lymph of considerable thickness in some places united the pleura which covers the lungs to the pleura that lines the chest. The lungs were of a dark colour quite inelastic and very much resembled indurated liver. The air cells were filled with lymph and pus - there was several small tubercles interspersed through the substance of the lungs, which contained a clot of hard dark blood, these tumours were surrounded with a number of small blood vessels containing dark blood, the heart was healthy.

Acute respiratory diseases were not confined to the epidemic events of influenza at Wybalenna. Prior to the first recorded pandemic/epidemic event of influenza in 1836-7, Allen (1837) reported that between 1834 and 1837, he had treated 230 cases of new, or aggravated pneumonia at the settlement in a population that averaged 140 individuals. Out of the 140 estimated on the settlement, forty of them had not displayed symptoms of pneumonia. That leaves, according to Allen, one hundred individuals who

presented 230 times with cases of pneumonia during the three year period leading up to the first influenza epidemic. A further indication of the prevalence of severe pneumonia comes from the records of death in 1835 (Plomley 1987: 941-941). Fourteen deaths were recorded in that year (Table 5.3), 11 (79%) of which were recorded as due to pneumonia. Of the remaining three deaths, two were recorded as 'anasurea' and one as phthisis. All this strongly suggests that pneumonia became an acute and chronic disease among the Aboriginal inhabitants of Wyballena soon after its establishment in 1831 and prior to the first recorded epidemic of influenza in the colony (Cumpston 1989; and Table 6.1).

Influenza caused serious morbidity and mortality at the Flinders Island settlement in 1837 during the course of the 1836-7 pandemic. On 16 January Robinson reported to the Colonial Secretary that a 'direful epidemic the influenza' had swept through the settlement. The Europeans were attacked by the virus but it was the Aboriginal inhabitants who, according to Robinson, were the most severely affected. There were 29 deaths recorded in the settlement for that year, an unusually high number. For most no details of death were given, but for three adult females pneumonia was listed as the cause of death, and four more died of the same causes in the following year (Plomley 1987: 941).

Surgeon Walsh performed an autopsy on one, an adult female who died in October 1837. The right lung revealed considerable empyema and the characteristic consolidation of pneumonia:

> On opening up the thorax the pleura was found considerably thickened and highly vascular... a large quantity of thick brownish fluid spurted out, which appeared to have occupied the greater part of the cavity... The right lung not only adhered firmly to the ribs but also to all the neighbouring parts; it was of a firm and solid consistency its cellular texture being quite obliterated... The adhesions of the left lung were not extensive and it was on the whole healthy in appearance (Walsh 1837).

A second wave occurred in 1839 when influenza again inflicted a severe morbidity and mortality among the Wybalenna Aboriginal population (Table 6.3). A week before his final departure from the settlement Superintendent Robinson wrote of the epidemic:

> I regret exceedingly to state that the influenza has already commenced its ravages among the aborigines supposed to have been communicated by the government vessels recently at this station. More than two-thirds of the natives are now affected, several of whom are not expected to recover. The aged and infants it is thought will suffer most although the worst cases at present are among those who are the most hale and robust. This epidemic has not confined its attacks to the natives, many of the whites being seriously indisposed, though I do not anticipate any fatal results as regards them (Plomley 1987: 785).

The two government vessels referred to by Robinson were the *Vansittart* and the *Eliza*. Both vessels had previously been to Hobart where an epidemic of influenza had been spreading among the European population for some months (Plomley 1987: 785). A second source of introduction may have been Robinson himself. He had returned from Sydney in January 1839 immediately prior to the Wybalenna epidemic suffering from influenza (Ryan 1996: 193).

George Robinson jnr., the son of the Commandant of the Flinders Island settlement, began to record the epidemic among the Aboriginal people on the day his father left to take up the position of Chief Protector of Aborigines on the mainland at Port Phillip. The following are extracts from his journal (Robinson jnr. 1839). The first entry begins on February 24 1839:

> The Commandant left the settlement about mid day to proceed to Port Phillip with several of the Aborigines, and as he proposed walking down to the Old Settlement Mr Clark with myself accompanied him part of the way thither. During our walk we met with several of the natives belonging to the settlement proceeding to some of the others who were staying in the "bush" with provision as we were informed they were in a bad state of health from the influenza which had not long visited the settlement.

> 25 February...
> The influenza to day shown itself to be very general amongst the natives who had all or most of them encamped themselves in the "bushes" environing the settlement.... Several of them were violently attacked with the epidemic.
>
> 27 February...
> The influenza was spreading itself to a still more alarming extent among them. The boat arrived near mid-night during a heavy gale of wind with the sick people from Long Point.
>
> 28 February...
> The native woman Semiramis died about break o'day. The rest of the natives returned home from their "breakwinds" this morning, those most affected with the influenza being brought in the truck by the others.
>
> 1 March...
> On proceeding as usual to the square early in the morning I was greatly surprised to hear that two of the natives, Phillip and George Robinson, had died both about one time during the night.... A native child belonging to Sarah died this afternoon. One of the native men "Billy" died in the early part of the night.
>
> 2 March...
> The natives still dangerously ill.
>
> 3 March...
> About midday "Sabina" died and in the evening "Jane". Both the females had been for several days in great suffering from the disease.
>
> 4 March...
> Interred at the usual hour, viz four o'clock pm, two of the deceased natives Sabina and "little Billy", and as rather a remarkable occurrence one of the sick natives Queen Adelaide breathed her last as we were placing the corpses in the coffins.

By March 6 Robinson jnr. was infected with influenza and soon after he records the disease striking several members of his family, the staff, and convict labourers of the settlement. The last reference to the disease in his journal is on March 14 when Robinson jnr. records that he was still suffering. How long it lingered among the Aboriginal population is unrecorded but by March 9 Robinson jnr. states that some of the males had began to recover. According to his journal and the register of births and deaths for the settlement eight of the Aboriginal people (3 males, 4 females, and a child) died of influenza during the epidemic (Plomley 1987: 942). The real total is almost certainly

much higher as many of the Aboriginal people had no faith in the efficacy of European medicines, and to escape the sickness, left the major settlement to live in the surrounding bush during the period of the epidemic. Their suffering was probably made greater by Robinson jnr. and his staff withdrawing rations, in particular freshly killed mutton, from the Aboriginal people during their illness:

> In consequence of the general prevalence of the present epidemic among the natives it becomes necessary to restrict them from the use of animal food for some time. I have therefore to direct that there be no issue of fresh meat made to them till further notice, but in lieu of which let them have an additional ration of the tea and sugar for dinner (cited in Plomley 1987: 787).

In August 1847 Wybalenna was abandoned. Two weeks before they left the settlement the influenza pandemic of 1847-48 reached them. It lasted ten or twelve days on the settlement and there was one more death reported as a result of the disease (Plomley 1987: 162). The remaining Aboriginal people, now numbering 47, (original population in 1831 was 150) were transported back to the Tasmanian mainland to the Oyster Cove settlement, an abandoned penal station (Ryan 1996: 205), located at the northern end of D'Entrecasteaux Channel (Figure 6.2).

Living conditions were worse at Oyster Cove than they had been at Wybalenna (Ryan 1996: 205-212). The station was built on the drainage plains of the Great Oyster Bay and Little Oyster Bay rivers. The buildings were made of wood which offered little protection from the cold southerly winds. The floors were mostly damp and often flooded by excess water draining from the rivers. The station had originally been built as a probation station and in 1843 used as a penal settlement but by 1847 it had been abandoned because it did not meet convict health standards (Ryan 1996: 205). James Bonwick described the station in 1859 as

> ...a miserable collection of huts and outbuildings ... profoundly dirty and swarming with fleas ... in a ruinous condition, roofs not waterproof, windows broken and furniture gone (Bonwick 1870: 276-85).

At the Oyster Cove settlement the Aboriginal population continued to suffer a high level of morbidity principally from acute and chronic respiratory infections. Mortality was persistently reducing their numbers until by the end of 1861 just two males and six women remained. There was scant concern shown by the Tasmanian government towards the Aboriginal people and little commitment was given to basic health care (Ryan 1996: 205-214). While the Flinders Island settlement was functioning there had always been a medical practitioner on staff to provide for the needs of the Aboriginal people, although in the majority of cases he would have been of little use to them. At Oyster Cove there was no resident doctor despite the well known poor state of health of the Aboriginal residents (Plomley 1987: 186-7). William Smith, a surgeon from Kingston, made periodic visits to the settlement between 1858 and 1869, summoned by the superintendent when emergency cases occurred; and more regular visits were made when outbreaks of influenza occurred on the settlement, particularly during the winter months.

During these visits Smith recorded his diagnoses and recommended treatments of the patients he examined in the settlement's visiting officers' book (Smith 1858-69). They appear in a chronological sequence according to the date of each of his visits to the settlement but when separated and rearranged into a sequence for each of the Aboriginal patients they reveal a progression of symptoms for each of the individuals Smith examined.

Influenza and pneumonia were common diseases throughout the period of Smith's visits. In the winter of 1860 Smith reported an epidemic of influenza on the

settlement with at least 8 of the 15 Aboriginal residents affected as well as the European superintendent and his family. During the period of the epidemic from July to September two females and two males died from symptoms suggestive of an infection of influenza (Smith 1858-69; Plomley 1987: 945). This epidemic was most likely part of the 1857-60 world pandemic which was recorded during 1860 all the colonies of Southeast Australia (Table 6.1).

I will use two individual cases to depict the recurring morbidity of these diseases at Oyster Cove. Both cases are adult Aboriginal women whose symptoms were recorded by Smith over a period of three years in the first case, and seven years in the second. The previous medical histories of both cases are unknown but both women had come to the Oyster Cove settlement from Flinders Island in 1847 where influenza, pneumonia, bronchitis, and tuberculosis were major health problems (see above). It is quite likely that both women had a long history of tobacco smoking and alcohol consumption. Pre-existing chronic bronchitis and emphysema may also have been present in both cases.

The first case is an elderly female, Sophie (Dray.dur.ic), c60 to 70 years of age. In 1829-30 she was living on Bruny island approximately 50 Km east of Hobart. She accompanied George Robinson on his first expedition around the island and was later sent to Flinders Island in September 1833 (Plomley 1987: 798; Ryan 1996: 124, 131). Smith records:

> [23 Sep 1858]
> Suffering from flu with swelling of the glands of the neck.
> [25 Sep 1858]
> Still some swelling of the glands of the neck but in other respects better.
> [27 Sep 1858]
> Appears much better.
> [27 Aug 1860]

> ... affected by the influenza [localised epidemic]. [She] appears to me to be in a depressed state and the physical powers lower than natural under the circumstances. I have recommended beer daily with beef tea and mutton broth and linseed tea as often as [she] can be induced to take it.
> [29 Aug 1860]
> Sophie not so well, her cough is very troublesome and like many others, the phlegm appears to accumulate in the air passages. The bowels are much constipated. I have recommended Croton oil liniment to rub into the chest.
> [1 Sep 1860]
> Sophie is still complaining of her cough and has some pain in the epigastrium. The conjunctiva of the eyes have a very yellow tinge.
> [5 Sep 1860]
> Sophie appeared somewhat better but still complained of cough, the Croton Oil liniment has produced rather more eruption on the chest.
> [21 Aug 1861]
> ... Sophie suffering from debility.
> [25 Aug 1861]
> Sophie has been poorly for several days and is apparently suffering from Bronchitis and general disability being upwards of seventy years old. I have recommended Brandy and Beer to be given at frequent intervals with as much Beef Tea as she can be induced to take.
> [27 Aug 1861]
> Sophie is sitting out having an Airing in the sun which she seems to enjoy much. Her breathing is still bad and there appears to be much mucus accumulated in the air passage. She does not take to much nourishment as she aught.
> [29 Aug 1861]
> Sophie died early this morning. Probably from exhaustion and the accumulation of mucus on the air passages (Smith 1858-69).

The second case is an adult woman, Wapperty (or Wobberrertee), c1797-1867. She was probably a Pyemmairrener born on the Northeast coast of Tasmania. Little is known of her early life. As an adult she was abducted by sealers and lived on the Bass Strait islands before being moved to the Flinders Island settlement in 1844 and thence to Oyster Cove in 1847 (Horton 1994 vol. II: 1149; Plomley 1987: 833; Ryan 1996: 214):

> [16 Jul 1860]
> Wapperty not so well. Complaining of pain in the chest with cold chills.
> [18 Jul 1860]
> Wapperty somewhat better and is apparently free from pain.
> [21 Jul 1860]
> Wapperty more lively.
> [14 Jul 1863]

Wapperty is suffering from influenza with pain on the left side attended with some cough. I ordered a mustard plaister [*sic*] and mixture with taitan emetic and compound camphor mixture.
[17 Jul 1863]
Wapperty is complaining of pain in the right side with difficult breathing and symptoms of Bronchitis, to continue the mixture and have the taitan emetic ointment rubbed into her chest and a blister on her right side.
[19 Jul 1863]
... the pain in the right side has left her, the cough somewhat better but the tongue coated with thick brown fur. Pulse not so strong, her appetite is not ample. To take Gin in linseed tea or milk if it be procured.
[21 Jul 1863]
... suffering symptoms of Bronchitis, in other respects better, the brown fur has left the tongue the bowels are acted upon, she does not sleep very well. To have Dovers Powder at bedtime in place of Gin, to have bottled porter with egg and nutmeg.
[23 Jul 1863]
Wapperty is still suffering from Bronchitis. The bowels are regular, appetite pretty good, breathing somewhat difficult. To continue the mixture and let her have rum in linseed tea.
[25 Jul 1865]
Wapperty is in many respects better but the cough is still troublesome. The Tartar Emilie has brought out a slight eruption on the chest, her appetite is pretty good.
[27 Jul 1865]
Wapperty appears greatly improved and the breathing seems natural. Her appetite is very good. She is allowed beer daily which is recommended to continue.
[22 Jul 1865]
Wapperty is complaining of pain in the stomach.
[29 Jul 1865]
... complains of cough.
[24 Jun 1867]
[Wapperty sent to hospital suffering from dysentery]
[29 Jul 1867]
[Wapperty recovered]
[8 Aug 1867]
Wapperty suffering from Catarrh and debility, respiration difficult.
[9 Aug 1867]
Wapperty improved.
[11 Aug 1867]
Wapperty does not appear well, pulse weak and pain.
[12 Aug 1867]
[Wapperty died]

During the periods leading up to their death both women suffered repeatedly from acute respiratory tract infections. No post mortem examinations were recorded for these women but the cause of death given for Sophie in the settlement's records was

'congestion of the lungs', and for Wapperty 'catarrh debility' (Plomley 1987: 945). While the official records giving causes of death among Aboriginal residents on settlements can be vague (Chapter 8) there appears little doubt that the deaths of these two individuals are closely related to the respiratory disorders they had.

6.4.2 Influenza among the Aboriginal population of Port Phillip, 1839

The 1836-39 influenza world pandemic reached the mainland Aboriginal population in 1839. In that year the disease caused serious morbidity and mortality among the European and Aboriginal population of Victoria. The first signs of the disease began to be noted early in the year and by July and August it reached a peak prevailing throughout the European population of the colony. In a report to the Royal College of Physicians, the Deputy Inspector of Hospitals stated that hardly a household had escaped the disease:

> It proved chiefly fatal to infancy and old age; the symptoms of this Complaint were common Cold, Cough, pains in the forehead, sneezing, Soreness and Spasms in the Chest, copious discharge of numerous Colours and various consistencies from the Lungs (McLeod 1830: 375).

The Sub-Protector of Aborigines, William Thomas, had just arrived in Melbourne and one of his first encounters with Australian Aboriginal people was with the Boonwurrung and Woiworung on 17th and 18th of January. He noted in his journal:

> Saw several blacks. The inhabitants of Melbourne say that they are about returning to the settlement, having left it on account of the influenza that prevailed. The natives considered that the whites brought the disorder (Thomas 1839a).

Some four weeks later Thomas noted in his journal that a family group of four, all suffering from influenza, were the first to come into the settlement for aid (Thomas 1839).

During February and March 1839 much larger numbers began to arrive in Melbourne. They came from the outlying regions to the north, south east, and south west. The reason for their going to the European settlement was seemingly to meet the newly appointed Chief Protector of Aborigines, George Robinson (Robinson 1839a). Robinson arrived in Melbourne by the government cutter *Vansittart* on 27th February and by this time four hundred Aboriginal people had gathered in a large encampment on the Yarra River opposite the main European settlement. On the very day of his arrival Robinson reports he was suddenly attacked with influenza symptoms and was so severely incapacitated that he was unable to perform his duties for several days (Robinson 1839b).

By the last week in March 1839, five hundred Aboriginal people had gathered in Melbourne to meet Chief Protector Robinson and his assistants (Robinson 1839b). It soon became apparent that many were suffering from influenza. Sub-Protector Thomas reported in his journal that he, in company with Chief Protector Robinson, visited the Aboriginal encampment on 5th May and found a scene 'truly appalling' (Thomas 1839a). There was little that Robinson could do. The Protectorate had limited financial reserves and no medicinal resources of its own to dispense among the Aboriginal communities. Repeated requests by Robinson for extra financial support were more often than not denied by Governor Gipps. The protectors could do no more than distribute just four blankets, which they took from their own beds, among the Aboriginal people who were sick.

On his return to the European settlement Robinson immediately instructed Dr. P. Cussen, the assistant surgeon for the colony, to go to the encampment and give what aid he could (Cussen 1839a). Cussen visited the encampment the next day and witnessed what he called 'a most distressing scene of disease, destitution and misery' (Cussen 1839a). According to his report he found the Aboriginal people displaying the symptoms of 'dysentery', accompanied with 'typhus fever of the worst description', 'rheumatism', and in many the symptoms of chronic syphilis. The prevailing influenza epidemic which Cussen referred to as 'acute catarrh' had already had a serious effect on the group gathered on the banks of the Yarra River with six of them having died within the four days preceding Cussen's visit; and many more were expected to die within the next days (Cussen 1839a).

The *Port Phillip Gazette* (1839) expressed its serious concern, not particularly about the well-being of Aboriginal people, but of the threat of influenza being further disseminated into the European population. It warned its readers that it was well known that influenza was epidemic and the Aboriginal people should be removed as quickly as possible. It further advised:

> ... all mothers to be careful of allowing their children to be taken in the direction of the native camps, the slightest attack of the influenza is sufficient to carry off an infant in a few hours (*Port Phillip Gazette* 1839)

Across Port Philip Bay the situation was similar among the Wathaurong (Addis 1839). In August Sub-Protector Sievwright requested Assistant Colonial Surgeon for the Geelong district, Dr. J. Clerk, to attend an individual who was suffering severely from influenza with secondary lung complications. Clerk's response succinctly reveals the plight of the Wathaurong in particular, and the Aboriginal population of the Port

Phillip colony in general, as well as the inadequacies of the Protectorate, and helplessness of the medical establishment to render assistance:

> In compliance with your [Sievwright's] request of yesterday, I visited the native black man and found him dangerously ill with inflammation of the pleura occasioned by exposure to the cold.
>
> I bled and prescribed for him accordingly but fear that medicine can produce little effect on him or any of the Aborigines whilst they are suffered to continue in their present exposed, helpless, and miserable state, destitute (when sickness attacks them) both of shelter and clothing and other necessaries for their comforts. It cannot under these circumstances be supposed that medicine can do much good.... Several to my knowledge this season have been carried off by inflammation of the chest (Clerk 1839).

By September the epidemic had began to subside among the European population of Melbourne but was still prevailing among the Aboriginal communities. Assistant Protector Thomas (1839b) reported to his superior in September that many Woiworung living along the Yarra River were 'labouring under affections of the chest'.

The number of Aboriginal people at Melbourne began to fall at the end of April when groups started returning to their homelands. By October 1839 most of the visitors had left Melbourne and there were less than fifty remaining; the majority of these were reported to be from the local region (Thomas 1839b).

6.4.3 Influenza and acute respiratory disease among Victorian Aboriginal populations 1845- 90.

An epidemic of influenza, restricted to the colonies of Victoria (Port Phillip) and South Australia, occurred between 1844-45. Although its origin is unclear, it was likely to have been introduced by ship and begun as an infection among the European populations of the two colonies reaching a level of excessive morbidity towards the end

of 1845 (Cumpston 1989:313). Robinson (1845) reported that few families living in Melbourne escaped infection, and out of a population of 12,000 at least two thirds had suffered attacks of influenza.

The Aboriginal people, according to Robinson, were the greatest sufferers, especially those living in the interior of the colony. At the Goulburn River Aboriginal station morbidity was high among the Taungurong with 'upwards of one hundred' reported to be affected and at least one death occurring for 1845 (Robinson 1845). A similar situation occurred among the Gunditjmara at the Mount Rouse Aboriginal station in the west of the colony. During the last three months of 1845 sixty-five individuals were treated most of whom presented with 'influenza and diarrhoea with occasional dysenteric symptoms' (Robinson 1845). No deaths were recorded among those living on the station.

Cumpston (1989: 313) documents the next pandemic/epidemic of influenza beginning in November 1851. It prevailed extensively throughout Victoria during the following three months and continued to exist in other settlements of the continent throughout 1852 and into 1853. The epidemic coincided with the beginning of the gold rush period which attracted thousands of seekers from Europe, Asia, and America. As stated previously the population of Victoria increased three-fold and that of New South Wales doubled during the ten year period between 1851 and 1861 (Molony 1987: 104). Few records of the effects of this epidemic upon the Aboriginal population have survived. The source of much of the early information on the Aboriginal people of Victoria had ceased when the Aboriginal Protectorate was abolished at the end of 1849. We can, however, assume with reasonable confidence that the morbidity was severe among Aboriginal people. The great influx of gold seekers to the newly established gold fields and with it the pervasion of legal and illegal liquor outlets made it easier for Aboriginal

people to obtain alcohol, much of which was of dubious quality. Thomas (1852), the former Assistant Protector, wrote that:

> The present condition of the Aborigines have no way improved but lamentably deteriorated, the discovery of Gold has greatly affected their moral condition, at all events those who locate these colonies or make transient visits to them...the consequence is that their frames are enervated in the absence of regular exercise and their blood corrupted through continued dissipation, so much so, that when seized with a violent cold inflammation follows so rapid [*sic*] that it is impossible in most cases to save them; the old and middle aged do not die in any proportion to the young, who, do not live out half of their days.

In 1861, following another pandemic/epidemic of influenza Thomas (1861-62) reported to the Aboriginal Protection Board on the mortality from respiratory disorders:

> I may state that eight-tenths of the mortality among the aborigines of Victoria arises through intemperance, bringing on pulmonary disorders, pleurisy, pneumonia, disorders of the chest, consumption, & c., which carries them off so speedily that the ablest medical treatment when available seldom save them. I may safely state, that when their respiratory organs are once affected, recovery becomes hopeless.

The second half of the nineteenth century saw much of the Aboriginal people of Southeast Australia centralised on mission and government settlements. Acute respiratory disorders were regularly reported on all these settlements (Appendix B). A discussion on the respiratory diseases among the Aboriginal communities on these settlements between 1876 and 1900 is contained in Chapter Eight which covers Stage III of the medical model (Table 2.2). It is, however, worthwhile giving a brief outline of respiratory diseases in the decade leading up to the settlement stage of the model.

Medical reports from the rural settlements of Coranderrk and Lake Condah reveal respiratory and possible respiratory disorders as recurring causes of morbidity. Dr.

L. Gibson made regular and special visits to the Government settlement of Coranderrk between 1865 and 1875 and reported his diagnoses and treatments to the Aboriginal Protection Board (Gibson 1869-75). Over the eleven-year period of his visits, Gibson's medical reports record periods of excessive morbidity from a possible outbreak of influenza in 1867; whooping cough (pertussis) in 1868; and influenza in 1873 (Table 6.4). The periods of high influenza morbidity were most likely localised outbreaks as they do not parallel the sequence of pandemics/epidemics of Southeast Australia (Table 6.1).

Gibson diagnosed the epidemic in April and May 1867 as 'intermittent fever', a common medical term of the day often given to the febrile symptoms of malaria (Farr 1854). It is unlikely, however, that this was the disease causing the symptoms. Nineteenth century cases of malaria among Aboriginal populations have been restricted to the north coast of Australia and there is no evidence for appearance of the disease in Southeast Australia (Basedow 1932: 10; Cleland 1928; Cumpston 1989: 330).

Table 6.4 Periods of excessive morbidity from respiratory and possible respiratory disease on Coranderrk Aboriginal settlement between 1865 & 1875 (Gibson 1869-75).

Date of Visit	No. of Inmates	No. with Respiratory Disorders	Percent	Remarks
1867				
21 Apr.	105	7	6.7	'Intermittent fever'. Several others recovering.
14 May	-	16	-	Many leaving due to epidemic.
8 Aug.	78	5	6.4	'Influenza'.
1868				
5 Dec.	74	-	-	Whooping-cough attacking all children & most adults.
29 Dec.	65	-	-	Whooping-cough still present but incidence abating.
1873				
16 Jun.	125	11	8.8	'Influenza'.
26 Jun.	129	13	10.1	Nine children & four adults infected.

The term 'intermittent fever' together with 'fog fever' were occasionally used as euphemisms for the various nineteenth century epidemics of influenza (Cumpston 1989: 331). By his November (1867) visit the symptoms had ceased and Gibson described the general state of health of the settlement as 'good' with just three cases of illness out of a population of seventy-six. The symptoms presented in both Aboriginal males and females between two years of age to adults, and the superintendent of the settlement and his children. In May of that year Gibson administered treatment to 16 (c.12%) of the residents but many others had left the settlement to escape the epidemic. Against these periods of high morbidity was a backdrop of periodic influenza cases of lower incidence and severity occurring mainly in the colder months of the year.

Morbidity from respiratory diseases at Lake Condah settlement during the years 1886 to 1890 was higher than previous years. The superintendent of the settlement,

J.H. Stahle reported in 1886 an epidemic of whooping-cough followed two years later by a severe attack of influenza in the spring and autumn. Initially the epidemic caused only the confinement of people to their homes but in 1889-90 it became a serious health problem:

> We had several deaths during the first half of the year, and the latter half was not better as regards the health of the people, for influenza visited the station and was very severe on the poor blacks, all of whom were attacked and will probably not feel much better until the warm weather sets in again (Stahle 1890).

The records of mortality from the settlement reveal nine deaths of a total of twenty-five during the period 1886 to 1890 that could be attributed to infectious respiratory diseases (Appendix B).

6.4.4 Influenza and acute respiratory disease among South Australian Aboriginal populations.

The first reliable records of influenza or acute respiratory diseases among the Aboriginal people of South Australia do not appear until 1850, fourteen years after the colony of South Australia was established. There is no evidence regarding the effect of the pandemics of 1838 and 1845-47, nor of the local epidemic of 1845. Little knowledge of the health of the Kaurna, the Ngarrindjeri and the Peramantgk, the populations closest to the European settlements is available in this period. In 1844 the Protector of Aborigines, Dr. Matthew Moorhouse, reported to the Governor of the colony on the state of the Aboriginal population (Moorhouse 1844: 358). He included a brief account of the diseases suffered by them, listing venereal diseases and tuberculosis as the main diseases and concluded that they were:

> subject to those [diseases] common to the human race, as inflammation and its consequences... (Moorhouse 1844: 358).

Moorhouse's reference to diseases was vague and whether or not he was making a reference to influenza epidemics (Cumpston 1989: 313) is uncertain.

By 1850 acute respiratory diseases were becoming a serious source of morbidity and mortality. In a quarterly report to the Governor, Moorhouse reported on an epidemic which was most likely influenza with cases of secondary pneumonia:

> I regret to have to remark, that the epidemic characterised by inflammation of the lungs, continues to afflict the natives ... The deaths of three children and three adults have been recorded by me... I have little doubt that many more have died in the scrub, whose deaths will remain unknown to me for months, if not altogether. The aborigines ... appear to have an instinctive knowledge of the approach of death, and a desire to let the breath of life depart at or about the place where it was first inhaled (Moorhouse 1850: 611).

In this instance Moorhouse may be referring to a local remnant of the major epidemic of 1847-48 in the eastern colonies.

Beginning in 1843 and continuing into the next decade, a regular migrational trend developed when the Ngarrindjeri from the lower Murray River and Coorong and the Meru from the eastern river region of the Colony began to gather in Adelaide (Moorhouse 1843a). They displaced the remnants of the local Kaurna and established encampments on the banks of the Torrens River with numbers often exceeding seventy or eighty individuals (Moorhouse 1843b; 1853). They became a common sight to the residents of Adelaide staying each year for most of the winter months. Exposure to the cold, poor nutrition, inadequate sanitary conditions, and alcohol abuse made them extremely susceptible to infectious disease. After several years a concerned citizen wrote to the *South Australian Register*:

> It is painful to every one who comes into contact with the natives to witness the dreadful colds with which they are nearly all afflicted in such a winter as the present. The severity of such complaints among them is no doubt chiefly owing to the want of shelter from the inclemency of the weather. Would it be

> too much for our Government to provide them, out of our abundance, with a building which would afford them some more substantial shelter than their wretched "wurlies"? Their rude wants would require nothing elegant or costly (*South Australian Register* 1855).

In 1852 the gold rush in the eastern colonies created a shortage of European labour on the rural properties and Aboriginal people were required by the settlers to assist with the crops. As a consequence, many of the Ngarrindjeri and Meru moved to the agricultural districts where good wages and ample food supplies were offered for their labour. A much larger number than usual came to the city late in the year. Moorhouse reported:

> In November, the City of Adelaide was visited by upwards of three hundred natives; Encounter Bay, Lake Alexandrina, and Murray River tribes, all met by appointment. The Lake Alexandrina tribe undertook to reap about ninety acres of crop... They would have done more, but the influenza was raging at the time, and the whole becoming more or less affected by it, ceased their reaping, and returned to their own district (Moorhouse 1853b)

The influenza epidemic had already reached the Ngarrindjeri before their arrival in Adelaide. Sub-Protector Mason reported to Moorhouse in July 1852 from Wellington on the lower Murray:

> I am sorry to report that a great amount of sickness prevails among the natives at the present time, many are suffering from complaints of the chest, and rheumatism. In such cases of sickness amongst them, they are generally treated with kindness by the settlers (Moorhouse 1852).

West of Adelaide on Eyre Peninsula, the Nawu who occupied most of the south and west of the peninsula were suffering the effects of the epidemic in 1852 and 1853. Archdeacon Hale informed Moorhouse:

> In reporting upon the state of this Institution for the past four months, the subject which, on account of its importance first demands our attention, is the great extent to which sickness and mortality have prevailed amongst the inmates during this period (Moorhouse 1853b).

It is not clear from Hale's report what the cause of the mortality was but a later report by him, which includes a brief autopsy report, alludes to an epidemic of acute respiratory disease:

> I am again under the necessity of referring ... to the frequent instances of mortality amongst our inmates. We had no less than three deaths since he date of my last report. In the last case a *post mortem* examination was made by our medical attendant, Dr. Lawson. He found the lungs to be extensively diseased; one lobe having a very large ulcer upon it and the other lobe being in a highly inflamed state (*Adelaide Observer* 1853)

The pandemic of 1850-52 affected the colonies of New South Wales and Victoria (Table 6.1) but appears not to have influenced the morbidity or mortality of the South Australian colonists. The origin of the above outbreak of influenza among the Ngarrindjeri and Meru living in Adelaide is then somewhat of a mystery. The best way to account for it is to assume that there was a low level of prevalence for the disease among the colonial population of Adelaide and the disease was transferred to the Aboriginal people where it developed a more serious morbidity.

Between the pandemic/epidemic events of 1852-53 and 1885 localised epidemics of influenza and respiratory disease recurred among the Aboriginal communities of South Australia. Acute respiratory disease continued to be a cause of high morbidity and mortality among the Nawu living both on and off the Poonindie settlement. In 1857, Octavius Hammond, a qualified medical practitioner, who succeeded Hale as superintendent reported that 'bronchitis' and 'pleurisy' were among the most commonly developed diseases (Hammond 1857). He observed the severe effects of the disease in the rapid onset of symptoms and little resistance offered by the patients. He further stated:

> While speaking on this subject, however, it should be borne in mind, that there has existed a similar state of disease and death among the natives in the bush, in this neighbourhood, in a proportion, probably equal to that experienced at Poonindie (Hammond 1857).

The 1860 pandemic saw the return of influenza universally across Southeast Australia. It was reported to be causing excessive morbidity and mortality, this time mainly among the Ngarrindjeri. George Taplin, from the Point McLeay settlement, informed the Aborigine's Friends Association that:

> The suffering of the natives from cold have been very great. The country from which they used to get their opossum-skins for rugs being nearly taken up, they are dependent on blankets for protection from cold. But it unfortunately happens that the blankets supplied by Government are not distributed when winter sets in. Had this been done, many who have this winter fallen victims to influenza might have survived (Taplin 1868).

The excessive morbidity and mortality among the Ngarrindjeri, implied by Taplin, was observed by an anonymous settler living on the Coorong. He informed the *Weekly Chronicle* that 'a great many died from the influenza', during the winter months of 1860, 'generally brought on from being warmly clad one day and being in a state of nudity the next' (*South Australian Weekly Chronicle* 1861)

By the mid 1860s respiratory disease among the Aboriginal people of South Australia was common and wide-spread. Reports of morbidity and mortality from respiratory infections in between the influenza epidemics of 1860 and 1885 come from several observers: Sub-Protector Buttfield noticed 'catarrhal affection' among Narangga on northern Yorke Peninsula in July 1866 (Buttfield 1866). Elderly Narangga suffering from acute respiratory disease would regularly go to the Point Pearce settlement to obtain treatment, and after many deaths from 'inflammation of the lungs' on the mission, several,

children were taken by their parents to live away from the settlement and avoid the continuing infection (Scott 1867). George Taplin noted among the inmates of Point McLeay in 1868 that, 'We have lost that very troublesome visitor the hooping [*sic*] cough which only carried off three or four children altho [*sic*] so many suffered with it' (Taplin 1868). In 1878 Hamilton reported that the Aboriginal people throughout the colony were 'exceedingly susceptible to colds, which often settle on their lungs (Hamilton 1879). The *South Australian Government Gazette* (1875, 1879) listed respiratory diseases (influenza, whooping cough, and 'inflammation of the lungs') as the highest cause of mortality after tuberculosis in the official returns of the causes of death in 1875 and 1879. The returns of sick Aboriginal people under treatment in the Adelaide Hospital and among the Buandig in the south east of the colony during 1874-76 show recurring cases of bronchitis, pneumonia, influenza, catarrh, and 'pleuritis' (*South Australian Government Gazette* 1874, 1875, 1876).

In 1874-76 acute respiratory diseases inflicted excessive morbidity and mortality. The respiratory infections accompanied a major epidemic of measles which spread throughout Southeast Australia (See Chapter Seven). Sub-Protector Hamilton reported a 'great deal of sickness' among the Ngarrindjeri on the south coast in February 1874; chiefly influenza accompanied by scarlet fever and rheumatism. In the north of the colony Buttfield (1874) reported high morbidity, though few deaths, from influenza.

While acute respiratory diseases were becoming established in the settled southern and western regions of the colony, a localised epidemic began among the Aboriginal populations in the north. A boom in colonial expansion in South Australia began in 1869 into areas specially selected for their suitability to cultivation (Williams 1969: 26). The spread of settlement north along the Flinders Ranges brought the Europeans into closer contact with the Banggarla, Kuyani, and Adnyamathanha. In May

and June of 1870 unusual heavy rainfall fell along the Flinders Ranges. In a six week period the rainfall was measured at ten inches (250 mm), almost the average annual amount. Sub-Protector Buttfield, responsible to the Commissioner of Crown lands for the welfare of the Aboriginal people in the Northern Districts, recorded an epidemic of what was most likely influenza among the Kuyani and Adnyamathanha around Blinman in June:

> Owing to the heavy rainfall ... travelling has been quite impracticable during the month of May. My time has, however, been fully occupied for the cold and wet has increase the number of patients. Coughs, colds, sore throats have been very prevalent. I have three cases under treatment of a more serious nature - haemoorhage [*sic*] of the lungs (Buttfield 1870a).

The three 'haemoorhage' cases may have been pulmonary tuberculosis.

Buttfield had been confined to the settlement and ration station at Blinman during the rains and had little or no contact with the people living away from Blinman and was unable to report on their condition. Two months later, however, another report by Buttfield reveals that the morbidity from respiratory diseases had increased:

> Owing to the extreme inclemency of the weather my sick list was considerably augmented during June. The Aborigines suffering from Catarrh and other disorders. Scarcely a day passed without bringing some poor creature seeking for relief (Buttfield 1870b).

By the end of the year the epidemic had waned and Buttfield reported in May 1871 that 'there has been less sickness during the past three months than at any similar period during my residence here' (Buttfield 1871a). During the remainder of 1871 Buttfield visited an area of the Flinders Ranges to the north and east of Blinman and had found the people in a satisfactory condition with only a few cases of sickness (Buttfield 1871b; 1872).

The continuing trend of improving health and the drop in morbidity from respiratory diseases reflected in Buttfield's reports was attributed to the high rainfall in 1870. Buttfield (1871b) reported that the rain had promoted growth of plant and animal food sources which had enabled the people to move away from the European settlements and ration depots into the countryside. This in turn might have helped them to throw off some of the less serious infections, and while being dispersed and not living in crowded damp conditions near colonial settlements, enabled them to avoid further infection.

In 1885 the disease was again reported in the northern regions of the colony, this time among the Dieri. It claimed several living on the Killalpaninna Mission settlement and an unknown number living in the Lake Eyre and Coopers Creek region (Stevens 1994: 112).

Of the remaining nineteenth century influenza epidemics the most serious was the one that appeared in the last decade. It was reported to have come from Melbourne in March 1890 (Cumpston 1989: 331) and spread rapidly in all directions when it reached Adelaide. Forty-eight deaths from influenza were registered among the 320,000 European population of the colony during the 1890 epidemic. The epidemic may have lingered among the Aboriginal populations in the north of the colony until 1893. Sub-protector Beasley (1893) reported influenza spreading among the populations following along the Transcontinental railway line to the Coopers Creek region, and among the Banggarla and Nukunu north-west of Port Augusta. The extent of morbidity and mortality from the disease is unknown as most cases were not seen by the sub-protector and most deaths went unreported.

It should be noted that Aboriginal populations continued to suffer high morbidity and mortality form influenza epidemics elsewhere in Australia and into the twentieth century. The most severe episode was the 1918-1919 Spanish Influenza pandemic. This pandemic cause 12,167 recorded deaths in Australia. In Queensland 1,030 died of which 315 (30.6%) were Aboriginal (Briscoe 1996: 1). The real figure is almost certainly much higher than this because many Aboriginal deaths, particularly in isolated regions, went unrecorded (Briscoe 1996: 7-9). Because few records of Aboriginal deaths were taken little is known of the impact of the pandemic on Aboriginal populations elsewhere although high morbidity and mortality would be expected. More than 60 years after the pandemic it was still remembered by Aboriginal groups. In 1982 Professor McBryde was shown an area of sand dunes south of Lake Eyre by an Arabana elder, who knew it as a place where many of his people who had died 'of the `flu' in 1918 and 1919 were buried (McBryde pers. com.).

6. 5 Discussion

The introduction into colonial Australia of pathogens causing acute respiratory diseases had a long-lasting and severe effect upon the Aboriginal population of the continent. The first recorded cases of influenza reaching the Aboriginal people of Southeast Australia are in 1820 during the period of the first pandemic to reach Australia. Was this then the first appearance of transcontinental respiratory disease to involve the Australian Aboriginal population?

Although we cannot be entirely certain that there were no outbreaks of influenza in Southeast Australia prior to 1820 (for example there is no record of the 1800 world pandemic reaching Australia) its apparent absence during the initial years of colonial settlement can be accounted for epidemiologically (Young 1979: 207). The

colony of New South Wales was isolated geographically and temporally from the major Old World sources of infection. Outbreaks of influenza, which were common enough aboard ships leaving from England, often flared up in the initial days following departure but in most cases were self limiting, failing to survive the long sea journey to Australia (Cumpston 1919). Even after calling in to ports where influenza was endemic en-route to the colony, the disease often burnt out before arrival at Sydney (Cumpston 1919). If the disease did persist onboard and was to reach the colony its progress would then have been impeded by the small and dispersed nature of the settlements in New South Wales, and the low proportion of susceptible children in the colony's population (Curson 1985: 9). Nevertheless, infections of influenza may have resulted prior to 1820, but if they did they were most likely to have been localised and self-limiting and so of minor impact on the colony in general.

A similar set of epidemiological circumstances applies to the Aboriginal people. Prior to 1820 many Aboriginal populations living along the south-eastern and southern coast had had contact with the sealing and whaling fleets as early as 1798. Large-scale commercial sealing operations quickly developed, particularly among the Bass Strait islands and the southern coastline. Kidnapping of women by the sealers for sexual purposes and to assist in the capturing of seals was a common practice which lasted into the 1840s when the industry declined (Ryan 1996: 66-72). There are no records from this period of influenza, or any other respiratory diseases, affecting the Aboriginal people who came into contact with the sealers (and perhaps little reason to expect such documentary evidence as many of the sealers were illiterate and those who did leave diaries were more concerned with the commercial aspects of the industry and had little concern about the health of the Aboriginal people). Nevertheless, the means and opportunity of passing on influenza did exist with contact between the Ngarrindjeri and

the sealing ships (for example I have discussed the likelihood of venereal disease being introduced to the Ngarrindjeri this way).

The pandemics/epidemics of influenza occurring among the European colonies of Southeast Australia were largely paralleled among Aboriginal communities. Table 6.5 shows the temporal relationship of periods of excessive morbidity and mortality from influenza and secondary respiratory infections among Aboriginal communities and their relationship with world pandemics and localised epidemics among the European population. The six nineteenth century influenza pandemics that reached Australia all had an effect upon Aboriginal communities of Southeast Australia. Preceding the first pandemic and following the second and fifth were four localised epidemics that spread through the European communities and into the Aboriginal communities. This table does not, however, detail the full extent of influenza throughout the Aboriginal communities in each of the colonies. There can be few doubts that many of the outbreaks either went unrecorded or those recorded events have not survived or are not included in historical archives. Nevertheless, what evidence is available shows a strong correlation between pandemics of influenza and outbreaks among Aboriginal communities.

Table 6.5 Evidence of influenza epidemics among Aboriginal populations in Southeast Australia showing their relationship to Pandemics and localised epidemics.

Year	Pandemic/epidemic	Colony/region	Remarks
1820	localised epidemic	New South Wales	Probable high morbidity & mortality
1826	localised epidemic	New South Wales	Effect unknown
1831	Pandemic	Gun Carriage Is. (Tas)	
1836-38	Pandemic	Victoria, Flinders Is.	High morbidity & mortality; acute respiratory diseases common on Flinders Is.
1845	localised epidemic	Victoria, South Australia?	High morbidity & mortality; acute respiratory diseases becoming established
1847	Pandemic	Flinders Is., Oyster Cove (Tas);	High morbidity & mortality
1852	Pandemic	Victoria, South Australia	High morbidity & mortality
1860	Pandemic	Oyster Cove (Tas), South Australia	High morbidity & mortality
1870	Localised epidemic	Northern South Australia	High morbidity & mortality
1874-76	Pandemic	Northern South Australia	High morbidity & mortality
1890-91	Pandemic	Northern South Australia	High morbidity & mortality

Little can be said of the first recorded influenza epidemics in the 1820s. From the handful of extant records it can be assumed with some confidence that influenza reached the Aboriginal people in Southeast Australia during the 1820 pandemic. The full epidemiological impact and spatial diffusion of this epidemic is, however, unknown. It went largely unrecorded among both the European population of eastern Australia and the Aboriginal populations of the southeast region. Many of the Aboriginal populations who would have suffered the impact of the disease were beyond the colonial frontiers and so beyond the cognizance of the colonists. The description of the clinical symptoms of this disease, its selective and sometimes swift mortality, and the wide-spread morbidity that is inferred by the few recorders is consistent with those of an epidemic of influenza or influenza-like disease in a population not previously exposed to the virus (Cook 1973, 1992; Carlson *et al.* 1992; Dobyns 1983, 1992; Guerra 1988; Lange 1984; Marshall 1993; McArthur 1967; McNeill 1976:195; Walker & Johnson 1992:131-133).

Influenza probably became endemic among the Aboriginal populations of Tasmania sometime in the 1830s and on mainland Southeast Australia between the decades 1840 and 1850. This period saw four pandemics reach Australia and at least one localised epidemic in the southern colonies. The 1836-38 pandemic began the establishment of influenza. This epidemic shows a pattern of first establishing a foothold among European colonists before spreading to Aboriginal people living in the immediate vicinity of the main colonial settlements and then possibly spreading out into the inland populations. It was first recorded in Sydney during October and November 1836 where the fatality among European hospital cases was 14.3% (Cumpston 1989: 313). It then moved south to Tasmania (Van Diemens Land) and Port Phillip probably by sea. Cumpston (1989: 313) states that the epidemic reached South Australia in 1838. There is, however, no evidence of it crossing to the Kaurna from the settlement of Adelaide until some eight years after the colony had been established.

The Woiworung, Boonwurrung, Taungurong, and the Wathaurong in the districts surrounding the Port Philip settlement were the first Aboriginal people to suffer the effects of influenza in the newly established Victorian colony. The large and conspicuous gatherings on the Yarra River which began in February and March 1839 had been unwelcome to the Government and by the European community as a whole. Their meeting with Chief Protector Robinson and his sub-protectors achieved little in the way of conciliation, and misunderstanding between the two cultures continued. For the large assemblage gathered together in such numbers on the edge of a European settlement, itself battling a severe epidemic of influenza, the meeting had other serious consequences. Many of them were infected by influenza and an unknown number of them died. While the number of deaths cannot be calculated, it is reasonable to assume that there was a substantial mortality from influenza among them while they were in Melbourne. Their situation there was worsened by the unavailability of the natural food resources of the

region which had been present in pre-European times but was now either severely depleted or denied to them by European land-holders. Possibly adding further to their susceptibility to influenza was the high prevalence of chronic and severe syphilis symptoms which affected both sexes and all ages. Severely suppressed immune systems could not function efficiently to adequately counter the invasion of the influenza virus.

Many of those who were among the first groups to leave Melbourne were in all probability still suffering from influenza and were contagious. Did they then take the infection back with them to their own homelands and further disseminate the disease? If influenza did indeed spread among the Aboriginal people living inland and far away from the main settlement of Melbourne, it went unrecorded and we have no knowledge of its impact. It would appear, however, that by the early 1840s it was absent among populations living away from the main European settlements. Chief Protector Robinson made extensive tours of the central and western districts of the colony in 1840 and 1841, contacting large groups of Aboriginal people (Presland 1977a, 1977b, 1980). He saw several with possible syphilis but makes no mention of active cases of influenza. Although he noted a sharp decline in numbers among some of the Aboriginal populations he considered it to have been from a disease or diseases which had occurred 'about the time when the first white men came', probably in 1834 (Presland 1977b: 48). Dr. Watton, who was in charge of the Mount Rouse Aboriginal station in western Victoria, records no occurrence of influenza or pneumonia among the Gunditjmara or surrounding populations between January and November 1840 (Watton 1840). In 1842 Watton examined thirty-three patients between October and December and found only one, a thirty-year old male, suffering from a respiratory disorder which he diagnosed as bronchitis (Watton 1842). Again in 1842 Campbell, the medical officer for the Goulburn River region, stated in his monthly report of the Aboriginal station that 'very few cases [of disease] of any importance had occurred among the Aborigines' (Campbell 1842). Two years later, in his

1844 annual report for the Loddon River Aboriginal Station, Sub-Protector Parker made no reference to influenza or the state of health of the Jardwadjali under his supervision. A total of 316 people had visited his station during that year with an average daily presence of sixty-two (Parker 1845).

The lack of documentation on influenza among the inland populations of Southeast Australia until 1845 should not be seen as evidence for its total absence. The highly contagious nature of the disease and a period of communicability ranging from three to seven days makes influenza an efficient traveller among humans moving from one place to another. There is then a high chance that it accompanied the many infected Aboriginal people leaving Melbourne to their homelands and disseminated further. The documentation does, however, suggest that if the epidemic did disseminate inland of Melbourne it had burnt itself out by 1840.

Soon after the colony of South Australia was proclaimed in August 1836 a pandemic of influenza was swept through the eastern colonies. Nothing is known of its impact on the Kaurna living along the coastal plains of Gulf St. Vincent. The initial founding colonists had come by sea from Britain and had no contact with the eastern colonies, so in all probability were free of the disease when they arrived. Cumpston (1989) and Warburton (1973) record local epidemics of influenza among the colonists in 1838, two years after the settlement and later in 1844-45. The reference by Moorhouse (1844: 358) to 'inflammation and its consequences' coincides with the second outbreak and may be the first evidence of acute respiratory disease among the Aboriginal people of South Australia. There is, however, no evidence that the epidemic inflicted the excessive morbidity which it had done among the Aboriginal people of the Port Phillip Colony. A clearer picture emerges during the 1850s. Social conditions arising from contact with European colonists would have aided the dissemination of influenza. Large groups of

Ngarrindjeri and Meru were annually moving out of their homelands to live in temporary camps along the banks of the Torrens River in Adelaide. Moorhouse (1854) estimated a group of over seventy, mainly Meru, in July 1854. They usually arrived in Adelaide at the beginning of winter and departed in the spring. By spending the winter months in Adelaide the Ngarrindjeri and Meru faced serious hazards that affected their health, impaired the function of immune responses, and increased their susceptibility to acute respiratory diseases. Alcohol and tobacco became newly desired commodities and each was readily obtained from the European community by cash payment to the publicans or by the prostitution of women. Addiction to both drugs, particularly alcohol, was widespread (Moorhouse 1854).

Little food was available to those in the encampments and the level of nutrition was poor. Although the government later set up food and ration depots away from Adelaide, very little was done in the way of supplying food to the encampments. The colonial government was put under pressure by residents of Adelaide to remove the groups from the Torrens and from the township. In their daily travel from one part of the township to another, the residents of Adelaide came face to face with the disease and squalor of the encampments and were often confronted by Ngarrindjeri or Meru men demanding money or women soliciting for alcohol, tobacco, or tea (Dowling 1990: 173-176; Summers 1986: 295-296). The epidemiological effects of these regular visits to Adelaide for the Ngarrindjeri and Meru was an annual exposure to respiratory infections which they often took with them when they left Adelaide and returned to their homelands. Influenza infections became an annual experience for them often having fatal results.

Influenza infection reached the populations west and north of Adelaide through the process of colonial expansion. During the 1850s and 1860s influenza was recorded among the Nawu and Banggarla of Eyre Peninsula and the Narangga of Yorke

Peninsula. Later during the 1870s as the colonial frontiers were pushing further north the Kuyani and Adnyamathanha in the mid-north regions became exposed. The construction of a railway line linking Peterborough on the edge of the wheat-lands to the mines at Broken Hill took influenza further to the north and west to infect the Dieri and Yawarawarka in 1891.

In many cases influenza reached into Aboriginal populations that had already been exposed to foreign pathogens and whose social structures were beginning to feel the impact from British colonisation. Aboriginal people already, immunologically compromised with acute chronic diseases, particularly syphilis and tuberculosis would have had suppressed immune systems further weakened by nutritional deficiencies experienced as a result of colonisation. They would have been highly susceptible to influenza, particularly at the times when the disease became epidemic and would have had little resistance to the progress of the disease leading to secondary bacterial pneumonia. In turn, pneumonia, often as a secondary complication of influenza, became a major morbidity and mortality problem. Among the Aboriginal people living at Wybalenna on Flinders Island and later at Oyster Cove on mainland Tasmania, respiratory disease displayed a long standing chronicity in some and in others it severe manifestations that lead to death within 3 weeks (Plomley 1987:915-942; Smith 1858-69).

The morbidity pattern of respiratory disease, changed from initial epidemics of influenza and pneumonia with few infections between epidemic periods, to a background of infection particularly on sedentary populations, interspersed with epidemics affecting both the Aboriginal and the European populations.

6.6 Conclusion

The history and pattern of epidemics of influenza among the European population in Australia has paralleled that of the northern hemisphere. Influenza outbreaks among the Aboriginal population paralleled that of the rest of the Australian population. Documentation of the severity of influenza among the Aboriginal population for most of the pandemics that reached Southeast Australia in the nineteenth century is missing, and little more can be said about them other than that the morbidity and mortality was severe in each case; probably equal to, or more than that among the non-Aboriginal population. The epidemics of 1839 and 1847 were notably severe, particularly in 1839 among the Tasmanians incarcerated on Flinders Island and among the populations in southern and central Victoria.

The documentary evidence reveals that Aboriginal populations suffered excessive morbidity and mortality when initially exposed to the influenza virus. The changing style of life in residential communities such as the large gatherings in Melbourne and Adelaide, and the incarceration of Tasmanians on Flinders Island and at Oyster Cove favoured the spread of respiratory diseases. Infection rates by the influenza virus increased and facilitated a rapid build-up of case-to-case transmission which resulted in explosive epidemics and a persistence of virus between major outbreaks.

By the 1850s the pattern of acute respiratory disease, particularly influenza and pneumonia as endemic diseases had become established in the Aboriginal populations of Southeast Australia. The diseases were regularly reported by medical and non-medical observers during inter-pandemic periods. While there were epidemic years with excessive morbidity and mortality, influenza and secondary respiratory complications became ever-present. It would probably be correct to state that every year, since its first introduction into Aboriginal populations, could be termed an influenza year.

CHAPTER SEVEN

Measles

7.1 Introduction

This chapter discusses the final of the major epidemic diseases responsible for excessive morbidity and mortality among the Aboriginal people of Southeast Australia during the nineteenth century. Like smallpox, measles has been one of the most devastating of the transoceanic diseases; it became a major cause of 'demographic disasters' in virgin soil populations throughout the world (McNeill 1976: 194). During the European colonial expansion into the western hemisphere and Pacific beginning in the sixteenth century, measles was responsible for excessive mortality among the Native Americans (Boyd 1990: 138; Cook 1973; Crosby 1972; Dobyns 1983; Reff 1992; Stodder & Martin 1992), and the Pacific Islanders (Cliff & Haggett 1985; McArthur 1967). McNeill (1976: 194) states that when measles swept through Mexico and Peru in 1530-31:

> Deaths were frequent, as is to be expected when such a disease encounters a virgin soil population dense enough to keep the chain of infection going.

Four centuries later measles reached the Fijian Islands. During the first four to six months of 1875 it was estimated to have killed between 27,000 to 40,000 people comprising 20 to 30% of the population (Cliff & Haggett 1985: 35). Earlier, in 1848, measles killed 40,000 (c.26% of the population) in Hawaii (Kim-Farley 1993: 874).

From the early 1830s when the first measles outbreaks were recorded in Australia, the disease occurred in regular epidemic events throughout the nineteenth century among the European population (Cumpston 1989: 305). Focusing on stage II

(early contact and diffusion) of the medical model (Table 2.1) the historical evidence for measles in the Aboriginal population of Southeast Australia will be examined.

7.2 Biology of Measles

Measles is a highly communicable, common, and acute systemic disease, principally of children. It is caused by the rubeola virus which today has a world wide distribution. Humans are the only reservoir for the virus, which means that a continual chain of infection must be maintained for the disease to spread and the virus to remain within a population. The virus is spread by contact with secretions from the nose and throat by airborne droplets or by direct contact with an infected individual. The virus can also be transmitted by contaminated articles such as clothing and blankets.

Measles commonly presents an initial fever followed by Koplik's spots on the buccul mucosa combined with a dry cough, coryza, and conjunctivitis. A second exanthematous phase is characterized by a spreading maculopapular rash beginning behind the ears and continuing down the face and body, reaching the feet in 2 to 3 days. The period of infectivity is from two days before the onset of the prodromal phase of the disease to four days after the appearance of the rash. The disease is usually benign and self-limiting in children but can have serious effects in the immunosuppressed, where nutrition and hygiene tend to be poor, and where there is a lack of supportive care. Post measles pneumonia (caused by the virus or by secondary bacterial infection), encephalitis, and otitis media causing deafness can occur as secondary symptoms, particularly during epidemics. Severe diarrhoea can follow as a complication of measles infection, and is often the main cause of measles-associated mortality (Benenson 1990: 269-271; Kim-Farley 1993: 873; Merlin *et al*. 1994: 342-343; Morley 1980: 121-126).

7.3 Measles among the European population of Southeast Australia

Table 7.1 lists the occurrences of recorded measles epidemics among the European population in the colonies of Southeast Australia during the nineteenth century.

Table 7.1 Nineteenth century epidemic years of measles in the European population of Southeast Australia by colonies (Cumpston 1989; Donovan 1970).

N.S.W.	Vic.	Tas.	S.A.
1834	-	1835	-
-	1853-54	1854	-
1860	1860	1861	1861-62
1867	1866-67	-	1868
1874-75	1874-75	1874-75	1875
1880-81	1880	1881	1881
1883-84	1884	1885	1884
1888	-	-	-
1893	1893	1893	1893
1898	1898	1898	1898

The date of first entry of measles into Australia is not entirely clear. Cumpston (1989: 305) states that measles was unknown in the colonies until 1842 when two deaths were reported in Tasmania But a disease resembling measles had previously been reported in the Illawarra district, south of Sydney in 1832. According to Donovan (1970: 5) measles was first introduced into New South Wales by infectious patients arriving in Sydney on board the *David Scott* in October 1834. It was diagnosed by military surgeons when an epidemic broke out in Sydney and the outlying areas. Measles was further diagnosed in Tasmania during January 1835 where it also became epidemic. The same disease was most likely carried to the South Island of New Zealand resulting in the first epidemic among the Maoris (Curson 1985: 13-14; Donovan 1970: 5-10).

Measles was a common traveller aboard ships departing Britain, particularly amongst small children. The arrival of large numbers of free immigrant families to Australia after 1838 increased the presence of measles among the Sydney population (Curson 1985: 14) and the other colonies. It became established when it was reintroduced into Victoria by the ship *Persian* in 1850 becoming epidemic there and in Tasmania in 1853-54. The disease reached New South Wales in 1853, and South Australia in 1859. From then regular epidemics occurred among the colonies interspersed with years of low incidence.

Table 7.2 shows the official mortality figures from measles for the two major epidemics in Victoria and South Australia in 1874-75 and 1893.

Table 7.2 Mortality from major epidemics of measles in European population of Victoria and South Australia 1874-75 & 1893 (*Statistical Register of Victoria* 1876, 1894; *Statistical Register of South Australia* 1876, 1894)

Year	Measles deaths	Total deaths	%
Victoria			
1874	256	12222	2.1
1875	1541	15287	10.1
1893	659	16508	4.0
South Australia			
1874	280	3434	8.2
1875	75	4136	1.8
1893	261	4520	5.8

The most severe mortality occurred in the epidemic of 1874-75. It affected all the colonies in Southeast Australia (Cumpston 1989: 305) and was carried by seas from Sydney to Fiji (Cliff & Haggett 1985). It is not known exactly when measles became truly

endemic in Australia (the nineteenth century epidemics may well have been reintroductions of the pathogen); but the regularity of the epidemics since 1875 (Table 7.1) fit the well established pattern of measles in a population able to sustain its endemicity.

7.4 Measles among the Aboriginal population - source material

There are few historical accounts of measles among the Aboriginal people for the first half of the nineteenth century. The first record of the disease among the Aboriginal population is from the Reverend Threlkeld at Lake Macquarie Mission (Newcastle) in 1835. Its origin was most likely the 1834 outbreak in Sydney. In his Annual Report the Reverend Threlkeld (1835) commented on its presence:

> During the present year the Measles have been very prevalent among the Aborigines, and have carried off many of the natives, from whom Mrs. Threlkeld and our nine children caught the complaint and were laid up at one time.

The epidemic was not confined to the Aboriginal people living on the settlement. Threlkeld's report alludes to high mortality from measles and respiratory disorders causing extreme mortality among inland populations, probably the Biripi, Worimi, and Geawegal:

> ...the Measles, the Hooping cough, and the Influenza, have stretched the Black victims in hundreds on the earth, until, in some places, scarcely a Tribe can be found. Of one large Tribe in the Interior, four years since, there were 164 persons, there are now only three individuals alive.

Threlkeld's observations of population loss following the measles epidemic are supported by official returns of Aboriginal people enumerated at annual distributions of Government issued blankets. Table 7.3 shows a sharp fall of 511 (24%) in the

enumerated population of the settled districts of New South Wales between 1835 and 1837.

Table 7.3 Official returns of estimated number of Aboriginal people in the settled districts of New South Wales between 1835 and 1837 enumerated during government blanket distributions.

Year	Male	Female	Total	% Decrease
1835	1195	898	2093	
1836	952	630	1582	24.4
1837	930	601	1531	3.2

From Threlkeld's report it is unclear whether measles or influenza was the main cause of mortality. However, in January 1836 Charles Darwin, on the return voyage of H.M.S. Beagle, visited the colony of New South Wales and travelled inland into the Great Dividing Range as far as Bathurst. He commented on the mortality of measles among the Aboriginal population:

> This decrease no doubt, must be partly owing to the introduction of spirits, to European diseases (even the milder ones of which, such as the measles, prove very destructive (Darwin 1889: 520)

Threlkeld's missionary attempts had to be eventually abandoned due to the steadily falling population in the district, and by 1841 the mission closed. While neither measles, nor indeed whooping cough and influenza, can be held entirely responsible for such a population loss (Aboriginal deaths had also resulted from acts of aggression by European colonists) it was probably a major factor. Threlkeld (1835) was of the opinion that many had died from the 'Act of God' during the measles epidemic.

The 1853-4 epidemic was restricted mainly to Victoria and Tasmania and little is known of its effects on Aboriginal populations in those colonies. There is,

however, evidence of measles breaking out among Aboriginal people in South Australia. In August 1854 Surgeon Clindening reported on an outbreak of measles among Aboriginal employees at the Encounter Bay Fishery on Fleurieu Peninsula. It caused the death of at least one Aboriginal worker and probably infected more:

> The natives will not take medicine, and I have no doubt, from exposure to the inclemency of the weather, there will be great sufferers [*sic*] from the disease (Clindening 1854).

Clindening observed the same disease among European settlers on the peninsula, although no deaths were reported among them. If the outbreak was indeed measles it was in what was otherwise a non-epidemic period for South Australia and was most likely a localised event; the infection arriving by sea from ports in either Victoria or Tasmania.

Two outbreaks of measles causing severe mortality were reported among the Ngunawal people of the southern highlands in what is now the Australian Capital Territory, in 1858 and 1862 (Schumack 1927: 9, 1977: 51). There was no recorded epidemic in the surrounding European population of New South Wales during the 1850s but there was one among the European population in Victoria in 1853-54 (Table 7.1). If the 1858 outbreak among the Ngunawal was measles it may have came from the south in Victoria along with the annual movement of Aboriginal groups to the highlands to collect aestivating moths (Flood 1980, 1983: 202-4). Large groups of Aboriginal people gathering in the highlands were recorded until 1890,almost 60 years after the first arrival of Europeans (Flood 1980: 68-69). Alternatively it may have been a localised epidemic originating from the European settlers. The second outbreak in 1862 was most likely associated with the general epidemic in New South Wales and Victoria.

During the Tasmanian epidemic in 1860 Surgeon Smith reported one woman transferred from the Oyster Cove Aboriginal settlement to Hobart under treatment for measles (Smith 1858-69). She survived the infection and was returned to the settlement seven weeks later. This is the only mention of measles by Smith who attended the inmates of the settlement for a period of ten years. During 1860 four deaths were recorded, none were from primary measles infection or secondary complications. Although the Oyster Cove people were subject to many ailments, measles was never reported to be a serious cause of morbidity.

The 1867-68 measles epidemic was widespread throughout Victoria. Surgeon Gibson administered to the Aboriginal people living on the Coranderrk settlement between 1865 to 1875. He mistakenly reported the epidemic as 'intermittent fever', a contemporary medical term for recurrent fevers and chills associated with malaria (Morgan 1987). The disease was affecting the Aboriginal residents, the superintendent, and his family (Gibson 1869-75). In March he noted seven out of 105 suffering while several others were recovering from the infection. He returned in April and reported one death, a male 22 years of age, and a further sixteen under treatment; four were adults, five were in their teens, and the remainder ranged between two and ten years of age. Many were leaving the settlement to avoid the epidemic. By June the general health of the residents had improved with only four convalescing and by August the epidemic had burnt itself out. There is no evidence to the fate of those who left the settlement during the height of the epidemic and the presence of measles among Aboriginal people living off the settlements.

The evidence for the 1874-75 measles epidemic is more substantial in terms of its occurrence among the Aboriginal people. This epidemic was one of the more severe episodes of measles in the nineteenth century, occurring throughout the European

settled regions of Southeast Australia. Table 7.4 shows the crude mortality rate of four nineteenth century epidemics including 1874-75 and an interepidemic period for which there is information available in South Australia.

Table 7.4 Crude mortality rate from measles in the European population of South Australia for which there is information (Cumpston 1927:223).

Year	Mortality Rate Per 100,000	
	Males	**Females**
1874	144	136
1881	26	25
1882	6	1
1884	49	41
1893	74	74

The epidemic was also severe among the Aboriginal populations of southeast Australia. Figure 7.1 depicts the extent of reported cases of measles infection discussed below. The epidemic extended into several of the mission settlements causing high morbidity among European and Aboriginal residents alike and in some cases exceedingly high mortality among Aboriginal people. Other Aboriginal settlements experienced high morbidity but recorded few deaths. Some regional centres of Aboriginal population and settlements escaped the epidemic completely.

The first reports of the epidemic among Aboriginal people began in Victoria. On 20th December 1873 Surgeon Gibson noted an 'eruptive disease' among Aboriginal people on the Coranderrk settlement. He had not seen any Aboriginal people displaying the primary stages of clinical symptoms but only the 'marks left on a few cases' (Gibson 1869-75). He diagnosed it as a species of *Papulae viccae,* and reported that there was no

danger from it. Whether these were the first cases of measles and Gibson was mistaken in his diagnosis will remain unknown, but by 20th February the following year measles was spreading through the community on the settlement; 15 cases were treated out of a population of 142. By 20th March, apart from a few of the elderly people, who may have developed immunity in a previous epidemic, all of the 142 residents had been attacked by the disease. Gibson noted that none of the cases was severe and with a little care he anticipated a satisfactory result (Gibson 1869-75).

Gibson's optimistic prognosis was grossly incorrect. By the end of 1875 measles had caused extreme morbidity and excessive mortality among the Aboriginal residents. So high was the mortality that it came to the notice of the Victorian Parliament. The report to the Legislative Assembly by the Colony's Chief Medical Officer is worthy of repeating at some length:

> ... in 1875, with a population of about 150 people, 31 deaths took place - one out of every five human beings in one year perishing from disease. This awful mortality was doubtless exceptional, an epidemic of measles having been prevalent in the early part of the year; but this epidemic prevailed all over the colony, causing a considerable increase in the general mortality; yet when the mortality of the whole colony, about 17 per 1,000 is compared with that of [the Aboriginal residents of] Coranderrk, the discrepancy is appalling, the latter amounting to 193 per 1,000, or in other words, for every person out of the general population who died, 11 deaths occurred at Coranderrk (McCrea 1879-80).

McCrea's assessment of the crude mortality rate at 193 per 1,000 is based on all the recorded deaths on Coranderrk settlement for 1875 and from a population which he estimates at 150. His estimate was close. An examination of the settlement's register of births, deaths, and marriages (Massola 1975) shows 27 deaths that could be attributed to the measles epidemic. Seven deaths were from measles directly, eleven caused by pleuro-pneumonia, and four others caused by respiratory disorders and dysentery. Three other deaths of children under one year old were given as 'thrush', a possible misdiagnosis

for the prodromal stage of measles ; and another, a two year old male given as 'disease of the ear affecting the brain', was possibly post measles otitis media developing into encephalitis. The population of the settlement just prior to and during the epidemic was 142, counted during Gibson's visits. The crude death rate from measles at Coranderrk would then be 190 per 1,000.

Other settlements in Victoria were affected by the epidemic although not to the same extent as Coranderrk. On 9th December 1874 Surgeon Brewer (1875) reported an 'exanthematous disease' causing some illness and sore throats at Lake Condah settlement. He reported the disease as scarlet fever. After his visit most of the children of the settlement were attacked by measles together with the superintendent's own children. The disease was seen as mild, causing no great stress to the Aboriginal children. By 3rd January, however, measles had spread to most of the population (numbering c.86) with twenty-four showing the signs.

> I found nearly all the adults down with the measles. They were much more severely affected than the children had been - two had severe pneumonia, three severe bronchitis, two diarrhoea, one constantly recurring haemorrhage from the nose, one woman just recovered from confinement had severe bronchitis. (Brewer 1875).

The children, who had initially been infected, had recovered with just two in a low febrile condition with pneumonia. The remainder of the establishment Brewer found in bed or lying around in various symptomatic stages. Three deaths were recorded by Brewer; two elderly females, one of whom died from post measles complications after recovery from the initial infection, and a fourteen year old. A fourth death was later recorded in official mortality statistics of the Aborigines Protection Board (APB 1876). All other victims gradually recovered. By the end of April the disease had passed Lake Condah settlement.

On Framlingham settlement Surgeon Jamieson (1875) reported measles affecting approximately 20 out of a population of 58. The disease had been brought to the settlement by returning Aboriginal males who had been working in the immediate district where measles was prevalent among the European population. By 30th April Jamieson reported all residents had recovered from their symptoms. No further cases were reported.

Table 7.5 shows the Aboriginal mortality statistics from measles or post measles complications during this epidemic which were reported to the Victorian Parliament by the Aborigines Protection Board. The deaths were all recorded on Aboriginal settlements and do not include Aboriginal people who may have died away from the settlement, for example, when fleeing from the epidemic; nor are those who were attached to European rural properties or residences included.

Table 7.5 Number of Aboriginal deaths on Victorian Aboriginal settlements attributable to the 1874-75 measles epidemic.

Settlement	Deaths attributed to measles	Approximate average population	%
Coranderrk	27	142	19.0
Lake Condah	4	86	4.7
Framlingham	6	58	10.3
Lake Hindmarsh	16	78	20.6
Lake Wellington	0	68	0
Lake Tyers	0	73	0

A differential rate of death from measles ranging from 0% to 20% is revealed in the above table. The two Gippsland settlements of Lake Wellington and Lake Tyers (Fig 7.1), located on the east coast, escaped the epidemic with no reported deaths

although the surrounding European population was affected. Separation of the Aboriginal residents from the European population, particularly during the period of the epidemic, most likely saved these two settlements from infection. Reverend Hagenauer, superintendent of Lake Wellington, reported:

> Different diseases have been about in the district, such as scarlet fever and measles, and not one of our natives have had them though the measles were within two miles of the place (Hagenauer 1877).

The settlements to the west were, however, involved in the epidemic with varying degrees of mortality reported. A similar spatial pattern of differential mortality during measles epidemics occurred during the epidemic of 1867 among the European population of Sydney (Curson 1985: 62-66) and in the islands of Fiji in 1875 (Cliff & Haggatt 1985).

Following the outbreaks in Victoria, measles appeared among the Aboriginal people in South Australia (Fig 7.1). In the northern districts, along the Flinders Ranges, the Sub-Protector of Aborigines reported from Blinman of the epidemic among the Adnyamathanha and Kuyani in February 1875:

> I am sorry to record the fact that in common with the settlers here and elsewhere the Aborigines have suffered extensively from measles. At the Sliding Rock [near Beltana] during one of my visits I found many suffering and instructed Mr. Powell the surgeon to attend to those in the neighbourhood of the mine professionally. Two of the Blacks - a man and woman have died in this neighbourhood [Blinman] and I have been informed that several aged Blacks at Beltana and at Numdowadana succumbed to the disorder named (Buttfield 1875).

The epidemic had probably been present among the Adnyamathanha and Kuyani since at least December (Hamilton 1876). The epidemic first appeared among the Europeans in the Flinders Ranges and afterwards spread among the Adnyamathanha and Kuyani who suffered severely (Hamilton 1876).

The epidemic was then reported to have moved into the western districts of the colony. Traditional ceremonial and trading routes linked the two regions (McBryde 1987) and most likely facilitated the spread of the epidemic. Hamilton reported that information passed to him indicated that measles made its first appearance among the Banggaria of the Gawler Ranges and from there spread west along the coast among the Nawu and Wirangu near the Venus Bay, Streaky Bay, and Fowlers Bay settlements and to southern Eyre Peninsula where it infected almost all the residents living on Poonindie mission. One resident of Fowlers Bay wrote to Protector Hamilton:

> The epidemic (measles) carried away some twenty old and young. It first made its appearance at Streaky Bay, and I hear, was brought there from Adelaide by Europeans. Some of the Fowlers Bay natives were there at the time, and caught it, bringing the disease here, and that I feel sure, was caught from the blacks (Hamilton 1876).

Measles was also reported among the Buandig at Guichen Bay in the colony's southeast. Police Trooper Morris reported to Protector Hamilton:

> There has been much sickness among them than usual, owing to the prevailing epidemic, measles and scarlatina; but they have all without exception got over it favourably; it has necessitated me issuing them extra blankets and medical comforts. The two deaths which have occurred here have been caused by disease of the lungs, by which they all seem more or less affected (Hamilton 1876).

Little could be done for those infected. Protector Hamilton supplied the Aboriginal depots in the infected areas with 'medicines specially prepared, and medical comforts' (Hamilton 1876) but with little effect. Some accepted the limited treatment and help from those Europeans settlers who offered it to them; but in many cases the Aboriginal people refused treatment, preferring their own traditional healers and methods.

Only one was treated for measles in the Adelaide Hospital in 1875, a one year old female who was admitted for treatment on 30 November (SA Government Gazette 1875; 1876).

The measles epidemic bypassed the Point McLeay Aboriginal Mission on the Lower Murray River, and the Nukunu living in the Port Augusta region at the head of Spencer Gulf. Taplin (1876), writing from Point McLeay in January 1876 after the epidemic had subsided stated:

> We never had the measles on the Station at all altho [*sic*] settlers had them within two miles of us and they raged violently at Milang and Meningie. Altho a large number of natives were gathered in camps at a sheep shearing and some of them, mostly half castes had measles, yet they did not spread generally. This is surprising since absolutely no care was taken to prevent infection.

In his half-yearly report on the Aboriginal people at Port Augusta, Police Sergeant O'Shanahan stated quite specifically that they had 'not suffered in any way from measles' (Hamilton 1876).

The failure of the epidemic to affect the Point McLeay settlement and the Aboriginal population of Port Augusta, even though no measures were taken to prevent infection, may indicate that many had been exposed to the virus during an earlier outbreak and had gained immunity to the present epidemic. The epidemics of 1861-62 and 1868 which reached South Australia may then have been the source of original exposure and protection from the 1874-75 epidemic. Although there is little evidence to support this Taplin (1876), in the same report does state that measles had been 'very prevalent' at some time in the past.

Although most sources reporting on measles indicate severe morbidity and many deaths, there are few statistics available giving a more precise indication of the

degree of mortality. Hamilton (1876) reported an exceptionally high death rate among Aboriginal people in the colony for 1875. Out of 140 deaths recorded from all districts, 55 (39.3%) were attributed directly to the measles epidemic. On Poonindie Mission 75 cases of measles were reported with 10 deaths (a case fatality rate of 13.3) in a population of approximately 90 (Brock & Kartinyeri 1989); at Fowlers Bay 20 people, both old and young, died (Hamilton 1876). These figures can only be accepted as an approximate at best, as many other deaths would have gone unnoticed and unrecorded, particularly in the Flinders Ranges among the Adnyamathanha and Kuyani, and in the western districts of the colony among the Banggarla and Nawu. No evidence is available for the desert regions of the colony further north and west of established European settlement.

There were five succeeding epidemics of measles in Southeast Australia during the nineteenth century. Apart from just a handful of reports mentioning measles there is little other evidence of the disease affecting the Aboriginal people. In 1887 at Lake Wellington Aboriginal Settlement, Hagenauer (1887) reported one case of measles which did not spread to the rest of the community. During the epidemic period of 1893-94, measles was reported among the populations (possibly the Bundjalung and Gumbainggiron) on the north coast of New South Wales:

> There are now fifty Aborigines in residence at the [Aboriginal] "Home" near Grafton. The health generally has been good during the year. Measles and influenza went through the settlement, but all the patients recovered, though several died of the same complaints in camps in other parts of the Clarence district (Frosby 1893).

Measles accompanied by respiratory disease was also reported in the New South Wales southern highlands at Tumut (Frosby 1893) and on the Point McLeay Aboriginal mission, South Australia in 1894:

> The epidemics of influenza and measles have been prevalent at the [Point McLeay] station, and this cause may have attributed, to a certain degree the comparatively heavy mortality over the year (*South Australian Register* 1894).

The extent of mortality from these epidemics is unclear. There were 19 deaths recorded among the Aboriginal people in the South Australian colony for 1894; 7 children, 2 youths, and 10 elderly. Eight of these appear on the register of births and deaths for Point McLeay Mission and 1 for Point Pearce Mission, but measles as a cause of death was not attributed to any.

The last epidemic of the nineteenth century reached the Arabana in South Australia's north in 1898. This was probably their first exposure to measles. The Sub-protector of Aborigines, F.J. Gillen, described the epidemic:

> The general health of the Aborigines was good until the month of December, when measles, first appearing at Oodnadatta, rapidly spread through the tribes, causing much suffering and a number of deaths, chiefly among the old people. Only such deaths as have occurred at or near, the depots have been recorded, and it is therefore reasonable to suppose that the mortality in outside districts, where the aborigines have no opportunity of getting medical treatment or advice, was much heavier. The disease, which is often complicated by pleurisy, is still raging, and will doubtless spread from tribe to tribe throughout the continent. Where the sufferers have access to a waterhole their favourite method of treatment is to plunge into the water for the purpose of cooling their bodies, and this very often proves fatal (Gillen 1898).

Although this last epidemic in the nineteenth century occurred in the European populations in all the colonies of Southeast Australia, the extent of its presence among other Aboriginal population centres is unknown. The records of death from Aboriginal settlements in Victoria and South Australia record no mortality from measles, nor do they report its presence among the residents. It would appear that the settlement Aboriginal population in those two colonies escaped the epidemic for that year.

It should be noted, however, that the 1898 measles epidemic was not the last among Aboriginal populations. Although outside the temporal and geographical scope of this study, reference should be made to two further epidemics which occurred among Aboriginal populations in the desert regions of central Australia in 1948 and 1956. During this time the Pitjantjatjara an Yanykunytjatjara populations were experiencing a period of intensifying colonisation from missionaries, mining companies, and pastoral activities (Goodall 1994: 57). At Ernabella in 1948 over 300 were suffering severe febrile symptoms and acute pneumonia after being infected with measles virus. Goodall (1994: 75, note 16) suggests a conservative estimate of between 25 and 33% mortality among the two populations with all ages being affected. The second epidemic in 1956 affected mainly children under the age of 9 years presumably with no acquired immunity to the virus. The 1948 epidemic was then most likely the first exposure these people had to measles.

7.5 Discussion

Little of substance can be said on the extent of Aboriginal mortality due to measles epidemics prior to 1874-75. The historical documentation presented above shows that the disease was present among the Aboriginal populations in Southeast Australia during the three major epidemic periods of 1835, 1853-54, and 1867.

Just what the levels of morbidity and mortality were, and the extent of their spatial distribution in these early epidemics is a matter of conjecture. Most of the Aboriginal deaths from measles went unrecorded or unnoticed by European observers and only indirect and anecdotal evidence survives. During the 1834-35 epidemic some impression of the extent of mortality can be reckoned from reports concerning population loss in the settled districts between Sydney and Newcastle (Lake Macquarie). The

Reverend Threlkeld noticed a sharp decline in the number of Aboriginal people he was able to contact after the epidemic:

> During the present year [1837] I have attempted to carry into effect the plan contemplated...of endeavouring to meet the Aborigines in the neighbouring districts; but the numbers are now so very much reduced, that it is almost impossible to form any settled plan to assemble them at any given time or place. Sometimes two or three are seen, at most, half a dozen...

Although such a severe population loss could have been caused by many factors other than measles it comes at a time immediately following an epidemic. In a population that had no previous experience with measles and no herd immunity to it, high morbidity and high mortality are to be expected (Cliff & Haggett 1985; McNeill 1976; Neel *et al.* 1970: 418). In this early stage of colonisation (Stage II of the medical model, Table 2.2) the Aboriginal populations of Southeast Australia had not been fully institutionalised and confined to designated settlements. Traditional lifestyles and economies were still present among many groups living away from major European centres and beyond the frontiers of colonial settlement. While these groups could not maintain measles endemically, they may have facilitated a chain of infection enabling dissemination of measles and the relocation of epidemic centres throughout regions beyond the European frontiers.

It is also quite likely that differential disease experience occurred, with some receiving the full force of the epidemic and others escaping the infection entirely. Linguistic and socio-cultural differences between groups would have acted as permeable or inviolable barriers restricting the spatial dissemination of the virus. Physical obstacles such as rivers, lakes, and mountain ranges would also have affected the spread of the disease, again acting as barriers slowing, shaping or even stopping the process of diffusion. Those groups that escaped one epidemic would then, however, have been equally susceptible to the next. Whatever the case may have been, it would be expected

that the dissemination of the epidemic was wide and severe population loss would have been experienced in the regions affected by the first three major epidemics leading up to 1874-75.

By the second half of the nineteenth century, Aboriginal population centres were not large enough, or closely associated enough, to keep the measles chain of infection going, enabling it to spread throughout most of Southeast Australia and remain endemic. However, during at least the 1875 epidemic the Aboriginal population did not need to be large enough to sustain an epidemic. The chain of infection was easily supported by the European population which, with its high numbers of immunologically susceptible, maintained the active disease at low incidence levels peaking periodically into epidemics. It is safe to assume then that because of the highly communicable nature of the virus and the associations between Europeans and Aboriginal people, the virus would have crossed repeatedly from the former population to the latter. It is most likely that the disease spread in this way throughout the Aboriginal populations of Southeast Australia.

For the 1874-75 epidemic, local death at a local rates on Aboriginal settlements in Victoria and South Australia can be estimated. Table 7.6 compares the Australian estimates of crude death rate (CDR) with those from Methodist Church centres in Fiji (Cliff & Haggett 1985) for 1875. The Fijian epidemic can be considered a trans-Pacific extension of the 1874-75 epidemic experience in Southeast Australia. While the crude death rates for Australian Aboriginal people are in most cases lower than those in Fiji they share a similar pattern of widely varying mortality between one population centre and another.

Table 7.6 Estimated crude death rates (CDR) from 1874-75 measles epidemic among Fijians (after Cliff & Haggett 1985) and Aboriginal Australians.

Centre	CDR	*n*
Australia		
Coranderrk	190	27
Lake Condah	47	4
Framlingham	103	6
Ebenezer	205	16
Poonindie	111	10
Point McLeay	0	0
Fiji		
Totoyo	137	75
Komo	192	20
Matuku	229	162
Lakemba	258	210
Moala	259	205
Fulaga	342	107
Kabara	363	107
Moce	383	59
Namuka	435	54
Oneata	755	120

There are two significant epidemiological points to consider arising from the figures of Table 7.6. The first is that the crude death rates among the local Fijian populations are generally all higher than those of the Australian Aboriginal populations. The exceptions are Coranderrk (CDR 190) and Ebenezer (CDR 205) which have higher crude death rates than those of Totoya (CDR 137) and Komo (CDR 192).

Two explanations may be offered for this. Firstly, the local populations are smaller for Australia and the crude death rates may be influenced by a small sample size. If this were the case, however, it would be expected that the bias would show random death rates both above and below that of Fiji. This does not occur.

A second explanation, and one which goes towards addressing the former problem, is that the low crude death rates among the Australian Aboriginal population when compared to the Fijian samples reflect the true nature of the epidemic. Prior to this epidemic, Aboriginal people in Victoria and South Australia had been exposed to three previous epidemics (see Table 7.1); twenty years, fifteen years, and eight to ten years before. An unknown number of Aboriginal people living through the 1874-75 epidemic had presumably been infected in one of the previous epidemics, survived, developed immunity to the virus, and so did not fall to the disease on its return in 1874-75. A small herd-immunity to the disease would have then existed. This was not the case for Fiji in 1875. This epidemic was the first experience the Fijians had with measles. Measles had spread from Australia into the Pacific in 1854 when it reached Tahiti and the Cook Islands (probably from an American ship on her way from Newcastle in New South Wales), but it did not reach Fiji (Cliff & Haggett 1985). The Fijians can be considered an island population with no herd-immunity to measles in 1874-75, whereas in Australia for the same period there was at least some degree, albeit unknown, of herd-immunity to the virus. The differences in the crude death rates between the two shown in Table 7.6 then are explained.

This explanation then raises further questions. If, as I think we can safely assume, there was no endemic measles in Australia prior to European settlement, and if the first recorded epidemic in 1834 was the initial exposure to the virus for the Aboriginal population, was the crude death rate in 1834 much higher than in 1874-75; and was it as high as seen for some areas in Fiji? There are of course no solid answers to these questions, but we can speculate by epidemiological inference, in this case the subsequent measles epidemics in Fiji. When a second epidemic of measles was introduced into Fiji in 1903 there was a much reduced mortality. Although there are no official records on the timing or extent of this epidemic, the estimated number of deaths was between 1,800 and

2,000 (Cliff & Haggett 1985: 40; McArthur 1967: 32). Based on a higher than average crude death rate for all deaths for Fiji in 1903, McArthur (1967: 32) estimates the crude death rate from measles for that year at approximately twenty per thousand. The reason for this much reduced figure can be explained by a higher herd-immunity at the time of the population's second exposure to the virus. The majority of the population who survived the first epidemic would have acquired an immunological protection from the virus and would have escaped the symptoms of the disease. The section of the population at risk to measles in 1903 comprised those born after the 1875 epidemic, that is those under the age of twenty-seven. A substantially reduced number of susceptible individuals would then account for the reduced crude death rate. There is of course nothing new in this. It follows the well-known epidemiological pattern of measles epidemics occurring throughout the world and their effect among virgin-soil populations (Adels & Gajdusek 1963; Black 1966; Neel *et al.* 1970).

We can then, I believe, assume by analogy with Fiji that the initial epidemics of measles among the Aboriginal people of Southeast Australia were responsible for a much higher crude death rate than those calculated for the 1874-75 epidemic. This assumption can be strengthened further if we take into consideration the supportive care available to the Aboriginal people living on missionary and government settlements during the 1874-75 epidemic, and the lack of it among those infected in previous epidemics. Whether one dies from measles largely depends on the availability of supportive care and the quality of hygiene and nutrition that is maintained throughout the course of the primary and secondary illnesses (Cohen 1989: 54). It is therefore important that the ill person receives care from those not infected or not immobilised from the disease. In a virgin-soil population with all susceptible to the infection, typically, many individuals of all ages are sick at once, and there is often no-one to provide for the basic needs (food, water, comfort) of the sick or attend to basic hygiene. Such a situation was

almost certainly the case among Aboriginal groups living beyond the European frontiers when they experienced the first epidemics of measles. The mortality from measles would thus have been reduced by the supportive care offered to the Aboriginal people living on settlements during the 1874-75 epidemic by those who were not incapacitated by the diseases. The crude death rates from the 1874-75 epidemic are then from localised populations who have developed a measure of herd-immunity to the disease and who had access to supportive care, food, water during the epidemic; all of which would have reduced mortality from measles.

The second epidemiological point involving the local level crude death rates presented in Table 7.6 is their irregularity, and by inference, the irregularity of the attack rates of the virus. A number of factors operating in conjunction are responsible. Unlike the influenza virus which undergoes regular and major genetic changes the measles virus is a stable myxovirus (Neel *et al.* 1970: 118). Therefore on the basis of present virological knowledge we can accept that the measles virus attacking a population centre during an epidemic is genetically the same as that which attacks another centre. The same can be said for epidemics several decades apart. For the same reason lifelong immunity to the virus is gained after the first infection. Further, the virus has a simple transmission mechanism. It is transmitted primarily from one human to another and requires no intermediate host or vector to intercede. Attack rates then depend on the amount of exposure each individual has to the virus and on their personal immunological immunity to the virus.

The mortality rate is also dependent upon the ability of the immune system to fight the virus effectively. Those with impaired immune systems due to other infectious agents, particularly chronic infections such as syphilis or tuberculosis, lose the ability to combat the virus effectively and are prone to more severe and debilitating primary

symptoms. These same individuals are more susceptible to post-measles secondary invasion by other pathogens. Further to this, as we have seen, the irregular pattern of crude death rates also relate to the effectiveness of local supportive health care available during an attack.

7.6 Conclusion

The history of measles in Australia can be seen as a series of recurring epidemic events beginning in 1834. Although there is little evidence available regarding the morbidity or mortality among the Aboriginal populations during the epidemics before 1874-75, there is evidence to suggest a similar pattern in the timing and periodicy of measles epidemics between the Aboriginal population and the European population in Southeast Australia. Excessive morbidity and mortality during these early epidemics on immunologically vulnerable Aboriginal populations could be expected. The few surviving reports referring to these early epidemics suggest that this is the case.

The 1874-75 epidemic in Southeast Australia was a part of a world pandemic of measles. Records of this epidemic provide the best evidence regarding the morbidity and mortality of measles among the Aboriginal populations. Historical accounts show that this epidemic spread throughout many Aboriginal population centres living both on, and away from, established settlements in Victoria and South Australia. The hardest hit settlements in terms of mortality caused by the disease were Lake Hindmarsh which lost 20.6% of its population and Coranderrk which lost 19%. Other population centres were bypassed by the epidemic. The settlements of Lake Wellington and Lake Tyers on the east coast of Victoria, Point McLeay in South Australia, and the population living in the European town of Port Augusta escaped the epidemic with no recorded morbidity or mortality.

The crude death rates from the 1874-75 epidemic, while indicating excessive mortality from measles, are much less than those from Fiji for the same epidemic. The experience of previous epidemics among the Australian Aboriginal population providing a substantial number of immunologically protected, can account for the lower mortality in Southeast Australia and in turn suggests greater severity of the earlier epidemics.

It cannot be ascertained whether measles became endemic among any of the Aboriginal population centres of Southeast Australia during the nineteenth century, although it is unlikely because of their small numbers and relative isolation from the main European centres.

After the 1874-75 epidemic records from Aboriginal settlements suggest that measles was no longer a major lethal disease among the Aboriginal population of Southeast Australia. An exception was the 1898 epidemic which penetrated into the Arabana regions of South Australia's north following pastoral expansion. This was the last epidemic of the nineteenth century seriously to affect an Aboriginal population.

CHAPTER EIGHT

Diseases on Aboriginal Settlements 1876 to 1900

8.1 Introduction

The previous chapters were concerned with stage two of the medical model (see Table 2.2) which dealt with the early years of contact between European and Aboriginal people and the diffusion of epidemic diseases among the latter populations in their traditional regions. This chapter will now consider stage three (settlement and acculturation) of the medical model.

The primary aim is to identify and compare the principal disease states causing mortality among the Aboriginal people living in settlements in Southeast Australia. The period concerned will be from 1876 to 1900 when a high proportion of the Aboriginal people of Southeast Australia were living on missionary and government organized settlements (Barwick 1971: 288: Broome 1995: 139; Christie 1979: 199; Jenkin 1976: 179-80; Smith 1980). Following the theme of previous chapters the focus will be placed on infectious and respiratory diseases.

Using the surviving records of births, deaths, and marriages kept by the administrators of six settlements in Victoria (Framlingham, Lake Condah, Lake Wellington, Lake Tyers, Coranderrk, and Lake Hindmarsh) and two in South Australia (Point McLeay and Point Pearce) causes of deaths will be extracted, collated and examined.

A further aim of this chapter is assess the health status of the Aboriginal populations living on the settlements. Infant mortality ratios calculated from the mortality records will be used as indicators of general health. Comparisons with other nineteenth

and twentieth century indigenous and colonial populations throughout the world will be made.

The processes of nineteenth century institutionalization of Aboriginal people, the development of settlements controlled by missionary bodies and colonial governments through Aboriginal protection boards, and the subsequent acculturation of the Aboriginal people of Southeast Australia have been the subject of previous discussions (Attwood 1989; Barwick 1971, 1972; Brock 1985, 1993, 1995; Christie 1979; Corris 1968; Foxcroft 1941; Gale & Brookman 1975; Jenkin 1979; Long 1970; Massola 1970, 1975; Rowley 1970; Woolmington 1973; Yarwood & Knowling 1982). They need not be repeated in detail here other than to discuss factors relevant to the themes of this thesis, especially those predisposing the Aboriginal people to disease.

The settlements, established to contain and confine Aboriginal people, were often established on land not immediately required or wanted by Europeans and out of the way of main European settlements (Summers 1986: 305). Although in many cases the land was unsuitable for agriculture, the managing authorities and the colonial governments expected the settlements to become largely self-supporting; efficient in growing crops and grazing sheep and cattle for their own consumption to supplement the government rations, thereby reducing the Government's financial commitment (Long 1970: 15).

The result was that food shortages were common (Barwick 1972; Jenkin 1976, 1979). Barwick (1972), discussing the mission settlements in Victoria, states that before the food ration was doubled in 1875 the men who were able to work subsisted on 5 pounds of flour, 1 pound of sugar, and 2 ounces of tea per week, while others had to do with even less. By the last quarter of the nineteenth century, traditional game species had

became scarce around the settlements and could no longer be relied on as satisfactory supplement for the inadequate rations. The able bodied men often had to disperse seasonally to find paid work in order to buy food as well as providing capital for the development of their settlement. Barwick (1972: 29) describes the meat ration provided to the Aboriginal people on Corandederrk settlement as meagre and always causing discontent among the residents.

> ...from 1871 'regular workers' were allotted one pound of meat per day and other men half that amount. From 1875 to 1877 all adults were entitled to six pounds per week with half rations for children, but for the next five years the Board expected residents to purchase meat, increasing wages on a sliding scale according to numbers of dependents, and gave rations only to the aged and ill.

This economic measure imposed by the settlement supervisors was the cause of real hardship for the residents, reducing their ability to purchase other necessities.

Although living conditions varied between the settlements and over the time of their operation, funding by the responsible missionary bodies and government agencies was almost always inadequate and failed to maintain long-term satisfactory housing and conditions (Barwick 1972; Jenkin 1976, 1979; McCrea 1879). Some Aboriginal families had huts or cottages. They were often of one or two rooms, and while adequate for a childless couple they were often inadequate when family size increased with surviving children (Brock 1993: 18) or when required for accommodation of extended family groups. The Aboriginal occupants were frequently required to meet the costs of maintenance themselves and with limited financial resources, maintenance requirements were often neglected (Barwick 1972). Not all those who lived on settlements had huts or cottages. Children often slept and ate communally in classrooms and dormitories (McCrea 1879). Single males and females were often required to sleep in segregated dormitories. Even by 1876, despite the passing of several years after their establishment,

some settlements could only offer bark shelters which the Aboriginal people constructed themselves (Jenkin 1976: 180). In 1876 the Chief Medical Officer of Victoria reported after a visit to Coranderrk which at that time had 32 huts:

> The huts of the natives are built on a street which runs nearly north and south across the face of the slope, instead of up and down it, the natural advantage of the fall of the ground for drainage being thus lost... The construction of the huts is, in a sanitary point of view, the worst possible; the walls are of slab, paling, or bark, mostly the latter, with openings in them so numerous that they may fitly be compared to bird cages. The roofs are almost always bark, with openings nearly as numerous as in the walls; some of the natives have endeavoured by paper and bags to cover the chinks and openings in the walls and roofs, but in most cases with little effect. The floors are of clay, and are damp even at this the driest season of the year, whilst in the winter the natives complain that the water rises to the surface of the floors after every shower of rain. In wretched hovels like these it is no wonder the mortality is excessive (McCrea 1879-80).

The sanitary and human waste disposal facilities on the settlement were reported to be little better:

> There are few closets of a common description, covering mere holes in the ground, scattered about irregularly over the establishment, insufficient in number for the wants and decency of the population [approximately 150]. Even these are not always used, the Superintendent stating that at night the children and some of the adults pass their excrement in the water channels in the street opposite their huts (McCrea 1879-80).

Few if any of the settlements had a resident doctor and medical needs were either met by the superintendent or by local medical practitioners who were called on to attend ill Aborigines. Lack of sufficient money often severely reduced the number of times doctors were called to the settlements. Phillip Pepper, writing his family's history centred on Lake Wellington and Lake Tyers settlements, remarks:

> Once the reserves were established it became obvious that doctors would have to be called in and the managers were authorised to seek medical assistance if required. Over the years their accounts were often questioned and the

> managers were told to find out from the doctors exactly what services they had rendered. As a result some doctors reduced their fees and many did not charge for visits (Pepper & De Arago 1985).

Despite the willingness of some doctors to forego full fees for their attendance, the Aboriginal Protection Board of Victoria saw the costs of medical assistance as too high. In 1886 they advised settlement managers that they could not call a doctor without due authority, unless the patient in their opinion was going to die (Pepper & De Arago 1985: 203; BPA 185-86). The result was that the limited medical care made available to the Aboriginal inhabitants could not meet their increasing health needs. Alcohol and tobacco consumption was high throughout the settlements adding further to the health hazards faced by the Aboriginal people.

While these conditions may be considered to be extreme they were common throughout the settlements in various degrees of severity for much of the last quarter of the nineteenth century. The state of the settlements in Southeast Australia during the nineteenth century and living conditions on them can be summarised as:

- Often small settlements (with the exception of Point McLeay)
- Located on poor land, often infertile for exotic crops, often badly drained
- Poor facilities to deal with health problems
- Few of the staff were properly trained to deal with a different culture
- Poorly constructed and maintained houses - often cold in winters and hot in summers; unsuitable for Aboriginal lifestyles
- Few services provided - fresh water and sanitation were minimal or entirely lacking
- Diet nutritionally inadequate
- Communal eating causing cross-infection.

(Barwick 1972; Brock 1993: 48; Broome 1982; Jenkin 1976, 1979, Long 1970: 15-23; Massola 1970; Pepper & De Arago 1985).

In attempts to convert the Aboriginal people to Christianity on the settlements traditional cultural practices were actively discouraged by settlement superintendents. The erosion of long-existing social structures by the frequent deaths, and the mixing of different socio-linguistic groups within the confines of the settlements caused further anxiety to many residents. Against this backdrop was the persistence of introduced infectious diseases and nutritional disorders many of which had become endemic.

8.2 Source materials

Records of Aboriginal births, deaths and marriages during the last quarter of the nineteenth century (1876 to 1900) were examined from eight Aboriginal settlements - six in Victoria and two in South Australia (Figure 8.1, Plate 8.1).

The period 1876 to 1900 was selected for examination of stage three of the medical model for a number of reasons. The period 1876 to 1900 saw a high proportion of the surviving Aboriginal population of Southeast Australia institutionalised on settlements. In 1876, 46% of the Victorian Aboriginal people enumerated in that year's census lived on the six settlements to be examined below (Broome 1995: 139; VPP 1877-78). By 1901, at the end of the period of examination, the figure had increased to 65% of the enumerated population (BPA 1901).

The proportion of the Aboriginal population in South Australia institutionalised during the period of concern is difficult to quantify from official census

statistics mainly due to the difficulties encountered by census official in reaching and counting the Aboriginal populations, particularly in the northern and western regions of the colony. In 1875-76 George Taplin, the superintendent of Point McLeay, completed a census of Narrindjeri showing 511 living on the lower Murray River, Lakes Alexandrina and Albert, and the Coorong (Hamilton 1876; Taplin 1876a). The Point McLeay settlement on Lake Alexandrina had a population of 135, or 26%, of the Narrindjeri. By 1896 the average population of the settlement and its nearby out-station 'The Needles', was 234 with few of the Narrindjeri living continuously on traditional lands (*South Australian Register* 1896). The Point Pearce settlement on Yorke Peninsula averaged 52 residents in 1878 (Hamilton 1879). The Aboriginal population on the peninsula was not enumerated in the 1876 census, but a figure of 439 was given for the Western District Pastoral Region (*South Australian Government Gazette* 1877a: 798). Many of these lived far from the influence of the Point Pearce settlement. By 1890, despite severe depopulation of the Western District, the population of the settlement was still roughly the same at 56 residents (Sutton 1890). The number of Aboriginal people living in the colony's northern and eastern pastoral regions was calculated in the 1876 census to be approximately 3,500 (*South Australian Government Gazette* 1877a). Distance would have precluded all but a few of these people having contact with the two settlements of Point McLeay and Point Pearce. Throughout the remainder of the nineteenth century the colonial governments maintained their policies of moving Aboriginal people (sometimes by force) on to settlements and the numbers living traditional lifestyles were steadily reduced (Barwick 1971: 288-290; Broome 1982: 73; Corris 1968; Foxcroft 1941; Long 1970; Saggers & Gray 1991: 69; Yarwood & Knowling 1982:162-3).

With many of the Aboriginal people living on settlements enumeration and the recording of vital statistics became more precise. It was from the beginning of this period that the most accurate official nineteenth century records of Aboriginal mortality

and birth were kept (Barwick 1971: 302). Each of the settlements examined was required by the colonial government to keep a register of births, deaths and marriages and to furnish quarterly and annual reports of the same. These records are today available from several sources. Massola (1970 & 1975) collected and published a list of births and deaths for Victorian Aboriginal settlements drawing on their original registers of births, deaths and marriages from the institutions and from those included in the quarterly and annual reports of the Aborigines Protection Board presented to the Victorian Parliament. The records of deaths from Lake Condah, Lake Wellington (Ramahyuck), Lake Hindmarsh (Ebenezer), and Coranderrk cover the full period 1876-1900. Records from the Victorian Government's Framlingham station cover the period 1876-1883. The original records of births, deaths, and marriages for Point McLeay mission station are housed in the State Library of South Australia and extracts of these together with extracts of the register from Point Pearce mission (Gale n.d.) are available in the library of the Australian Institute for Aboriginal and Torres Strait Islander Studies, Canberra. The original registers of births, deaths and marriages from Lake Tyers, held in a private collection, were made available for this study. Table 8.1 lists the selected settlements, the respective temporal distribution of mortality data, and the abbreviations used for each below.

Table 8.1 Aboriginal settlements in Victoria and South Australia for which there are reliable nineteenth century mortality data.

Aboriginal settlement	Abbreviation	Time period
Victoria		
Framlingham	FRM	1876-1883
Lake Condah	LCD	1876-1900
Lake Wellington (Ramahyuck)`	LWL	1876-1900
Lake Tyers	LTY	1876-1900
Coranderrk	CRD	1876-1900
Lake Hindmarsh (Ebenezer)	LHD	1876-1900
South Australia		
Point McLeay	PMC	1876-1883
		1888-1900
Point Pearce	PPR	1880-1900

A total of 714 Aboriginal deaths were identified and extracted for the period (Table 8.2). For a detailed listing of each settlement see Appendix B.

Table 8.2 Number of deaths recorded on selected Aboriginal settlements in Southeast Australia for period 1876-1900.

Settlement	Number of deaths recorded
Victoria	
Framlingham	18
Lake Condah	96
Lake Wellington (Ramahyuck)	89
Lake Tyers	93
Coranderrk	71
Lake Hindmarsh (Ebenezer)	82
South Australia	
Point McLeay	223
Point Pearce	42
Total	**714**

8.2.1 Nature of the data

Deaths were recorded by the various superintendents of the settlements and medical practitioners who had diagnosed either the cause of their death or the major clinical signs leading up to death. The individuals whose deaths were recorded had for some period of their lives lived on designated settlements under the control of colonial governments or missionary bodies (Plate 8.2). There is little doubt, however, that many individuals living and dying in the last quarter of the nineteenth century never became fully institutionalised. The Aboriginal populations of central and northern South Australia were among the last to move from their own lands onto settlements, many during the twentieth century (Brock 1995: 220). Others in both South Australia and Victoria lived on the settlements for only short periods during the year, coming and going largely at will (Brock 1995; Broome 1995; Jenkin 1979). The demographic circumstances of these people are lost to historical record. Others spent only limited times away from the settlements, working for European employers on a seasonal round (Broome 1982: 73, 1995: 139). The sample used in this study therefore does not take into account Aboriginal deaths that occurred off the selected settlements and which were not recorded in the death registers. Nevertheless, because of the temporal range used (25 years) it takes into account over 700 deaths of Aboriginal people who lived most of their lives on the settlements or closely attached to them, and so provides a statistically useful sample.

The sample under consideration comprises residents of eight localised settlements, each with fluctuating numbers caused by migration on and off the settlements and between them. Individuals in this sample were exposed to chronic disease and poor nutrition. It should also be noted that the twenty-five year period (1876-1900) under examination does not cover the complete life-spans of all individuals. Many individuals whose deaths are recorded in the time period would have been born and lived a greater or

lesser part of their lives outside the period (before 1876). The same goes for those who were born within the period but whose deaths are not considered as they died after the period ended (after 1900). Others in the sample would, however, have lived and died (before the age of 25 years) within the 1876-1900 period.

After the 'Aborigines Act of 1886' the population structure changed on Victorian settlements. The Act differentiated the Aboriginal populations throughout the colony into 'full bloods' and 'half-castes'. Those determined as 'full bloods', 'half-castes' over 34 years of age and their 'half-caste' wives and children were allowed to remain on the settlements supported by missionary and government funding. 'Half-castes' under the age of 34 years were either forced to move into the general community or required special licences to reside on the settlements. The Act caused great distress among the Aboriginal population, isolating individuals and splitting families. It resulted in settlement populations being reduced and the communities losing many of their young adults up to the age of 34 years. For instance, the population of Framlingham fell from 94 to 35 (a reduction of 63%) after the Act while that of Coranderrk fell from 120 to 60 (a reduction of 50%) (Broome 1995: 139-140).

Such a sample would have inherent problems in some forms of demographic analysis (e.g. life tables) yet the causes of death derived from it and analysed in the form of ratios and rates, can prove extremely useful in making assessments of the principal diseases occurring among the inhabitants of the settlements.

8.3 Methods of analysis

The sex and age at death of each individual death were noted where recorded. It was often the case, however, that the sex of the individual went unrecorded in the

registers. When this occurred, sex could sometimes be determined by the European name given. If no name, or an Aboriginal name only were recorded, the sex of the individual remains undetermined. In the entries where age at death was not recorded attempts were made to trace the individual back through the register of births. This proved difficult for a number of reasons. Many deaths with no recorded age also had no recorded names, making a check of birth impossible. European names were sometimes untraceable because the same name was given to more than one child. Finally, many of those who had their deaths recorded did not have their births recorded. In some cases, however, where age was not given in the records the individual could be assigned an age range based on the recorded cause of death. For example, 'old age', or 'senile decline' as causes of death were allocated to the 15+ age range. This method reduced redundancy. Of the 714 individual deaths extracted, 632 (88.5%) had sex, and cause of death recorded.

Table 8.3 displays a breakdown of the data.

Table 8.3 Breakdown of data set - extracted from records of Aboriginal deaths between 1876-1900 on selected settlements in Southeast Australia.

	No.	**Per cent**
Sexed, aged, cause of death	632	88.5
Unsexed	45	6.3
Unaged	18	2.5
Unaged & unsexed	1	0.1
Sexed, aged, no cause of death	14	2
Sexed, unaged, no cause of death	1	0.1
Unsexed, aged, no cause of death	3	0.4
Total	**714**	

Where the cause of death was given for an individual it was noted and grouped according to the International Classification of Diseases (ICD.9.CM 1991).

Particular attention was needed when ascribing causes of mortality given in the registers to a particular ICD group, but in most cases (81%) the recorded disease was unambiguous and classifiable according to specific groups. The Nosological index of 1863 (Morgan 1987) was used as a guide when specific diseases were given more than one name (e.g. 'phthisis', and 'consumption' were listed as tuberculosis). In the other remaining cases, however, the recorded cause could only be classified under symptoms, signs, and ill-defined conditions (ICD 780-799). For example, 'senility', 'general decay', and 'old age', given as causes of death are closely linked to the longevity of the individual, and while contributing to death, cannot be ascribed as the cause. Likewise, causes such as 'weakly child', 'general debility', and 'decline' which are ambiguous were included in the ill-defined category. In all 121 (19%) of all the causes of death had to be ascribed to the ill-defined category making it statistically the third highest cause of death.

Table 8.4 lists the ICD to which the recorded causes of death have been assigned and the abbreviations used for the purpose of this analysis. A detailed breakdown of each cause of death and the ICD category in which it was placed is in Appendix B.

Table 8.4 International Classification of Diseases categories and abbreviations used (ICD.9.CM 1991).

Abbreviation used	ICD Group	Diseases
RES	460-519	Diseases of the respiratory system
INF	001-139	Infectious and parasitic diseases
CIR	390-459	Diseases of the circulatory system
DIG	520-529	Diseases of the digestive system
NEO	140-289	Neoplasms
INJ	800-999	Injury and poisoning
GEN	580-629	Diseases of the genitourinary system
PRG	630-676	Complications of pregnancy, childbirth, and the puerperium
END	240-279	Endocrine, nutritional, and metabolic diseases and immunity disorders
NER	320-389	Diseases of the nervous system and sense organs
PER	760-779	Certain conditions originating in the perinatal period
ILL	780-799	Symptoms, signs, and ill-defined conditions

Before the causes of death from the eight settlements were combined the frequency of each ICD category was compared to detect any differences in their occurrence. A multi-variate statistical technique, correspondence analysis (CA), was chosen to compare the frequency of each of the ICD groups in each of the settlements. A null hypothesis proposed that there was no difference between the settlements in the frequency of reporting of the various causes of death. In other words, all the settlements were expected to show a similar pattern of causes of death throughout the twenty-five year period, 1876 to 1900.

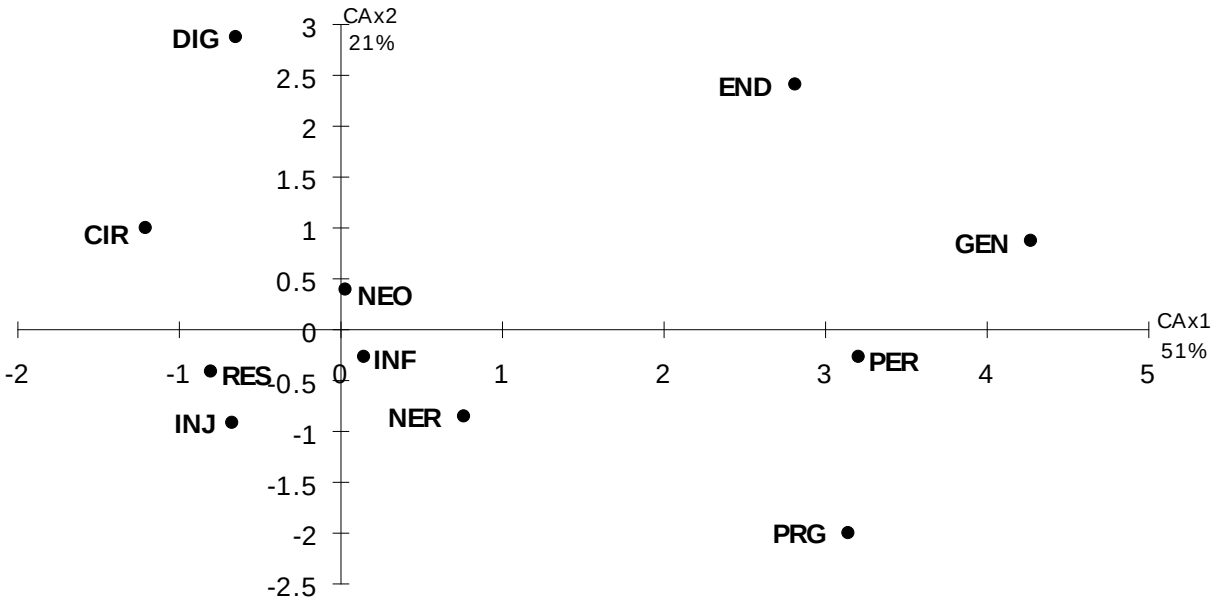
Correspondence analysis is particularly suited to the analysis of tables expressed as raw counts (in this case the number of times each disease group was reported as a cause of death) where some of the objects (settlements) have a greater abundance than others. In Appendix B it can be seen that the counts of different disease groups vary considerably within each settlement and also between each settlement. There

are quite a number of very small or zero counts and some large ones. Correspondence analysis is designed to cope with such a data set and gets rid of any general factor of abundance of counts (Wright 1992, 1994).

Figure 8.2 displays the relationship of ICD disease groups. There are five groups - respiratory diseases (RES), infectious diseases (INF), circulatory disorders (CIR), neoplasms (NEO), injuries (INJ), and nervous system disorders (NER), forming a cluster around the centre of the two axes. As will be shown below respiratory diseases and infectious diseases are the most commonly reported groups on all settlements (Table 8.5, see also Appendix A). In correspondence analysis the nearer any variable is towards the centre of the territorial map the less it contributes towards the overall differences between the objects (Wright 1992, 1994). The clustering around the centre of the territorial map (particularly that of respiratory and infectious diseases) can be interpreted as a comparable pattern of reporting for each group of diseases throughout the settlements with neither group contributing greatly towards the overall differences between the settlements.

The remaining ICD groups are scattered along the first two principal axes indicating higher differences in frequency of reporting. At first sight this distribution seems at odds with the null hypothesis and might suggest that combining these groups would introduce a bias coming from one or more of the settlements. However, as will be

Figure 8.2 Relationship of ICD categories reported as causes of death according to their occurrences on Aboriginal settlements with respect to the first two component axes of correspondence analysis.



RES = Diseases of the respiratory system; **INF** = Infectious and parasitic diseases; **CIR** = Diseases of the circulatory system; **DIG** = Diseases of the digestive system; **NEO** = Neoplasms; **INJ** = Injury; **GEN** = Diseases of the genitourinary system; **PRG** = Complications of pregnancy, childbirth & puerperium; **END** = Endocrine, nutritional and metabolic diseases; **NER** = Diseases of the nervous system; **PER** = Conditions originating in the perinatal period; **ILL** = Ill defined conditions.

shown below (Table 8.5, also see Appendix B), these groups have low frequencies of reporting on all the settlements and would therefore contribute little to the combined rate of reporting. The null hypothesis stating that there is no difference in the frequency of the various causes of death among the settlements can be supported at least for the two major disease groups - infectious and respiratory. These groups will be the major focus of the following discussion.

8.3.1 Proportional Mortality Ratios (PMR)

In order to ascertain the major diseases and their impact on the Aboriginal residents of the settlements for the period 1876-1900 proportional mortality ratios (PMR) have been calculated for age ranges 0-4, 5-14, and over 15 years of age. Proportional mortality is calculated:

$$\frac{\text{Number of deaths from a particular disease per region per time}}{\text{Total number of deaths per region per time}} \times 100$$

The PMR does not directly measure the risk or the probability of a person in a population dying from a specific disease. Rather, it is a measure of the relative importance of a particular disease within a population over a period of time (Lilienfeld & Lilienfeld 1980: 74).

8.4 Results and discussion

An overview of the principal mortality ratios of ICD disease categories and the major causes of death is given first. This is followed by an analysis of the principal causes by age ranges 0 to 4 years, 5 to 14 years, and 15+ years.

Table 8.5 Proportional mortality ratios (PMR) for disease categories on selected Aboriginal settlements in Southeast Australia 1876-1900.

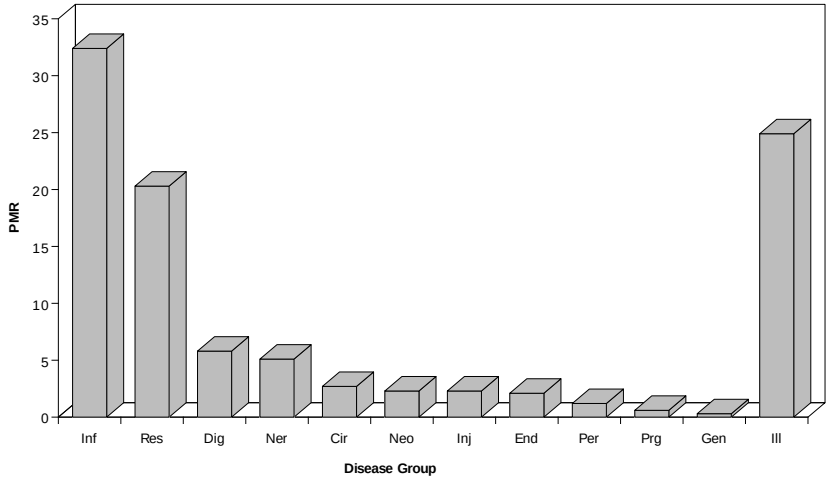
Group	n	PMR
INF	238	32.4
RES	143	20.3
CIR	19	2.7
DIG	41	5.8
END	15	2.1
GEN	2	0.3
INJ	16	2.3
NEO	16	2.3
NER	36	5.1
PER	9	1.2
PRG	4	0.6
ILL	175	24.9
Total	**714**	

Inf = Infectious and parasitic diseases; **Res** = Diseases of the respiratory system; **Cir** = Diseases of the circulatory system; **Dig** = Diseases of the digestive system; **End** = Endocrine, nutritional and metabolic diseases; **Gen** = Diseases of the genitourinary system; **Inj** = Injury; **Neo** = Neoplasms; **Ner** = Diseases of the nervous system; **Per** = Conditions originating in the perinatal period; **Prg** = Complications of pregnancy, childbirth & puerperium; **Ill** = Ill defined conditions.

Table 8.5 shows the proportional mortality ratios (PMR) of disease categories for all age ranges combined from the Aboriginal settlements.

Figure 8.3 shows the same principal mortality ratios of the disease categories in order of their relative frequencies.

Figure 8.3 Proportional mortality ratios of ICD categories in their relative order on Aboriginal settlements in Victoria and South Australia 1876-1900.



Inf = Infectious and parasitic diseases; **Res** = Diseases of the respiratory system; **Cir** = Diseases of the circulatory system; **Dig** = Diseases of the digestive system; **End** = Endocrine, nutritional and metabolic diseases; **Gen** = Diseases of the genitourinary system; **Inj** = Injury; **Neo** = Neoplasms; **Ner** = Diseases of the nervous system; **Per** = Conditions originating in the perinatal period; **Prg** = Complications of pregnancy, childbirth & puerperium; **Ill** = Ill defined conditions.

Infectious diseases (PMR 32.4) and respiratory diseases (PMR 20.3) stand out from all others as the two major causes of mortality among the Aboriginal residents of the settlements. Apart from the ill defined category of diseases (PMR 24.9) all other identified disease groups have a PMR of less than 6 and were of substantially lesser importance as causes of mortality.

Two hundred and twenty eight deaths were recorded for the infectious disease category (INF) (Table 8.6).

Table 8.6 Frequency (%) of diseases in the infectious disease category (INF) recorded as causes of death.

Disease	n	%
Tuberculosis	143	62.4
Diarrhoea/dysentery	45	19.7
Hydatids	13	5.7
Measles	6	2.6
Unspecified fevers	6	2.6
Others	15	7.0
Total	228	

Tuberculosis infection was by far the commonest recorded cause of death (62.4%, n = 143) followed by diarrhoea & dysentery (19.7%, n = 45). Only 6 deaths were recorded from measles throughout the settlements for the period 1876-1900 (see below), which contrasts sharply with the previous mortality from this disease (Chapter 7).

One hundred and forty three deaths were recorded from respiratory diseases (RES) (Table 8.7)

Table 8.7 Frequency (%) of diseases in the respiratory disease category (RES) recorded as causes of death.

Disease	n	%
Bronchitis	53	37.3
'Inflammation of the lungs'	24	16.9
Pneumonia	23	16.1
'Congestion of the lungs'	6	4.2
Whooping cough (pertussis)	15	10.6
Influenza	11	7.7
'lung disease'	6	4.2
Other	5	3.5
Total	143	

Bronchitis (37.3%, n = 53) was the major recorded cause of death in this category. It should be noted that the cause of death 'inflammation of the lungs' may be referring to pneumonia (Morgan 1987: 27) in which case it would make the frequency of this cause of death 32.4% (n = 47). Pneumonia would then be the second most common cause of death from respiratory system disorders. The term 'congestion of the lungs' is less clear in its meaning. It could refer to chronic passive congestion of the lungs resulting from the failure of the left ventricle (Mergner & Trump 1988:254) in which case the primary cause of death would be from a cardiac disorder. On the other hand, the term could indicate a non-specific infection or disorder of the lungs causing a build up pulmonary fluid. The term 'lung disease' is also unclear and could refer to any number of specific disorders of the lungs.

Table 8.8 displays the total number of deaths and proportional mortality ratios of each category of disease by age ranges. Of the 714 deaths recorded, 18 lacked information on age at death sufficiently specific for categorization into age groups.

The ill-defined category represents 24% of the reported deaths. It is due to the vagaries of reporting the cause of death mainly during the periods of early childhood and later adulthood (Appendix B) rather than to any specific non-identified condition or conditions. It is included in the denominator for the following analysis therefore making the defined causes of death 76% of the total data set.

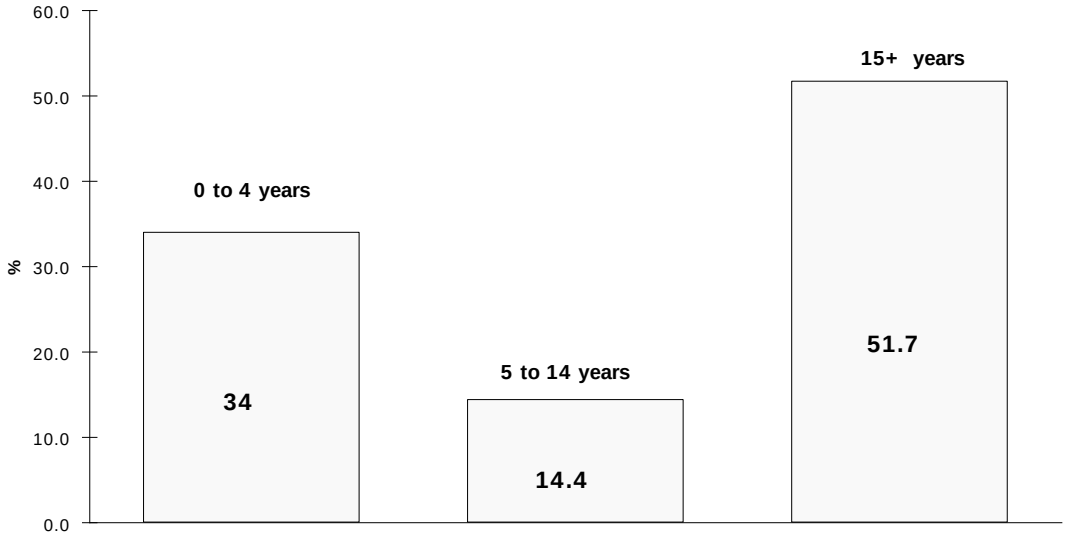
Table 8.8 Numbers of deaths and principal mortality ratios for age ranges 0 to 4, 5 to 14, and 15+ years for Aboriginal settlements in Southeast Australia 1876-1900

	0 - 4		5 - 14		15+		Total	
	n	PMR	n	PMR	n	PMR	n	PMR
INF	59	25.1	54	54.0	121	33.7	234	33.6
RES	66	28.0	13	13.0	59	16.4	138	19.8
CIR	2	0.8	0	0	17	4.7	19	2.7
DIG	4	1.7	10	10.0	27	7.5	41	5.9
END	13	5.5	1	1.0	1	0.2	15	2.1
GEN	0	0.0	0	0	2	0.5	2	0.3
INJ	4	1.7	4	4.0	8	2.2	16	2.3
NEO	1	0.4	2	2.0	13	3.6	16	2.3
NER	14	5.9	6	6.0	15	4.1	35	5.0
PER	9	3.8	0	0	0	0.0	9	1.3
PRG	0	0.0	0	0	4	1.1	4	0.6
ILL	64	27.1	10	10.0	93	25.8	167	24.0
	236		**100**		**360**		**696**	

Inf = Infectious and parasitic diseases; **Res** = Diseases of the respiratory system; **Cir** = Diseases of the circulatory system; **Dig** = Diseases of the digestive system; **End** = Endocrine, nutritional and metabolic diseases; **Gen** = Diseases of the genitourinary system; **Inj** = Injury; **Neo** = Neoplasms; **Ner** = Diseases of the nervous system; **Per** = Conditions originating in the perinatal period; **Prg** = Complications of pregnancy, childbirth & puerperium; **Ill** = Ill defined conditions.

The following discussion will centre on an examination of the causes of death in the three age ranges 0 -4 years, 5 - 14 years and 15+ years (Figure 8. 4).

Figure 8.4 Total recorded deaths (%) by age ranges for Aboriginal settlements in Southeast Australia.



Infectious and respiratory diseases were the major causes of disease in all age ranges, having a combined PMR of over 50, and will be the main focus of discussion.

8.4.1 Age range 0 to 4 years

One third of all deaths (33.5%, n =-236) were recorded for this age range (Figure 8.3). Infectious diseases (PMR 25.1; n = 59) and respiratory diseases (PMR 28.0; n = 66) were the major causes of death in the age range. There is no significant difference between these two groups (x^2 =0.392, p = 0.531, 1df).

8.4.1.2 Infectious Diseases

Diarrhoea and dysentery

Diarrhoea and dysentery are terms often used synonymously to distinguish between two clinical syndromes of diverse aetiology (Sommers 1980: 526). Diarrhoea is associated with the abnormal frequency, urgency, and liquidity of faecal discharges often accompanied by vomiting and fever. Dysentery refers to various disorders marked by intestinal inflammation and attended by abdominal cramps, tenesmus, and frequent stools containing blood and mucus (Benenson 1990: 129; Sommers 1980: 526). The two clinical syndromes often present simultaneously in individuals infected with enteric pathogens, or the symptoms can range from a mild, transient diarrhoea to full-blown dysentery. In general use of the terms, dysentery usually refers to diarrhoea with abdominal cramping and tenesmus (von Lichtenberg 1989: 355).

The two syndromes are discussed together here because of their frequent diagnostic confusion by pre-twentieth century medical practitioners. In reviewing the history of diarrhoeal diseases in Australia, Cumpston (1989: 224-5) points out that no precise opinion as to the nature of each of the syndromes can be formed.

Both terms were used loosely in nineteenth century Australian medical literature and official nomenclature to cover either or both sets of clinical syndromes, particularly when seen in infants. Both syndromes were recorded in the European populations of Southeast Australia soon after colonisation and were described as attacking all ages, but having a high mortality rate among infants (Cumpston 1989: 224-5).

The pathogenic micro-organisms responsible for the syndromes are numerous (Benenson 1990: 129-138; DuPont 1994: 678; Marcy 1976: 892-978; Mata 1980: 4; Sack: 1980:53-66; Sommers 1980: 526). Bacillary dysentery is the most frequent form among humans today and was most likely among the Aboriginal people living on settlements. Enteropathic *Escherichia coli, Shigella, Salmonella,* and *Staphylococcus* are commonly implicated and there is little reason to doubt their presence among the European and Aboriginal populations of nineteenth century Australia (Cumpston 1989: 224-225). Cumpston states there is little evidence for amoebic dysentery in Australia last century.

Table 8.9 is an extract from the mortality records showing the reported distribution of diarrhoea and dysentery reported as causes of death.

What is immediately noticeable in Table 8.9 is the large proportion of deaths under 3 years of age from diarrhoea and dysentery. This age group was responsible for 81% of all deaths reported as diarrhoea and dysentery. It is quite likely that the two syndromes were responsible for more infant deaths when other causes are taken into consideration. The term 'teething' was given as a cause of death in 19 cases of children between the ages of 0 and 1 years. What this term implies when it is given as a cause of death is unclear and hence I have placed it in the ill-defined category (Appendix B), but it may be a euphemism for diarrhoeal symptoms accompanying tooth eruption. If so, then up to 87% of deaths from diarrhoea and dysentery would come from this age range.

The high number of deaths in the under 3 age range is typical of diarrhoeal diseases (Marcy 1976: 892; Mata 1980:11). Epidemiologically, both syndromes are common amongst infants and very young children, particularly those living in underdeveloped countries (DuPont 1994: 677; Marcy 1976: 892; Rubin & Farber 1988:

666; Sommers 1980: 540-1). Despite the improvements in public health in recent decades, most children are still expected to suffer attacks of diarrhoeal diseases (Mata 1980:3) and

Table 8.9 Reported occurrence of diarrhoea and dysentery as causes of death on Aboriginal settlements in Southeast Australia 1876-1900.

Settlement	Year	Sex	Age
Diarrhoea			
Coranderrk	1889	Female	1
		Female	2
	1890	Female	7
	1893	Female	0
Point McLeay	1876	Male	0
	1877	Male	2
		Female	0
	1878	Male	2
		Female	0
		Female	0
		Male	1
	1880	Female	0
	1882	Female	1
		Female	2
		Female	0
	1883	Male	1
		Unknown	0
	1888	Female	Unknown
		Female	Unknown
	1892	Male	1
	1895	Male	18
	1896	Male	0
		Female	0
	1897	Female	0
Dysentery			
Lake Condah	1877	Male	17
Lake Tyers	1881	Male	2
Lake Hindmarsh	1891	Female	1
Point McLeay	1880	Female	1
		Female	0
	1882	Male	30
	1890	Female	0
	1893	Male	0
	1894	Male	65
		Female	1
	1899	Female	0

1900	Female	1

many in the world today live through a substantial risk of acquiring a fatal infection (Marcy 1976: 892; Hardy 1956). Living conditions that are characterised by a lack of plentiful clean water, inadequate sewerage, poor food and personal hygiene, poor nutrition, and inadequate health care are favourable to the spread of infectious enteric microorganisms. Infants usually acquire *E. coli* during the first days of life by ingesting maternal organisms at birth and later in life from the hands of other infants, toddlers, and elder carriers, who may be symptomatic or asymptomatic, and are shedding the bacteria (Marcy 1976:905). The young are susceptible to the symptoms when loss of maternal antibody protection occurs after breast feeding ceases and before contact with the various bacteria has generated a sufficient level of self protection (Marcy 1976: 904).

The distribution of deaths throughout the period 1876 to 1900 is suggestive of endemicity of the pathogens. The highest number of deaths in any one year was 4 in 1878 at Point McLeay, which regularly had the largest population of all the settlements; the remaining deaths occurring at levels of 1 or 2 per year. If living conditions allow bacteria to become locally endemic, a high morbidity and mortality can be expected in the early years of life. But when an enteric infectious organism becomes endemic a degree of antigenic homogeneity can develop among the strains, particularly *E. coli* and *Shigella* sp., in turn leading to a more efficient immune response by the host with increased age. This leads to lower morbidity and less mortality after the initial years of life.

There is little other evidence in the historical literature from which an assessment of the overall prevalence of diarrhoeal diseases can be made. From the causes of death, particularly in those under three years of age, and the conditions under which the

Aboriginal people lived on the settlements (Barwick 1972, Jenkin 1976, 1979; McCrea 1879) we can assume that it was high and that most children acquired an infection at least once during their life. If infections led to chronic or repeated acute diarrhoeal illness it would have had an adverse effect on the growth of the children and contribute to further malnutrition. DuPont (1994: 678) has estimated that there is a 20 to 60% decrease in the caloric intake during a bout of diarrhoea, depending on the severity and the length of the symptoms. Mata (1983: 13) identifies a protein-losing enteropathy occurring in diarrhoea due to tissue-invading bacteria and viruses.

Chronic diarrhoea in children is a contributing factor in marasmus and kwashiorkor, particularly during the first two years when weaning occurs (Mata 1983: 13). There were 9 causes of death given as marasmus at Point McLeay (Appendix B); eight were in children under 3 years of age and a further case in a 60 year old female. The age range and the prominence of children (89%) are similar to those whose deaths were recorded as diarrhoea and dysentery. These individuals may therefore have suffered from chronic diarrhoeal infection prior to death causing a deficiency in food absorption leading to prolonged protein-energy malnutrition (PEM).

It appears then that a high number of childhood diarrhoeal diseases infections can be inferred as a character of life on Aboriginal settlements. In many cases it was a principal cause of death. In other cases chronic or repeated diarrhoeal infection would have led to anorexia and weakened the immune response, exposing the young to a further threat from other infections. Diarrhoea and dysentery would thus have been common underlying conditions predisposing to death from other causes.

Other infectious diseases

Table 8.10 lists all other infectious diseases reported in this age range less diarrhoea and dysentery.

Table 8.10 Infectious diseases less diarrhoea and dysentery reported as causes of death in age range 0-4 years.

Reported cause of death	n	%[1]
Tuberculosis	8	9.6
Measles	4	4.9
Thrush	4	4.9
Hydatids	1	1.2
Scarlet fever	1	1.2
Low fever	1	1.2
Total	**19**	
(Ill defined causes	64	77.1)

1 Includes the ill defined category in the denominator

Because of the large number (n = 64) in the ill defined category it is difficult to make strong inferences from this table. It can be assumed with some safety, however, that for most of the diseases the figures represent a minimum number of the total causes of death. The 8 cases of tuberculosis can, I believe, be certainly seen as a minimum number when its generality in the Aboriginal populations prior to 1876 (Chapter 5) and its frequency or reporting in later age ranges (see below) is taken into consideration.

The four deaths from measles may, however, reflect a more reliable frequency. Three of the deaths reported, 1 at Point McLeay in 1893 and 2 at Lake Wellington in 1894, were during an epidemic period among the European populations (Chapter 7). The last death was at Lake Tyers in 1900. Its relative absence among the Aboriginal people can be explained, however, by their isolation from the main European population centres. The missionaries and superintendents of the settlements made

concerted efforts to restrict contact between the Aboriginal inmates and the nearby European townships. Their reasons were not so much to do with protecting the Aboriginal people from infectious diseases but rather to regulate their movements, Christianise them and shield them from what they saw as moral corruptness within European society (Broome 1982: 69-86; Saggers & Gray 1991:69). Their efforts were encouraged by the colonial governments who saw institutionalisation and isolation as a major step in controlling the Aboriginal people. While their efforts at isolation were not always successful and contact between the two groups continued it was much reduced compared with earlier years. The result was to shield the inmates of the settlements to a large degree from localised outbreaks of measles which regularly arose in the European population. There were five major epidemics of measles in the European population between 1876 and 1900 (Chapter Seven, Table 7.1); the latter two in 1884 and 1893 had high cause-specific mortality rates of 41-49 per 100,000 and 74 per 100,000 respectively. As discussed in Chapter Seven there is little evidence of these epidemics among the Aboriginal people living on settlements in Southeast Australia.

Thrush was reported in what would appear as a localised outbreak at Point McLeay in 1893 (Appendix A). Thrush (or candidiasis) is usually a benign condition presenting in the oral cavities during the first weeks of life. The disease can become life-threatening under two situations. In children, who are immunosuppressed by other infections or under antibiotic therapy. The former would most likely have been the cause in these children. In such a situation candidiasis can cause severe and fatal disseminated infection involving almost all tissues, with the kidneys, liver, spleen, heart, and brain often implicated (Miller 1976: 638, 641).

The reporting of this disease as a cause of death in such temporal and spatial isolation is odd when it is considered that candidiasis is common in newborn infants and

that the causative organism *Candida* sp. is part of the normal human flora. The reason for the outbreak may lie in the varying pathogenicity of the species or strains of *Candida* concerned. It is usually *Candida albicans* but several other species can be implicated. The degree of pathogenicity depends on the toxin produced by the organism and can vary between each strain (Benenson 1990:72-73; Miller 1976: 638). Another alternative that should be considered is that of misdiagnosis. Candidiasis can be confused with other oral and disseminated infections particularly when only clinical diagnosis is available. Herpesvirus and coxsackie virus lesions in infants with disseminated infection may appear clinically similar to those with sepsis due to other organisms (Miller 1976: 643). Post natal infection with *Treponema pallidum* cannot be ruled out as a contributing factor.

8.4.1.3 Respiratory diseases

With a PMR of 28.0 respiratory diseases were the second most definable disease group recorded as cause of death in age range 0 to 4 years. Table 8.11 lists the causes of death from respiratory diseases.

Table 8.11 Respiratory diseases reported as causes of death in age range 0-4 years.

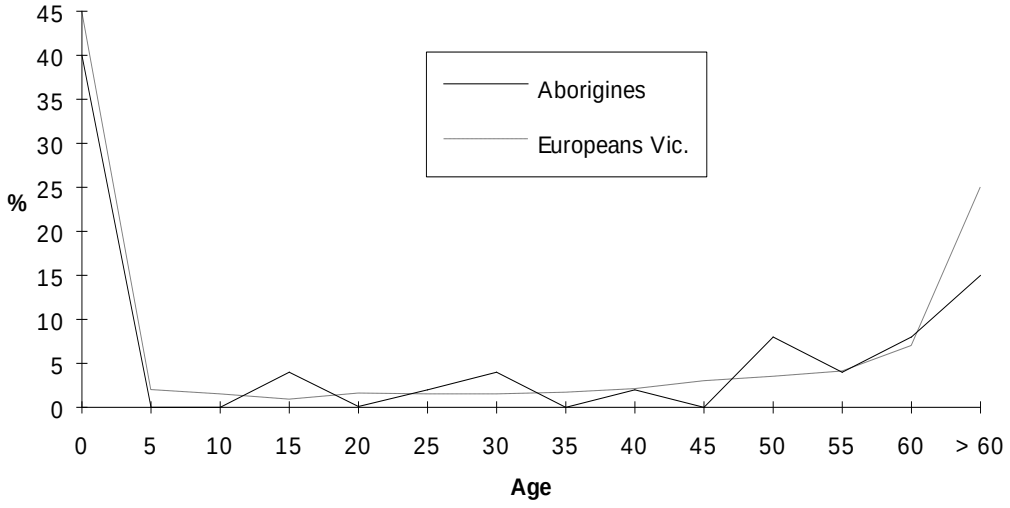
Reported cause of death	n	%[1]
Bronchitis	29	22.3
Whooping cough (pertussis)	14	10.8
Pneumonia	10	7.8
Influenza	6	4.6
'Inflammation of lungs'	3	2.3
'Congestion of lungs'	2	1.5
'Lung disease'	2	1.5
Total	**66**	
(Ill defined causes	64	49)

1 Includes the ill defined category in the denominator

Bronchitis

Bronchitis (n = 29, 22.3%) was the outstanding disorder amongst respiratory diseases. Age distribution of deaths from bronchitis among the Aboriginal settlements is compared with the European population of Victoria for the years of 1876, 1880, 1890, and 1900 in Figure 8.5. Both curves show similar high frequencies of death during early childhood and late adulthood with a long period of low frequency during the intervening years. The peaks during middle adulthood displayed by the Aboriginal curve do not indicate a high mortality from bronchitis when compared with the European population, but rather reflect a much smaller sample size.

Figure 8.5 Age distribution of deaths from bronchitis, among Aboriginal populations living on settlements in Southeast Australia 1876 to 1900 compared to European population of Victoria for the years 1876, 1880, 1890 & 1900.



Rubin and Farber (1988: 584) define bronchitis as the hyperplasia and hypertrophy of the bronchial mucous glands and an increased production of mucus in the

bronchial passages. The principal clinical sign of bronchitis is a persistent productive cough, often more severe in the winter months. Acute respiratory failure can occur in advanced cases leading to insufficient oxygenation of the blood (hypoxaemia) and ventricular failure. Chronic bronchitis can often be an underlying factor for a number of degenerative disorders. Because of retained mucous secretions, individuals with chronic bronchitis are at an increased risk of bacterial infections of the lungs, particularly pneumonia. Further, the risk of lung cancer and cardiovascular disease is increased by chronic bronchitis. As such, the significance of bronchitis in its effects upon the mortality experience of a community is often unrealized because of its contributory role in the deaths formally attributed to other causes (Young 1979: 320).

Three important causative factors have emerged in the development of the condition: infection, tobacco inhalation, and atmospheric pollution (Benenson 1990: 367-8; Cotran *et al.* 1989: 771-3; Thurlbeck & Miller 1988: 585-591). Episodes of bronchitis ranging from mild to severe symptoms can be initiated by pathogenic invasion particularly from *Haemophilus influenzae* and *Streptococcus pneumoniae*. Symptoms resulting from these bacterial invasions may also be associated with concurrent viral infection of the respiratory tract or with chronic exposure to atmospheric pollutants (Rubin & Farber 1988: 584). Age is a significant factor in infectious bronchitis (Cotran *et al.* 1989: 772). Figure 8.5 shows a high frequency of deaths from bronchitis among both Aboriginal and European children, particularly during the first year of life and then a levelling out at age three years.

During the first year of life children are particularly susceptible to bronchial disturbances initiated by bacterial or viral infection. Predisposing factors for acute and life-threatening symptoms for these early ages would include the unfavourable socioeconomic circumstances and poor nutrition experienced by most Aboriginal people

on the settlements. A further predisposing factor would have been the dwellings which were often over crowded, dusty in summer and damp during the winter (Barwick 1972; Jenkin 1979: 222; McCrea 1879-80). Passive tobacco smoke inhalation would have been common among new born and young Aboriginal children, further promoting the onset of bronchitis.

Whooping cough (pertussis)

Whooping cough (n = 14, 10.8%) was the second major cause of death from respiratory disease. Whooping cough is an acute bacterial disease common to children 0 to 5 years of age worldwide, regardless of climate, or geographic location. The causative agent is *Bordetella pertussis.* Infection is primarily by direct contact with airborne discharges from respiratory mucous membranes of infected persons (Benenson 1990: 318-319). The disease would have been readily recognised in the nineteenth century. Observable symptoms begin with a catarrhal phase which gradually develops into severe paroxysms characterised by repeated violent coughing. Each series of coughs continues without intervening inhalations and is followed by the characteristic high pitch inspiratory whoop from which the diseases gets its common name. The paroxysmal stage can last for 1 to 2 months or longer with as many as 50 coughing episodes per day (Benenson 1990: 319; Connor & Gibson 1988: 364-365). Immunity is often conferred after the first infection by *B. pertussis* but second attacks can occur. Compromised immune systems can assist subsequent infections in infants and initial infections in adolescents and adults (Benenson 1990: 321).

Whooping cough deaths were confined to the 0 to 4 years age range with the exception of one 8 year old male. This is the common pattern of mortality from the disease with death rates highest in children up to the age of 5 years (Connor & Gibson 1988: 365). Of the deaths within the 0 to 4 years age range 8 (57.1%) were in the first

year of life; 3 (21.4%); in two year olds; and 1 in a three year old. There was no sex difference.

Other respiratory diseases

Pneumonia was recorded as a cause of death in 10 cases (Table 8.8). If the term 'inflammation of the lungs' is referring to the same disorder (Morgan 1987) then another three cases can be added giving pneumonia a similar ranking to whooping cough. The low reported frequency of influenza is most likely a misrepresentation of the real threat this disease had to the Aboriginal infants and children due to the reporting of other symptoms. Infection by the virus can cause a range of secondary conditions that if left untreated can be life threatening. They include viral and bacterial pneumonia, bronchitis, the common cold, gastrointestinal manifestations presenting as nausea, vomiting, diarrhoea, prolonged fever, and convulsions (Benenson 1990: 224). It is quite likely that much of the mortality from respiratory causes was precipitated by an influenza virus infection.

8.4.2 Age range 5 to 14 years

One hundred deaths (14.4% of the total) were recorded in this age range (Table 8.5). Infectious diseases (PMR 54.0, n = 54) and respiratory diseases (PMR 13.0, n = 13) were the leading causes of death. Combined, they accounted for 67% of all the deaths in this age range.

8.4.2.1 Infectious disease

Table 8.12 shows the number of deaths from infectious disease for age range 5 to 14 years.

Table 8.12 Causes of death from infectious diseases in age range 5 to 14 years.

Reported cause of death	n	%[1]
Tuberculosis	35	64.8
Intermittent fever	2	3.7
Fever	1	1.9
Rheumatic fever	1	1.9
Enteric fever	1	1.9
Diarrhoea	2	3.7
Hydatids	2	3.7
Total	**44**	
(Ill defined)	(10)	(18.5)

1 Includes the ill defined category in the denominator

Tuberculosis

Tuberculosis (n =35, 64.8%) was the major infectious disease responsible for mortality in this age range accounting for 35% of all deaths. Table 8.13 shows the number of male and female deaths reported from tuberculosis in the 5 to 14 years age range.

Table 8.13 Number of deaths from tuberculosis in age range 5 to 14 years.

Age	Male	Female
5	2	2
6	2	1
7	0	0
8	0	1
9	0	0
10	0	4
11	1	3
12	0	8
13	3	3
14	1	4
Total	**9**	**26**

Female deaths (n =26) were significantly more than male deaths (n =9) (x^2 = 8.26, p = 0.004, 1df).

The age and sex of a tuberculosis infected individual have a strong influence over the outcome of the disease. The main age periods of high susceptibility are during infancy, puberty, and old age. The specificity of tuberculosis mortality for each of the sexes also varies. In youth and early adulthood females suffer from a heavier mortality than males (as seen above). This trend can reverse itself in later adulthood, with males suffering a higher mortality. The onset of menarche and higher rates of mortality from tuberculosis in young women are linked (Johnston 1993: 1060) although the reasons are not fully understood. The metabolic changes during menarche increase the body's need for protein and this in turn may reduce the efficiency of the immune system. Its response to new mycobacterial infection, or a present latent infection, would then be less effective, resulting in the onset of acute clinical symptoms of tuberculosis.

Another factor that could be influential in the deaths of females from tuberculosis is the differing daily lifestyle of males and females of this age range. Both sexes spent much of their day in classrooms under instruction by the missionaries (Massola 1970, 1975). Crowded classrooms can be fertile breeding grounds for tuberculosis if one individual is infected and capable of spreading the mycobacteria. Evidence of this can be seen of the sporadic epidemic outbreaks of tuberculosis in school children in western countries this century (Proust 1991a: 227-230) where the disease is not a major cause of morbidity. In such cases both sexes would be equally exposed to the infection. Out of the class rooms, however, the young females were often required to spend much of their time indoors learning domestic skills while young males spent time out of doors learning labouring and agricultural skills (Jenkin 1979: 97-111; Masssola

1970, 1975) . The young females would then have more daily contact with tuberculosis sufferers in confined spaces and would be more exposed to infection.

8.4.2.2 Respiratory diseases

Table 8.14 shows the frequency of respiratory diseases recorded as a cause of death.

Table 8.14 Causes of death from respiratory diseases in age range 5 to 14 years.

Reported cause of death	n	%[1]
Pneumonia	5	21.7
'Pulmonary'	1	4.3
'Inflammation of lung'	2	8.6
'Congestion of the lungs'	1	4.3
'Lung disease'	1	4.3
Whooping cough	1	4.3
Bronchitis	2	8.6
Total	**13**	
(Ill defined	10	43.5)

1 Includes the ill defined category in the denominator

Pneumonia with 5 deaths (or 7 if 'inflammation of the lungs' is grouped with it) is the major cause of mortality in this age range. The small number of deaths in this category precludes any meaningful analysis or statements other than to note the total of deaths (n = 13) attributed to respiratory disorders. The highest levels of mortality from the majority of respiratory diseases are usually during infancy, early childhood and the aging (Benenson 1990: 330). The low level (PMR 13, n = 13) of these diseases in the 5 to 14 years age range is therefore not unusual . Low socioeconomic circumstances and/or underlying medical conditions can promote this disorder to life threatening situations in the young (Benenson 1990: 330).

8.4.2.3 Digestive system diseases

Diseases of the digestive system had a PMR of 10.0 (n = 10) in this age range. Peritonitis was given as the cause in 5 cases; 3 females and 2 males aged between 5 and 9 years. Other causes of death in this category were given as bowel, stomach and liver disorders.

Peritonitis (inflammation of the peritoneum) is a serious, life-threatening disorder. The most effective treatment is surgery. Peritonitis is most commonly caused by perforation of the abdominal viscus leading to bacterial invasion of the peritoneum. An escape of bile from a ruptured gall bladder can also initiate inflammation. A primary form of peritonitis is caused by toxins from the blood stream entering the peritoneum. Common primary disorders leading to peritonitis are peptic ulcer, inflamed appendix, colonic diverticulum, and strangulation of the bowel. Tuberculosis infection may also be involved (Rubin & Farber 1988: 717-718). For nineteenth century medical practitioners it was readily diagnosed by the sudden onset of abdominal pain in the area of initial invasion becoming general as inflammation spreads, and a characteristic abdominal rigidness caused by contraction of abdominal muscles (Dayal & DeLellis 1989: 905; Rubin & Farber 1988: 717-718). The likely causes of the disorder among the 5 children would have been a ruptured appendix or an underlying tuberculosis infection.

8.4.3 Age range 15+ years

Three hundred and sixty deaths (51.7% of total deaths) were recorded in this age range (Table 8.5). Infectious diseases had a PMR of 33.7 (n = 121) and respiratory diseases a PMR of 16.4 (n = 59). Combined they account for 50% of the total deaths in

this age range. The next highest category was digestive system diseases with a PMR of 7.5 (n = 27). The remaining disease categories had PMRs of 4.7 or less. The ill defined category (n = 93) had a PMR of 25.8. This was mainly due to the reporting of 70 deaths given as either 'senile decay', 'old age', or 'general decay'.

8.4.3.1 Infectious diseases

Table 8.15 shows the frequency of infectious diseases reported as causes of death.

Table 8.15 Causes of death from infectious diseases in age range 15+ years.

Reported cause of death	n	%[1]
Tuberculosis	97	45.3
Hydatids	10	4.7
Dysentery	4	1.9
Diarrhoea	1	0.5
Enteritis	4	1.9
Measles	2	0.9
Scarlatina	1	0.5
Typhoid	2	0.9
Total	**121**	
(Ill defined)	(93)	(43.4)

1 Includes the ill defined category in the denominator

Tuberculosis

Tuberculosis (n = 97, 45.3%) was the outstanding cause of death in the infectious disease category. Overall it accounted for 27% of all deaths in this age range. Table 8.16 compares the reported causes of death from tuberculosis infection for the 15+ age range in age increments of 10 years for each sex. .

Table 8.16 Recorded cases of tuberculosis deaths in age range 15+ years where both age and sex were recorded.

Age	Males		Females	
	n	%	n	%
15-24	18	41.9	19	38.0
25-34	9	20.9	14	28.0
35-44	6	14.0	6	12.0
45-54	6	14.0	6	12.0
55+	4	9.3	5	10.0
	43		50	

There is no significant difference between males and females (X^2 = 0.5268, p = 0.4679) for the age range.. What is noticeable is that for both sexes the majority of deaths from tuberculosis occurred before the age of 35 years (males 62.8% and females 66%). After 35 years of age the number of deaths from tuberculosis falls. Age has a strong influence in the final outcome following infection by *M. tuberculosis.* Infancy and puberty are typically periods of low resistance and high susceptibility to tuberculosis. The lowest mortality rates usually occur after 3 years old and before the age of 15 years in both males and females. The risk of developing life threatening symptoms of the disease increases again among adolescents and young adults (Benenson 1990: 460; Dubos & Dubos 1953: 194; Johnston 1993: 1066). This can result either from reactivated symptoms of long latent infections or primary exposure to the pathogen (Benenson 1990: 460; Connor & Gibson 1988: 395). Among the Aboriginal populations on the settlements the high mortality from the disease in early adulthood is typical of endemic tuberculosis, ineffectively treated.

Hydatids

Hydatid disease (echinococcosis) as a cause of death was reported 10 times (5.0%) in the 15+ age range (Table 8.17). (Three other cases; a 4 year old male, 10 year

old female and a 13 year old female were reported). The cases occurred throughout 6 of the 8 settlements. Coranderrk settlement recorded the most deaths with 5 (38%) cases, 4 of which occurred in a 3 year period between 1879 and 1881. Because of the small number or reported cases no age/sex pattern can be statistically determined but in the 15+ age range male deaths (n = 7) outnumbered female deaths (n = 3). Among the European population males were more susceptible to hydatids than females (Thomas 1884: 150-168) most likely due to more males than females being employed in rural activities.

Table 8.17 Reported occurrence of hydatids as a cause of death on Aboriginal settlements in Southeast Australia 1876-1900.

Settlement	Year	Sex	Age
Framlingham	1879	Male	50
Lake Wellington	1884	Female	45
	1885	Female	10
Coranderrk	1879	Male	45
	1879	Female	26
	1880	Male	35
	1881	Male	50
	1900	Male	56
Lake Hindmarsh	1879	Male	32
Point McLeay	1880	Male	32
	1891	Female	27
	1892	Male	4
Point Pearce	1883	Female	13

Hydatids had most likely become common among the Aboriginal populations of the Southeast by the early 1860s (Hudson 1861: 75). Probably the first reference to hydatids disease among the Aboriginal populations of the Southeast comes from Hudson (1861: 75) who carried out a post mortem on an Aboriginal male and identified hydatid cysts. Hudson states that the male had been 'living in the bush' and had eaten under-cooked 'diseased mutton' given to him on the sheep stations. Another early reference to hydatids among Aboriginal populations comes from Point McLeay in 1876. Taplin

(1876a), reviewing the diseases he had observed among the Ngarrindjeri during the previous 18 years, lists 'hydatids in the liver' as one of the diseases they were subjected to. He did not record any deaths from hydatids during this period.

From the second half on the nineteenth century hydatid disease had become a serious disease among the European populations of Victoria and South Australia. During the period 1861 to 1880, 584 deaths (mean death rate per annum 16.23 per 1,000) were recorded in Victoria from hydatids. In South Australia between 1865 and 1880 the mean death rate per annum was 15.16 per 1,000 (Thomas 1894: 150, 167).

Hydatid disease is a zoonotic infection of humans occurring mainly in rural areas. Although no organ is exempt, the disease causes cyst formation mainly in the liver and lungs and less frequently in the kidneys, spleen, bone, and central nervous system (Stevenson & Hughes 1980: 40; Thomas 1884). *Echinococcus granulosus* (a dog tapeworm) was most likely the causative agent of the disease on the settlements. Today it is the most common cause of hydatid infection in rural communities of Southeast Australia. *E. granulosus* occurs where dogs are used to muster grazing animals, particularly sheep, and where they have an intimate contact with humans. These conditions were common to most of the Aboriginal settlements where attempts at self-sufficiency were made by grazing sheep and cattle on lands adjacent to living areas. Dogs were popular both as working animals and as pets on the Aboriginal settlements (Gibson 1869-75; Plate 8.1).

The major life cycle of the tapeworm is dog-sheep-dog. Transmission to humans is usually by hand to mouth transfer of the mature tapeworm’s eggs from dog faeces. Exposure occurs in handling infected dogs and objects soiled by dog faeces. Exposure can also occur through contaminated food and water (Benenson 1990:149).

Another life cycle occurs in endemic Australian mammals of dingo-wallaby-dingo (Stevenson & Hughes 1980: 40) which may have involved different variants of *E. granulosus* (Thompson & Kumaratilake 1982: 16).

Although 13 deaths from hydatid disease were recorded on the settlements during the period the real extent of morbidity and mortality is unclear because of the difficulties involved in diagnosis last century. There is no record of post mortems. Diagnosis of hydatids would have involved an assessment of the clinical symptoms, most likely by tactile detection of cysts lying close to the body surface, and by fragmenting cyst portions, particularly those occurring in the peritoneal cavity, lung or intestinal tract, being coughed up, vomited or passed in the faeces (Thomas 1894): If hydatid disease was not diagnosed by these means other causes of death would have been recorded. Causes of death such as 'tumified liver', 'enlarged liver', 'liver disease', 'lung disease', or those involving the brain may in reality be related to advanced hydatids infection. Thus hydatids may have been directly or indirectly responsible for a greater number of deaths than is reflected in the settlements' records of death.

Enteric fever (typhoid)

There were 2 causes of death listed as typhoid and 4 listed as enteritis or enteric fever in this age range (Table 8.15), and 1 cause of enteritis listed for the 5 to 14 years age range (Table 8.18).

Table 8.18 Causes of death in 15+ age range possibly attributed to typhoid.

Settlement	Year	Sex	Age	Cause of death
Lake Hindmarsh	1877	Female	16	Enteritis
Lake Condah	1877	Male	30	Typhoid Fever
Point McLeay	1878	Female	15	Enteritis
	1880	Female	12	Enteric Fever
	1880	Male	40	Enteric Fever
Point Pearce	1891	Male	16	Typhoid

There was considerable confusion over the diagnosis of typhoid in nineteenth century Australia (Cumpston 1989: 230). Typhoid is easily confused with other diseases displaying a febrile onset; for example, hepatitis, tuberculosis, typhus (LeBaron & Taylor 1993: 1073). Typhoid (or enteric fever, enteritis) has been present in Australia since the arrival of the First Fleet but did not become a major medical problem until after 1850 when the gold fields were opened and a massive increase in population ensued (Cumpston 1989: 230). From then until 1890 typhoid was responsible for high death rates among the Europeans of between 30 and 80 per 100,000. Deaths were highest between the ages 15 and 44 years. A gradual decline in the incidence of the disease then occurred in the eastern colonies (Cumpston 1989: 230). The disease almost certainly did not exist among the Aboriginal population of Australia prior to 1788. Its history among the Aboriginal people during the colonial period is largely unknown but the deaths reported on the settlements suggest that a typhoid-like disease was present among them although at a minor level.

8.4.3.2 Respiratory diseases

Table 8.19 shows the frequency of respiratory system diseases reported as causes of death.

Table 8.19 Causes of death from respiratory diseases in age range 15+ years.

Recorded cause of death	n	%[1]
Bronchitis	22	14.5
Inflammation of lungs	18	11.8
Pneumonia	7	4.6
'Lung disease/complications'	4	2.6
Congestion of lungs	3	2.0
Influenza	2	1.3
Pleurisy	1	0.7
Laryngitis	1	0.7
Asthma	1	0.7
Total	**59**	
(Ill defined causes)	(93)	(61.2)

1 Includes the ill defined category in the denominator

Bronchitis

Bronchitis (n = 22; 14.5%) was the second most recorded cause of death from respiratory disorders if 'inflammation of the lungs' and pneumonia are grouped as the most recorded cause. There was no difference between male (n = 11) and female (n = 11) frequencies. Table 8. 20 displays a breakdown of deaths in 10 year age increments.

Table 8.20 Breakdown by age of recorded deaths from bronchitis (male and female) in age range 15+ years.

Age	n	%
15 - 24	0	0
25 - 34	2	9.1
35 - 44	2	9.1
45 - 54	6	27.3
55 - 64	3	13.6
65 - 74	6	27.3
75+	3	13.6
Total	**22**	**100**

A sharp increase of deaths from bronchitis from age 45 years and onwards is noticeable. Nineteen deaths or 81.8% occurred after this age. This trend is quite likely to have been a result of chronic bronchitis due to many years of tobacco smoking. The most

important factor in the development of chronic bronchitis in later adulthood is the practice of tobacco smoking. The mortality rate from bronchitis is significantly higher in tobacco smokers than in non smokers (Rubin & Farber 1988: 584). In recent years 90% or more of all cases of chronic bronchitis have been found to occur in smokers; the frequency of the symptoms in chronic smokers is more than 25%, in moderate smokers 10 to 15%, and in non-smokers it is less than 5% (Rubin & Farber 1988: 584).

The European colonists smoked a lot of tobacco, often seeing it as a necessity of the hard life in the colonies rather than as a luxury. Contact with Europeans introduced Aboriginal people to tobacco and smoking quickly became a particularly common practice among most communities (Bulmer 1876; Mason 1859; Saggers & Gray 1991: 86-88). A currency of addiction was created from tobacco. The colonial governments provided Aboriginal people with regular weekly or monthly rations of tobacco, often giving extra proportions to the elderly or sick. The substance was further bartered or bought from European traders. Pastoralists often used tobacco rather than money as an enticement to Aboriginal people to work for them.

Tobacco was easily obtained on the settlements and addiction was widespread among both males and females. It took the place of indigenous narcotics such as pituri (*Duboisia hopwoodii*) which had in the past been traded through the extensive trade and exchange networks (McBryde 1987; Watson 1983) and which now no longer operated. On Lake Tyers settlement in 1878 there were sixty people (many of them elderly women), out of just over one hundred residents who were addicted to tobacco (Bulmer 1876). The manager of the settlement, Rev. J. Bulmer, allowed a three-monthly ration of 30 lbs (14 kg) a month for the whole settlement which he rationed out at no more than two 'figs' per week. He could not get them to abstain from smoking tobacco. Tobacco was seen by

many Aboriginal people as the only drug in the British pharmacopoeia that was of any use. Opium was also smoked by some (Bulmer 1879).

The third aetiological factor in the development of bronchitis is atmospheric pollution, particularly from industrial sources. The precise agent or agents in atmospheric pollution remain uncertain but high levels of sulphur (as SO_2 and H_2SO_4) in the air may be responsible for initiating the symptoms of chronic bronchitis (Rubin & Farber 1988: 584). It would be expected that this factor played a minor role in the development of bronchial disabilities among the Aboriginal communities concerned in this study. Nevertheless, domestic fire-places have been associated with high levels of bronchitis. Ashley (1967) examined the distribution of bronchitis in 83 county boroughs of England and Wales and found that pollutants derived from domestic fire-places rather than industrial sources were primarily indicated in the aetiology. Poorly ventilated dwellings were common on the Aboriginal settlements and smoke from internal domestic fires may have played a part in the development of chronic bronchitis among the residents.

Pneumonia/Inflammation of lungs

Pneumonia (n = 7; 4.6%) and 'inflammation of the lungs' (n = 18; 11.8%) taken jointly accounted for 25 (16.4%) of recorded respiratory disease deaths. As discussed above and in Chapter 6 high levels of mortality from respiratory diseases are common in the ageing. Pneumonia is often a secondary complication of other disorders, particularly influenza infections and is often aggravated by chronic tobacco inhalation and alcohol consumption (Thurlbeck & Miller 1988: 567-569). Both were common habits among Aboriginal populations (Bulmer 1876; Cannon 1988: 64; Lewis 1992: 152-156; Mason 1859). Even with modern treatment case fatality rates among patients with substantial underlying disease can reach 20 to 40% (Benenson 1990: 330). Among the Aboriginal people on the settlements, however, the case fatality would be expected to be

much higher when the lack of effective treatment of the disease and the socioeconomic circumstances are taken into consideration. The high mortality from pneumonia here can be seen as a continuation of this disease among adult Aboriginal people living on the settlements which began during the incarceration of Tasmanians on Flinders Island (Wybalenna) between 1831 and 1847 and later at Oyster Cove (Plomley 1987:915-942; Smith 1858-69).

8.4.3.3 Digestive system diseases

Table 8.21 lists diseases of the digestive system (PMR 7.5; n = 27) recorded as causes of death.

Table 8.21 Causes of death from digestive system diseases in age range 15+ years.

Reported cause of death	**n**	**%[1]**
Liver disorders	9	7.5
Bowel disorders	7	5.8
Peritonitis	3	2.5
'Chronic disease of stomach'	5	4.5
Hepatitis	1	0.8
Gastric Fever	1	0.8
Dyspepsia	1	0.8
Total	**27**	
(Ill defined causes)	(93)	(61.2)

1 Includes the ill defined category in the denominator

It is difficult to make any substantial statement regarding this disease group other than to recognise that disorders of the liver and disorders of the bowels are the two most prominent recorded causes of death. Disorders of the liver included 'cirosis [*sic*] of liver', 'enlargement of liver', 'liver inflammation', 'tumefied liver', 'liver complaint', and 'disease of liver'. Although the liver is vulnerable to a wide range of metabolic, circulatory, toxic, microbial, and neoplastic insults (Cotran *et al.* 1989: 915) the recorded

causes, together with one cause of death named as 'hepatitis', may suggest the presence of acute viral hepatitis A (HAV). Hepatitis has been recognised by medical practitioners and observers for millennia and was usually associated with jaundice (Rubin & Farber 1988: 739). This latter term, however, does not appear in the Aboriginal mortality records although the reason for this may be that it is less visible in darker-skinned people. Acute viral hepatitis usually causes inflammation and necrosis of the liver to which the above terms may apply. If so the disease was probably hepatitis A. HAV is spread from person to person by the faecal oral route usually by oral ingestion of contaminated water or food supplies. With the poor sanitation and hygiene conditions of the Aboriginal settlements HAV would most likely be the most common form of hepatitis. Its low frequency as a cause of death is most likely due to the fact that HAV never pursues a chronic course, there is no carrier state, and infection provides lifelong immunity (Rubin & Farber 1988: 739).

There were 6 causes of death recorded as 'inflammation of the bowels'. The term is ambiguous but may be referring to what is recognised today as nonspecific inflammatory bowel disease. The aetiology of this disease category is uncertain but ulcerative colitis and Crohn's disease (regional enteritis) have been implicated as either two separate entities or opposite ends of a single inflammatory spectrum (Dayal & DeLellis 1989: 868; Rubin & Farber 1988: 692).

8.4.4 Less frequent causes

This section will briefly cover the remaining disease categories not discussed above. Because of their low PMRs and consequent lesser importance to Aboriginal mortality they will be discussed in overview and not according to age range.

The less frequent conditions recorded as causes of death were diseases of the digestive (PMR 5.9), nervous system (PMR 5.0), circulatory system (PMR 2.7), perinatal conditions (PMR 1.3), injuries and poisonings (PMR 2.3), neoplasms (PMR 2.3), endocrine system (PMR 2.1) complications of pregnancy (PMR 0.6), and diseases of the genitourinary system (PMR 0.3). As shown by their respective principal mortality ratios these categories were of less importance than infectious and respiratory system diseases and did not pose serious mortality and health problems to the Aboriginal inhabitants of the settlements.

Forty-one deaths were recorded from digestive system disorders. This group included abnormal conditions of the mouth, stomach, peritoneum, bowels, and liver. Diseases of the nervous system were reported in 38 deaths. They included a broad range of disorders and symptoms - disorders of brain tissue, hydrocephalus, meningitis, paralysis, convulsions and fits. Heart disorders accounted for 19 deaths. Sixteen deaths from injury included drowning, suffocation, sunstroke, falls, burns and gunshot wounds.

Fifteen cases of marasmus were reported; 5 from Lake Hindmarsh and 10 from Point McLeay. All but one case, a 64 year old female, were of children under 9 years of age. The link between marasmus and diarrhoeal disorders has been discussed above and it is possible that these deaths may have been due a chronic diarrhoeal state. Their recording on just two of the settlements is most likely due to the different diagnostic or recording practices rather than a differential occurrence. 'Cancers' and 'tumours' were recorded in 13 cases. The bladder, tongue, intestines, chest, leg, and unspecified regions of the body were given as sites. Perinatal and pregnancy complications, and disorders of the genitourinary system accounted for 11, 4, and 2 deaths respectively.

No reliable epidemiological inferences can be drawn from these disease groups other than to note their presence and their respective principal mortality ratios. The two with the highest PMRs, digestive system diseases (6.4), and nervous system diseases (6.0), each represent less than two deaths per year over the 25 year period for each of the eight settlements; the others even less. Whilst there would have been more than the occasional error in their diagnosis as a cause of death, their universally low reporting in the eight settlements strongly indicates that these causes of death were overshadowed by infectious and respiratory system diseases.

Two final references to infectious diseases are needed. The first is the infrequent reporting of scarlet fever (scarlatina) among the settlements. Two deaths due to 'sraclatina' were reported, one in a 45 year old (sex not recorded) and the other in a 2 year old female. The low frequency of recorded deaths from this disease reflects the pattern in the general population of Southeast Australia for the period. In both Victoria and South Australia scarlet fever had its highest recorded death rate in 1876; (280 per 100,000 for Victoria and 112 per 100,000 for South Australia) and throughout the remainder of the century it was in a steady decline with localised minor epidemic occurrences (Cumpston 1989: 301). The disease was thus never a major threat to the Aboriginal settlements.

Finally, there was one reported death from congenital syphilis. Syphilis had been one of the major causes of morbidity and mortality among Aboriginal people since the arrival of European colonists (Chapter Four). There are frequent references to the disease in the historical literature from all three mainland colonies in Southeast Australia. The majority of these references are to the disease existing in Aboriginal populations living in regions bordering the European settlement frontiers. Syphilis had been reported as a cause of death on at least one of the settlements prior to 1876. In 1876, J. Green, the

former manager of the Coranderrk settlement, reported that during the first 14 years after its establishment there were approximately 51 deaths in total:

> Fifteen of those who died came to the station sick with a complication of diseases, principally pleuro [sic] and syphilis. Four died of old age, two were killed, and one drowned, and several others (29) were born while their mothers were suffering from the effects of syphilis (four at the very least), in all twenty-six (26), leaving twenty five (25) to have died of diseases that could be in any way ascribed to the climate at Coranderrk. And eleven (11) of the twenty-five were the children of syphilitic parents; they died of "pleuro", the other deaths were principally of low fever... nearly all the aborigines, old and young, when they first came to settle at Coranderrk, were suffering in some way or other from syphilitic disease... (Green 1877-78).

While similar situations probably existed on other settlements prior to 1876 there are no further records of death from syphilis on the settlements in Southeast Australia apart from the one incident above. This does not however mean that it was entirely absent. The case of congenital syphilis indicates that at least the mother of the child had the disease. Congenital syphilis in newborn infants and acquired tertiary syphilis in adults may have gone unnoticed or unrecorded if more recognisable disorders, or other secondary causes could be diagnosed as cause of death. Treponemal infection could then have been an underlying foundation predisposing for other and more fatal infections.

8.5 Infant mortality

So far the emphasis of this chapter has been on the identification and discussion of the principal diseases causing mortality among the Aboriginal populations of the settlements. An assessment will now be made of the general state of health of these populations. This will be done by way of calculating infant mortality rates (deaths in the first year of life)

8.5.1 Method

Infant mortality (deaths in the first year of life) has provided a common way of measuring the health, health services, and social progress of populations. This is largely because the baby, in its first year of life, is at a critical stage of disease risk and is entirely dependent on the care of others and their ability to provide a healthy and safe environment (Cox 1976:110). It follows then that measurements of death during this period of life will be an assessment of the general health state of the community concerned. This measurement does, however, have limitations in that it assumes that the period of greatest danger for children is only in the first year and that it is the same for each community examined.

Infant mortality for Aboriginal and non-Aboriginal populations were calculated as:

$$\frac{\text{Deaths under age one for period}}{\text{Live births during period}} \times 1000$$

In addition to the infant mortality rates a rate ratio was calculated to show the differential between Aboriginal and non-Aboriginal infant mortality.

The differential rate ratio was calculated as:

$$\frac{\text{Non-Aboriginal infant mortality}}{\text{Aboriginal infant mortality}}$$

The Aboriginal and non-Aboriginal infant mortality rates and differential rate ratios were compared with corresponding twentieth century rates.

8.5.2 Results and discussion

Table 8.22 displays the infant mortality rate of Aboriginal people living on the settlements in the period compared with other pre-twentieth and twentieth century world populations.

Table 8.22 Infant mortality rates of Aboriginal people living on settlements in Victoria and South Australia 1876-1900 compared with selected pre-20th and 20th century populations.

Population	Period	IMR (deaths per 1000)	Reference
pre 20th Century			
Aboriginal settlements Victoria & South Australia	**1876-1900**	**184**	This study
Non-Aboriginal Victoria	1876-1900	119	*Statistical Register of Victoria* 1876-1900
Non-Aboriginal South Australia	1876-1900	118	*Statistical Register of South Australia* 1876-1900
Six London Parishes	1690s	185	Landers 1990
London	1870-1879	152	Stockwell 1993
English towns	1870-1879	174	"
East Prussia	1876-1895	220	Vogele 1994
West Prussia	1876-1895	230	"
20th Century			
South Australian Aborigines	1986-1988	20	Thomson & Briscoe 1991
Queensland Aborigines (settlement communities)	1972-1988	44	"
Northern Territory Aborigines	1972-1988	49	"
Central Australian Aborigines	1980-1982	56	"
Australia	1986-1988	9	"
Papua New Guinea	1946	251	van de Kaa 1967
	1980	78 (m) 66 (f)	Bakker 1983
Irian Jaya	before 1921	202	Groenewegen & van de Kaa 1965
Islands Papua New Guinea	1949-1952	189	Scragg 1969
	1962-1967	49	
Japan	1924	166	Louden 1991
U.S.	1990	10	Stockwell 1993
Northern Europe	1990	10	"
Ethiopia, Guinea, Mali, Sierra Leone		>150	"

The Aboriginal infant mortality rate of 184 deaths per 1000 is higher (1.5-1.6) than both the non-Aboriginal Victorian and South Australian rates. In comparison with the Victorian and South Australian non-Aboriginal populations the rate reveals that sixty-five more Aboriginal infant deaths occurred for every 1000 live births. However, when compared with other communities, for example, 17th century London, 19th century Prussia, Irian Jaya (West Irian) before 1920, and mid 20th century Papua New Guinea islands, the Aboriginal infant mortality is not especially high. The infant mortality rate indicates that the Aboriginal people on the settlements were less healthy than the non-Aboriginal Victorians and South Australians but healthier than say the West Irian communities under Dutch colonial rule. The infants and children of West Irian had to cope with similar introduced diseases but had an added risk with a high infection rate from naturally occurring malaria. Thomson (1990: 7; 1991: 235) has pointed out a similar pattern in the levels of 20th century Aboriginal infant mortality which show rates approximately three to four times higher than other Australians but relatively low compared to some European, Asian and Pacific populations.

The high infant mortality among the Aboriginal children is not fully explainable with the absence of health records of the Aboriginal people living on the settlements. These do not exist in any comprehensive form. But among the prime contenders for the extension of mortality, and by nature morbidity, into early childhood is a combination of malnutrition and diarrhoeal infection.

There is little to be gained in attempting to reach a reliable assessment of the nutrient value of the foods consumed by the Aboriginal people on the settlements last century. Little quantitative data is available in the historic records and that which does occur is vague and often unreliable. Even recent attempts at quantifying and assessing the daily nutrient intake of contemporary Aboriginal communities over varying periods have

been beset with problems of methodology and inaccuracies. What meaningful assessments have been made are of an indirect nature usually centred on comparative measures of growth and weight retardation among children and adults (e.g. Cheek et al. 1989; Cameron and Debelle 1986; Gracey et al. 1983; Gracey and Sullivan 1987; Kamien 1976; Kirke 1969; Maxwell & Elliot 1969; Roberts *et al.* 1988). What such studies have unanimously determined is that Aboriginal nutrition is substantially below that of the European communities throughout Australia and Aboriginal children suffer from growth retardation and high rates of infection and intestinal parasites (Thomson 1982).

On the nineteenth century settlements the Aboriginal people were almost totally reliant upon government issued food which was controlled by the supervisor or missionary in charge of each settlement. Flour, sugar, tea, rice, and tobacco were the mainstays of the daily diet, providing bulk but little protein. The meat, mainly beef and lamb, which was supplied to the settlements by private contractors was often of poor quality, principally bone, and was sometimes diverted by the supplier for sale elsewhere (Murray 1898). The cultural practice of sharing food among those not eligible under government legislation to receive rations further reduced the available intake (Murray 1898). In the early years of each settlement the Aboriginal people could supplement the government diet with endemic foods collected form available lands surrounding the settlement. But as the land was taken up by Europeans for grazing, the endemic food sources rapidly declined and became unavailable to the majority of the settlement residents. The result was a nutritionally poor diet, high in bulk, high in refined carbohydrates, and low in protein, fibre and essential fatty acids.

Much of the diarrhoea and dysentery recorded as cause of death in the first two years of life (Table 8.9) on the settlements quite likely occurred during the weaning period of the Aboriginal children. A trend showing substantial mortality in the first year

of life and extending into the early childhood period is demonstrative of the weaning process taking place in an undernourished environment (Winikof 1980:117). Little, however, is known about the weaning practices on the settlements. There is no evidence available to indicate at what ages children were weaned, how this may have differed over time or between settlements, how much influence the European supervisors and medical practitioners had in the process, nor what cultural beliefs were involved. But whatever the case, the Aboriginal children living on the settlements were subjected to an extended period of health risk that was largely, if not wholly, due to poorer nutrition and living standards.

Table 8.23 shows the relative difference (Aboriginal IMR / European IMR) between nineteenth and twentieth century Aboriginal and European infant mortality rates.

Table 8.23 Infant mortality rates and differential rate ratios between nineteenth and twentieth century non-Aboriginal and Aboriginal populations (*Statistical Register of Victoria* 1876-1900; *Statistical Register of South Australia* 1876-1900; Thomson & Briscoe 1991).

Population	Period	IMR	Differential rate ratio
19th Century			
Non-Aboriginal Victoria	1876-1900	119	
Non-Aboriginal South Australia	1876-1900	118	
Aboriginal Settlements	1876-1900	184	1.5-1.6
20th Century			
Australia	1986-1988	9	
South Australian Aborigines	1986-1988	20	2.2
Queensland Aborigines (settlement communities)	1972-1988	44	4.9
Northern Territory Aborigines	1972-1988	49	5.4
Central Australia Aborigines	1980-1982	56	6.2

The Aboriginal infant mortality rate shows a substantial improvement from the last quarter of the 19th century to a corresponding period in the 20th century. The rate shows a nine-fold improvement over the period from 1876-1900 (184) to that of South Australia for 1986-1988 (20). Comparative rates from Aboriginal settlements in Queensland for the period 1972-1988 show a more than four-fold improvement over the nineteenth century settlements of Victoria and South Australia (184 to 44). Infant mortality for the European population also showed improvement but at a much higher level. Comparison of the nineteenth century rates of 119 and 118 for non-Aboriginal Victoria and South Australia respectively and the rate of 9 for Australia in 1986-1988 shows a thirteen-fold improvement.

The differential ratios between the infant mortality rates reveal that while the general health of the two populations has improved from the nineteenth to the twentieth century the improvement has been much stronger in the European populations. Calculations on recent data confirm this trend.. Thomson (1990,1991) and Thomson & Briscoe (1991) have shown that Aboriginal people are the least healthy identifiable sub-population in Australia, with death rates two to four times higher than the total Australian population. What the above infant mortality rates reveal is that this has been the case for at least the last 125 years (and probably longer). Despite the many attempts by state and commonwealth governments to improve the health status of the Aboriginal people the health differential between the two populations has widened.

8.6 Conclusions

Infectious diseases and respiratory diseases were the major disease categories recorded as cause of death among the Aboriginal populations living on settlements. Together they accounted for just over one half (53.4%) of the total deaths on the 8

stations between 1876 and 1900. For the three age ranges combined infectious diseases had a PMR of 33.6 and respiratory diseases a PMR of 19.8. There were no significant differences between these two groups in the 0 to 4 years age group but in the two later age ranges, 5 to 14 years and 15+ years, the infectious disease category (PMR 54 & PMR 33.7 respectively) was responsible for more deaths.

The major causes of death were from tuberculosis, bronchitis, pneumonia, diarrhoea and dysentery all of which were endemic on the 8 settlements.

The major disease in the infectious disease category was tuberculosis. It was responsible for 35.0% of all deaths in the 5 to 14 years age range and 26.7% in the 15+ age range. In the 5 to 14 years group female deaths from tuberculosis outnumbered the males by almost 3 to 1. The higher mortality in females may be a result of their suspected increased susceptibility to the development of life-threatening symptoms of the disease during the onset of menarche and/or to an increased period of exposure to other sufferers. There was no sex differential in mortality from tuberculosis in the 15+ years group but most of the deaths occurred before 35 years of age.

Diarrhoea and dysentery were major causes of death in the 0 to 4 years age range particularly among infants and young children between 0 and 2 years of age The living conditions on the settlements characterised by a lack of a plentiful supply of clean water, inadequate sewerage, poor food and personal hygiene, poor levels of nutrition, and inadequate health care were the factors largely responsible.

Bronchitis and whooping cough were the main respiratory diseases among the 0 to 4 years age range. Bronchitis deaths showed a typical pattern of high mortality during the first 2 years of life, levelling off during childhood and early adulthood, then

climbing to high frequencies after 45 years of age. In the 0 to 4 years age range deaths from bronchitis were more common during the first year of life. Predisposing factors for acute and life-threatening symptoms for these early ages would include the unfavourable socioeconomic circumstances and poor nutrition experienced by most Aboriginal people on the settlements. A further predisposing factor would have been the dwellings which were often dusty in summer and damp during the winter. Passive tobacco smoke inhalation would have been common among new born and young Aboriginal children, further promoting the onset of bronchitis. A sharp increase in deaths from bronchitis occurred after age 45. This trend is quite likely to have been a result of chronic bronchitis due to many years of tobacco smoking.

Pneumonia and inflammation of the lungs caused deaths in all age ranges but showed a pattern of increasing mortality in the 15+ years age range. Pneumonia was most probably a secondary complication of other disorders, particularly influenza infections aggravated by chronic tobacco inhalation and alcohol consumption both of which were common on the Aboriginal settlements.

Deaths from hydatid disease occurred in 6 of the 8 settlements. Ages of death ranged from 4 years to 56 years in both sexes with no discernible age/sex pattern emerging.

The environment in which the Aboriginal people lived and their particular socio-economic circumstances played a great part in the continued presence of the pathogens causing these diseases and their ability to move from one host to another. The confined living conditions in poorly ventilated, and often damp, houses favoured the transmission of tuberculosis. The same living spaces and the widespread practice of smoking tobacco facilitated bronchial infections. The poor sanitation and waste disposal

common to the settlements promoted enteric infection and was a strong factor in the high rates of childhood mortality from diarrhoeal diseases.

On the other side of the coin the environment of the settlements protected the Aboriginal inhabitants from other infectious agents. The five measles epidemics between 1880 and 1898 that spread through the European populations had little effect on the settlements. The small populations and the geographic separation of the settlements from larger European population centres, together with the cultural isolation of the Aboriginal people acted as buffers, protecting the settlements

Infant mortality was high when compared to the non-Aboriginal populations of Victoria and South Australia. This measurement reveals a substantial health differential between Aboriginal and European populations. Aboriginal infant mortality has improved into the latter quarter of the twentieth century but the corresponding improvement in European infant mortality has been at a higher rate. The gap between the health status of each has widened rather than narrowed over the last one hundred years.

CHAPTER NINE

Conclusions

This thesis has been the first attempt to investigate and assess the early post-contact disease environment of Aboriginal people in Southeast Australia. While other authors have approached this topic in various ways their combined works have highlighted serious gaps in our knowledge of this part of our history. The central aim of this thesis was to begin filling in those gaps.

A model of the changing medical circumstances in Aboriginal populations of Southeast Australia has been presented beginning with the arrival of the First Fleet in 1788 and covering the colonial period to 1900.

The major elements of change to Aboriginal morbidity, mortality and population decline were introduced epidemic diseases in the first stage of European contact and chronic endemic diseases during the settlement phase.

The first of the major epidemic diseases to enter the Aboriginal population of Southeast Australia was smallpox in 1789. Evidence for this epidemic suggests that it was confined mainly to the eastern coastal regions. Two other epidemics of smallpox have been recorded in 1828-31 and 1866. The three epidemics had little effect in terms of morbidity and mortality among the European population but in the immunologically unprepared Aboriginal populations it is reasonable to expect that 30% or more population loss occurred in areas reached by each epidemic.

Sexually transmitted diseases, particularly syphilis, spread rapidly among the Aboriginal populations following the establishments of the major European settlements Sydney (1788), Melbourne (1835) and Adelaide (1836). A second source of introduction was most likely from sealing and whaling crews visiting coastal South Australia sometime after 1803. The fulminating symptoms and high mortality from syphilis were commonly referred to in ethnohistorical texts. Supporting the written evidence is osteological evidence of tertiary syphilis along the Murray River. The pattern of infectivity and severity of syphilis seen in Southeast Australia is similar to other continents where the disease has been introduced for the first time.

Tuberculosis probably accompanied the arrival of the First Fleet in 1788 but little is known of it among the Aboriginal populations until the 1830s. The first medically identified cases of tuberculosis were from autopsies conducted in 1837 at Wybalenna Aboriginal settlement on Flinders Island. These cases showed extensive pulmonary and systemic miliary dissemination. In the second half of the nineteenth century tuberculosis became well established in Aboriginal populations. Mortality records on Aboriginal settlements between 1859 and 1900 reveal 19.9% of recorded deaths attributable to tuberculosis in its several forms. A higher mortality from the disease should be expected due to misdiagnosis of symptoms at death and unrecorded Aboriginal deaths. Changes in lifestyle and living conditions forced upon Aboriginal people were largely responsible for the establishment of tuberculosis in the populations of Southeast Australia.

From 1836 influenza epidemics among the European population of Southeast Australia paralleled that of the world pandemics. Epidemics among the Aboriginal populations showed a similar occurrence. Little is known of the effects of early influenza outbreaks but those of 1839 and 1847 were notably severe among the Aboriginal

Tasmanians on Flinders Island, and among the populations of southern and central Victoria respectively. Documentary evidence shows that Aboriginal populations suffered excessive morbidity and mortality when initially exposed to the influenza virus.

Changes in lifestyle and residential manner favoured a build up of case-to-case transmission resulting in a persistence of influenza between major epidemic episodes. By the 1850s influenza and pneumonia had become endemic in the Aboriginal populations.

The 1874-75 measles epidemic in Southeast Australia was part of a world pandemic. Records of this epidemic provide the best evidence regarding the morbidity and mortality of measles among the Aboriginal populations. Population loss of up to 20% resulted on some of the settlements reached by the epidemic. Other population centres escaped. Crude death rates up to 205 per 1000 population, while indicating excessive mortality, were much less than those from the same epidemic in Fiji which ranged from 137 to 755 per 1000 population. The experience of previous epidemics between 1834 and 1868 providing a substantial number of immunologically protected, can account for the differential in crude death rates. It cannot be ascertained when measles became endemic among Aboriginal population centres during the nineteenth century, but after the 1874-75 epidemic records indicate that measles was no longer a major disease. An exception was the 1898 outbreak in northern South Australia. This was the last epidemic of measles to seriously affect an Aboriginal population in the nineteenth century.

During the final phase of contact in the colonial period infectious and respiratory diseases were the major causes of death among Aboriginal residents on settlements in Southeast Australia. Together they accounted for 53.4% of all recorded

deaths. Tuberculosis was the major cause, responsible for 35.0% of all deaths in the 5 to 14 years age range and 26.7% in the 15+ age range. A differential in mortality of 3 to 1 for females and males occurred in the age range 5 to 14 years. High susceptibility during menarche and an increased period of exposure are suggested as reasons for the differential. Diarrhoea and dysentery were major causes of death in the 0 to 4 years age range. Settlement living conditions characterised by inadequate sewerage, lack of clean water, poor nutrition levels and inadequate health care were factors largely responsible. Bronchitis and whooping cough were the main respiratory diseases among the 0 to 4 years age range. A sharp increase in deaths from bronchitis occurred after age 45. Unfavourable socioeconomic circumstances, poor nutrition, active and passive tobacco smoke inhalation are seen as strong predisposing factors. Pneumonia caused deaths in all age ranges but showed a pattern of increasing mortality in 15+ age range. Chronic tobacco inhalation and alcohol consumption were predisposing factors.

Comparison of infant mortality between Aboriginal and European populations reveal a substantial health differential for the period 1876 to 1900. While both populations' health has improved, modern comparisons show that the gap between European and Aboriginal health has widened.

An afterword

At the beginning of this thesis I characterised it as beginning an investigation and assessment into the medical circumstances of Aboriginal populations following colonisation. I consider that still to be the case as I see much more work remains to be done on the subject. The obvious extension of this work would be into other regions of the continent not covered here - for example, the west and southwest coastal regions of

Australia, inland desert regions, and the tropical north of the continent. These regions are physically and climatically different from the Southeast and I would suspect the nature of introduced diseases may be different to what has been described here.

Another expansion of this work would be to examine Aboriginal morbidity and mortality on a more local scale. In many regions of the Southeast such a study would be difficult due to lack of archival documentation, but the records from Tasmania may have much to offer in this case. Although they are incomplete (what set of historical data is ever complete?) the birth and mortality records from Gun Carriage Island, Wyballena, The Lagoons, and Oyster Cove still require a detailed study. Most of these records have been published as an appendix by Plomley (1987: 915-947) and others are stored in the Archives Office of Tasmania in Hobart.

A further extension would be to examine the medical circumstances of the indigenous populations of South Africa and New Guinea during their colonial periods.

Appendix A

Autopsy reports diagnosed as tuberculosis

Flinders Island Aboriginal Station

20 August 1837
Female adult

Having examined the body of the deceased ... I beg obedience to your directions to forward for your information the opinion I have arrived at relative to the cause of her death consequent upon the examination. The first that presented itself on examining the chest of the deceased was the remains of a very extensive inflammation of the lungs which must have been of some months standing. The left lobe was firmly adherent to the investing membrane of the inner surface of the ribs, to the bag containing the heart with tubercles in every direction throughout the substance of the lung. The principal valve of the heart was partly cartilaginous (the effect perhaps of age). The right lobe had also formed considerable adhesions but not so firmly as the left. The bag containing the heart contained rather more than a healthy portion of fluid. The liver especially the right lobe was considerably enlarged with small caseous spots impacted in several places throughout its substance. The gall bladder considerably relaxed. The mucous membrane of the two firsts of the small intestines presented in several places and inflammatory appearance, the vessels being numerous and large. The rest of the abdominal viscera healthy. The brain also healthy excepting rather a large quantity of fluid in the left ventricle. From these appearances I beg to state I can arrive at no other opinion than that expressed in my memorandum of the 16th inst that is that she died of typhus niction or low nervous fever.

(Austin, A. 1837 Robinson Papers, ML A7067: 149-150)

30 December 1837
Male adult

In the cavity of the thorax there was general adhesions of the lungs; they were dense and heavy and covered externally with small hard tubercles, the same being found contained in their internal structure. Of the abdomen - on opening this cavity it was found full of water; the quantity was at least a gallon full. The surface of the small intestines and their appendages were literally studded quite thick with small tubercles mostly of a yellowish colour and containing pus. There were also a number of small patches of the same intestines in a state o gangrene and encircled with these tubercles. The surface of the liver also exhibited the same appearances. The spleen was considerably enlarged and of a quite soft and friable substance.

(Walsh, M. 1837 Robinson Papers, ML A7068: 225)

27 February 1838
Male adult

The body was quite emaciated prior to his dissolution and was in reality a living skeleton. On opening the cavity of the thorax the lungs presented chronic adhesions to the ribs, pericardium and sternum were for the greater part solid and hepatised, particularly the

upper lobe of the left lung, which contained two or three large patches of a white caseous consistence. Both lungs were thickly interspersed with small hard white lumps of a tuberculated nature, although none of them had assumed a puriform state. The pericardium was much enlarged and contained about six ounces of fluid.

The liver was of extraordinary size, was hard and adhered to the diaphragm. On separating this connection a large ulcer was discovered on its upper surface of a dirty gangrenous appearance; it contained a large quantity of blood in a fluid state, was thickly covered with white hard small lumps similar to those found in the lungs, which pervaded its internal structure also. The whole peritoneal lining of the intestines exhibited the same tuberculated appearance but much larger in size and containing a thick yellowish purulent matter. The spleen was of natural size but hard and solid and on cutting into exhibited the same tuberculous formation with the aforesaid viscera. The peritoneal sack contained about four pints of water. The kidneys appeared healthy.

(Walsh, M. 1838 Robinson Papers, ML A7069: 111-112)

12 May 1838
Male adult

The contents of the thorax formed one mass of compact chronic adhesions. The lungs adhered to the ribs and all surrounding membranes; there was no trace of mediastinum remaining. They were dense and heavy containing numerous hard small tubercles. The right lung contained a large abscess filled with purulent matter. There were chronic adhesions formed between the heart and pericardium.

In the abdominal cavity the liver of natural appearance. The gall bladder was much distended with black bile resembling tar both as to colour and consistence, which deeply stained the neighbouring parts. The spleen was remarkably small and firm. The kidneys were natural. The folds of the mesentery and omentum with the peritoneal appendages of the small intestines were thickly covered with round hard tubercles about the size of a pea of a whitish colour.

(Walsh, M. 1838 Robinson Papers, ML A7070: 53)

2 June 1838
Female adult

In the cavity of the thorax was found an effusion of colourless serum (about two English pints). The right lung adhered to the pleura but was otherwise pretty healthy. The left formed one uniform mass of purulent matter scarcely holding together but breaking down at the slightest touch, adhering to all the neighbouring parts; its anterior surface was covered with a thick coat of coagulated lymph. The heart appeared healthy.

In the cavity of the abdomen there was also found an effusion of clear serum of about four pints. The liver was of natural size but quite friable in substance and easily breaking down under the finger. The gall bladder was much distended with a deep orange coloured bile. The spleen also equally friable with the liver of the usual size. Nothing remarkable appeared in the other viscera. This old woman absolutely refused medical interference.

(Walsh, M. 1838 Robinson Papers, ML A7070: 85)

21 June 1838
female two years of age

The lungs formed adhesions to the pleura, ribs, sternum, diaphragm and pericardium on either side, were dense and hepatised, thickly interspersed with small white tubercles both externally and internally. The left lung contained three large cysts filled with a pale yellowish matter of a cheesy consistence.

The abdomen contained about a gallon of colourless serum. The liver was extremely enlarged and indurated; was thickly dotted externally with small tubercles similar to those found in the lungs; on cutting into [it] exhibited a dark yellowish colour. The bile was of a lighter colour and thicker consistence than natural. The spleen was quite indurated of a dark purple colour interspersed with large whitish tubercles which pervaded its entire substance and exhibited a most curious and singular specimen of pathology. The pancreas was also indurated and their posterior surface exhibited a large cyst of similar appearance to that found in the left lung. The mesenteric glands were considerably enlarged and indurated.

(Walsh, M. 1838 Robinson Papers, ML A7070: 131)

2 July 1838
Female seven years of age

The lungs were healthy except a few adhesions of the left of a chronic nature with two or three small specks on its posterior surface in an incipient state of suppuration. Heart and pericardium natural.

Liver much enlarged and considerably indurated and replete with tubercles on the external surface as well as in its internal structure. Bile of a pale straw colour.

Spleen remarkably diseased being much indurated and covered with large tubercles of a yellowish colour each surrounded with a bright scarlet circle or base on a dark purple ground; about the centre was found a large superficial abscess of a vesicular nature of about an inch in diameter filled with a viscid fluid of a yellowish green colour much resembling bile in appearance. The pancreas were indurated and contained a few small abscesses containing purulent matter. The intestines and appendages exhibited marks of chronic inflammation and were thickly interspersed with small hard yellowish tubercles. The mesenteric glands were much enlarged and indurated. There was an effusion of colourless serum of about six ounces in the abdominal cavity.

(Walsh, M. 1838 Robinson Papers, ML A7070: 131)

6 August 1838
Male adult

The cavity of the abdomen contained about four pints of water. The liver appeared healthy; on making an incision into it a number of tubercles were discovered. The gall bladder was smaller than natural and contained a black and thickish fluid. Spleen natural in external appearance but being divided was found tubercular. Pancreas natural.

In the cavity of the thorax was a serious effusion of about a pint, the whole contents of which formed one lump of adhesion and firmly incorporated with the ribs sternum and diaphragm. Both the lungs with the exception of a small portion of the upper and posterior lobe of the right lung which was light and crepitus, were solid heavy and hepatised, containing a number of cysts filled with a firm yellowish cheesy like substance. The heart and its envelope were concealed in the substance of the left lung. The heart was soft and flabby. The blood was quite fluid and the body and limbs flexible.

(Walsh, M. 1838 Robinson Papers, ML A7070: 183-184)

3 September 1838
Male three years of age

In the cavity of the abdomen there was an effusion of about one pint of serum. The mesenteric glands were considerably enlarged, contained cysts filled with concrete purulent matter. The liver was much enlarged and indurated. Contents of gall bladder natural. Spleen indurated and thickly dotted with tubercles containing a bright yellowish matter of a cheesy consistence. Pancreas indurated and partly in a semi-purulent state. The contents of the thorax adhered closely together and to all the surrounding parts. The right lung was externally hepatised, heavy and contained on division an extensive abscess filled with pus. It exhibited also in its substance numerous tubercles in a state of separation. The left lung was also much diseased, a small portion only of which was light and crepitus, it exhibiting many tubercles in the same state as the former.

This child has been weakly these twelve months and although about three years old had never been able to walk. The appetite was always considerable till within a few days of its death, the belly relaxed and the breath at all times was highly offensive.

(Walsh, M. 1838 Robinson Papers, ML A7070: 249)

29 October 1838
Female adult

Cavity of the thorax - adhesion of the lungs with the ribs, sternum, mediastinum, pericardium and diaphragm. Both lungs heavy [sic] hepatised and tuberculated externally; exhibiting a similar appearance internally except being considerably larger, forming cysts containing a thickish purulent like matter; only a small portion of the left lung was crepitus.

Abdomen - liver covered with small whitish tubercles; also thickly interspersed with the same internally but larger and of a yellowish colour. Spleen much enlarged, friable in substance, full of large yellowish tubercles throughout its entire substance. There was about two pints of serous effusion in the cavity of the peritoneum.

(Walsh, M. 1838 Robinson Papers, ML A7070: 315

9 November 1838
Female adult

General chronic adhesions of the lungs to all the surrounding parts; right lung tuberculated but otherwise rather light and crepitus; the left lung formed one entire mass of purulent matter. There was about three pints of serous effusion in the cavity of the abdomen. The abdominal viscera were natural; the kidneys were remarkably large but healthy.

(Walsh, M. 1838 Robinson Papers, ML A7070: 383)

17 December 1838
Female adult

Thorax. The lungs adhered to all the neighbouring parts being of a chronic description and of long standing; were dense covered with small whitish tubercles externally and thickly interspersed internally with the same but of a much larger size and in a state of suppuration; only a small portion of the upper lobe of the right lung was crepitus. The pericardium was full of water.

Abdomen. This cavity contained about a gallon of water. The liver formed adhesions to the right side of long standing; was covered with small tubercles and was otherwise of unnatural appearance. The bile was thickish of a dark brown colour. The spleen was enlarged, of a dark purple colour, full of tubercles both externally and internally of a large size than those of the liver or lungs. There was a firm adhesion of the upper surface of the spleen to the stomach. The kidneys were quite flabby. The other viscera natural except the appearance of tubercles all along the peritoneal adhesions of the intestinal canal.

(Walsh, M. 1838 Robinson Papers, ML A7070: 427)

Appendix B - Recorded causes of death on Aboriginal settlements - 1876 to 1900

Settlement	Date	Sex	Age	Cause of Death	ICD
Framlingham					
Victoria	**1876**	?	17	Consumption	INF
	1877	?	45	Scarlatina	INF
		M	50	Hydatids	INF
		?	90	Senility	ILL
	1878	M	80	Senility	ILL
		M	80	Senility	ILL
	1879				
	1880	M	?	Inflammation of lungs	RES
		M	40	Inflammation of lungs	RES
		F	8	Consumption	INF
		F	10	Inflammation of lungs	RES
	1881				
	1882	M	55	Enlargement of liver	DIG
		M	50	Disease of heart	CIR
		F	45	Enlargement of liver	DIG
		M	60	Enlargement of liver	DIG
	1883	?	0	Inflammation of lungs	RES
		M	14	Chronic enlargement of the liver	DIG
		M	50	Phthisis	INF
		M	55	Phthisis	INF
Lake Condah	**1876**	M	30	Inflammation of lungs	RES
Victoria		M	50	?	ILL
		M	60	Old Age	ILL
		M	45	Consumption	INF
	1877	M	17	Chronic dysentery	INF
		F	40	Inflamation of bowels	DIG
		M	65	Old Age	ILL
		M	50	Paralysis	NER
		M	30	Typhoid Fever	INF
	1878	M	40	Inflammation of the lungs	RES
		M	45	Congestion of the lungs	RES
	1879	M	0	Suffocation	INJ
	1880	F	24	Consumption	INF
		M	17	Consumption	INF
		M	5	Hydrocephalus	NER
		F	12	Consumption	INF
		F	2	Consumption	INF
		M	0	Haemorrhage	PER
	1881	M	0	Bronchitis	RES
		M	70	Inflammation of the lungs	RES
		F	14	Consumption	INF
		F	18	Tuberculosis	INF
		F	50	General decay	ILL
	1882	M	0	Pneumonia	RES
	1883	F	0	Teething	ILL
		F	45	Peritonitis	DIG
	1884	M	75	Old age	ILL

Settlement	Date	Sex	Age	Cause of Death	ICD
		F	12	Congestion of the lungs	RES
		F	48	Asthma	RES
		F	1	Injury from fall	INJ
		M	0	Pneumonia	RES
		F	12	Consumption	INF
	1885	F	1	Convulsions	NER
		F	1	Disease of throat and mouth	ILL
		F	10	Tuberculosis	INF
		M	60	Peritonitis	DIG
	1886	F	0	Whooping cough	RES
		M	2	Whooping cough	RES
		M	2	Pneumonia whooping cough	RES
	1887	M	26	Pleurisy and lung congestion	RES
		F	46	Chronic disease of liver	DIG
	1888	M	50	Peritonitis	DIG
		M	50	Chronic disease of stomach	DIG
		F	16	Consumption	INF
	1889	F	0	Congestion of the lungs	RES
		F	3	Chronic bronchitis	RES
		F	33	Consumption	INF
		M	47	Consumption	INF
		F	13	Consumption	INF
		F	12	Extensive burns	INJ
		M	0	Bronchitis	RES
		F	25	Phthisis	INF
		F	12	Phthisis	INF
		M	60	Disease of stomach	DIG
		M	22	Phthisis	INF
		F	54	Bronchitis	RES
	1890	M	90	Old age	ILL
		M	2	Chonic bronchitis	RES
		M	50	Phthisis	INF
		M	53	Congestion of the lungs	RES
		M	60	Congestion and bronchitis	RES
	1891	M	78	Heart disease	CIR
		F	0	Premature birth	PER
		F	45	Fatty degeneration of heart	CIR
	1892	M	24	Drowning	INJ
	1893	F	65	General decay	ILL
		M	56	Blood poisoning	ILL
		F	32	Bronchio-pneumonia	RES
		M	62	Dyspepsia	DIG
	1894	F	0	Exhaustion and intense heat	ILL
		F	30	Tuberculosis	INF
		M	70	General decay	ILL
		F	1	Pneumonia	RES
		M	67	Congestion of the lungs	RES
	1895	F	2	Whooping cough	RES
	1896	F	25	Phthisis	INF
		M	18	Phthisis	INF
		M	36	Laryngitis	RES

Settlement	Date	Sex	Age	Cause of Death	ICD
		F	1	Bronchitis	RES
		F	60	Phthisis	INF
	1897	F	15	Tubercular peritonitis	INF
		F	19	Phthisis pulmonatis	INF
		F	80	Senile decay	ILL
		M	5	Acute phthisis	INF
		F	12	Phthisis pulmonatis	INF
		M	18	Phthisis pulmonatis	INF
	1898	M	6	Tubercular peritonitis	INF
		F	56	Chronic bronchitis	RES
		F	23	Phthisis	INF
		F	11	Phthisis	INF
		F	12	Phthisis	INF
		M	7	Pneumonia	RES
		F	3	Phthisis	INF
	1899				
	1900	M	19	Tuberculosis	INF
		M	60	Peripheral neuritis	NER
		M	75	Cancer of the bladder	NEO
Lake Wellington (Ramahyuck) Victoria	**1876**	M	25	Abscess	ILL
		?	49	Disease of heart	CIR
		F	60	Old Age	ILL
	1877	?	40	Consumption	INF
		M	1	Teething	ILL
		F	18	Consumption	INF
		F	7	Peritonitis	DIG
		F	5	Peritonitis	DIG
		M	8	Whooping Cough	RES
	1878	M	16	Consumption	INF
		F	14	Lung disease	RES
		F	30	Abcess	ILL
		M	46	Brain fever	NER
	1879	M	3	Bronchitis	RES
		?	0	Teething	ILL
		F	12	Consumption	INF
		F	70	Old age	ILL
		F	0	Teething	IKLL
		F	18	Consumption	INF
	1880	F	58	Inflammation of the bowels	DIG
		F	45	Inflammation of the bowels	DIG
		M	54	Drowning	INJ
		F	54	Consumption	INF
	1881	M	0	Weakly child	ILL
		M	0	Inflammation of the lungs	RES
		?	70	Old age	ILL
		?	46	Inflammation of the lungs	RES
		M	32	Consumption	INF
		F	29	Bursting blood vessel	CIR
		F	65	Old age	ILL

Settlement	Date	Sex	Age	Cause of Death	ICD
		M	21	Consumption	INF
		M	40	Inflammation of the lungs	RES
	1882	F	3	Lung disease	RES
		M	1	Bronchitis	RES
		M	54	Dropsy	ILL
	1883	F	19	Inflammation of the bowels	DIG
		F	0	Bronchitis	RES
		F	14	Consumption	INF
		M	6	Consumption	INF
		F	1	Bronchitis	RES
	1884	M	27	Inflammation of lungs	RES
		M	56	Abscess	ILL
		F	5	Consumption	INF
		F	45	Hydatids	INF
		F	10	Inflammation of the bowels	DIG
	1885	F	10	Hydatids	INF
		M	1	Teething	ILL
		M	9	Pneumonia	RES
		M	3	Dropsy	ILL
		F	10	Consumption	INF
	1886	F	12	Consumption	INF
		M	47	Spinal disease	ILL
		F	54	Inflammation of the lungs	RES
		M	0	Bronchitis	RES
	1887	M	56	Disease of heart	CIR
		F	16	Inflammation of heart	CIR
	1888	M	24	Consumption	INF
		M	22	Consumption	INF
		F	?	Teething	ILL
	1889	F	79	Old age	ILL
		M	22	Consumption	INF
	1890	F	26	Spinal disease	ILL
		M	41	Phthisis	INF
	1891	M	59	Inflammation	ILL
	1892	M	1	Teething	ILL
		F	?	Old age	ILL
	1893	M	?	Old age	ILL
	1894	F	1	?	ILL
		M	1	Measles	INF
		M	14	Spinal disease	ILL
		M	70	Old age	ILL
		F	0	Measles	INF
		M	68	Inflammation of lungs	RES
		F	2	Heart failure	CIR
		F	?	Bronchitis	RES
		F	?	Influenza	RES
		F	?	Whooping cough	RES
	1895	F	42	Heart disease	CIR
	1896	F	0	Teething	ILL
		F	25	Consumption	INF
		F	0	Teething	ILL

Settlement	Date	Sex	Age	Cause of Death	ICD
		M	1	Decline	ILL
		F	22	Fits	ILL
		F	57	Consumption	INF
		F	2	Decline	ILL
		M	24	Inflammation of the bowels	DIG
	1897	F	22	Exhaustion	PRG
	1898	M	26	Consumption	INF
	1899	M	67	Cancer	NEO
	1900				
Lake Tyers	**1878**	M	50	Hepatitis	
Victoria		F	70	General decay	ILL
		M	60	Inflammation of the lungs	RES
		F	65	Inflammation of the lungs	RES
		M	50	Inflammation of the lungs	RES
		M	5	Inflammation of the lungs	RES
		M	40	Gastric fever	DIG
		F	45	Inflammation of the lungs	RES
		M	60	Cancer of the tounge	NEO
	1879	M	19	Inflammation of the lungs	RES
	1880				
	1881	M	2	Dysentery	INF
		M	14	Pneumonia	RES
		M	0	Bronchitis	RES
	1882	F	80	Senile decay	ILL
		F	1	Theething	ILL
		M	0	Erysipelas	
		M	50	Dropsy of the extremities	ILL
	1883	F	80	Senile decay	ILL
		M	86	Senile decay	ILL
		F	1	Bronchitis	RES
	1884	F	56	Tumified liver	DIG
		F	1	Bronchitis	RES
		F	5	Pneumonia	RES
		F	65	Inflammation of lungs	RES
		F	1	Teething	ILL
		M	45	Heart disease	CIR
	1885	M	1	Teething	ILL
		M	50	Inflammation of brain	NER
		F	19	Phthisis & spinal congestion	INF
		F	0	Premature birth	NEO
		F	65	Phthisis pulmonias	INF
		M	3	Scrofula & suppuration in joints	INF
		M	70	General debility	ILL
	1886	M	76	Senile decay	ILL
		M	50	Drowned	INJ
		F	1	Teething	ILL
		M	54	Cancer of the intestines	NEO
	1887	F	1	Thrush	
		M	1	General deility since birth	ILL

Settlement	Date	Sex	Age	Cause of Death	ICD
		M	16	Disease of brain, Amaurosis	NER
		M	86	Senile decay	ILL
		F	3	Dysentery	INF
		M	0	Whooping cough	RES
		F	0	Whooping cough	RES
		F	3	Whooping cough	RES
		M	30	Hydatids of liver & Ascites	INF
	1888				
	1889	M	32	Pneumonia	RES
		M	0	Inflammation of the lungs	RES
		F	1	Diarrhoea	INF
		M	0	Diarrhoea	INF
		F	0	Congenital syphilis	
		F	32	Phthisis	INF
		M	11	Cerebral tumor	NER
		M	50	General debility	ILL
	1890	F	20	Chronic dysentery	INF
		F	25	Phthisis	INF
		M	0	Influenza	RES
		M	0	Influenza	RES
		F	3	Tuberculosis	INF
		M	1	Bronchitis	RES
	1891	M	60	Heart disease	CIR
		M	11	Injury to head & menengitis	INJ
		F	20	Cirrhosis of liver & dropsy	DIG
		M	50	General debility	ILL
		M	3	Tabes mesentrica	INF
	1892	F	0	Bronchitis	RES
		M	1	General debility	ILL
	1893	F	39	Laryngitis	
		F	39	Pneumonia following measles	
		F	0	Diarrhoea	INF
		M	30	Acute Phthisis following measles	
		F	0	Diarrhoea	INF
	1894	F	65	General debility following measles	
	1895	M	80	Senile decay	ILL
	1896	M	1	Influenza	RES
	1897				
	1898	M	1	Sunstroke	INJ
		F	7	Chorea, abcess on brain	NER
	1899	M	1	Diarrhoea & vomiting	INF
		F	60	Phthisis	INF
		F	57	Disease of brain, heart failure	NER
		M	22	Bursting of internal abcess	ILL
		M	46	Influenza & pneumonia	RES
	1900	M	4	Diarrhoea	INF
		M	6	Diarrhoea	INF
		F	76	Influenza, senile decay	RES
Coranderrk	**1878**	M	21	Consumption	INF

Settlement	Date	Sex	Age	Cause of Death	ICD
Victoria		M	3	Consumption	INF
		M	5	Consumption	INF
		F	16	Softening of the brain	NER
		F	25	Consumption	INF
		M	35	Consumption	INF
		?	0	Low fever	INF
	1879	F	20	Consumption	INF
		M	45	Hydatids	INF
		F	5	Convulsions	NER
		M	0	Bronchitis	RES
		F	26	Hydatids	INF
	1880	F	0	Convulsions	NER
		F	13	Convulsions & dropsy	NER
		M	30	Consumption	INF
		M	35	Consumption	INF
		M	35	Hydatids & Phthisis	INF
		M	0	Bronchitis	RES
	1881	F	35	Consumption	INF
		M	14	Consumption	INF
		M	50	Hydatids	INF
		M	13	Consumption & Dropsy	INF
	1882	F	0	Dentition	ILL
		M	13	Consumption	INF
		M	1	Dentition	ILL
		F	7	Rheumatic fever	INF
	1883				
	1884	F	10	Consumption	INF
		F	60	Bronchitis	RES
		M	35	Gunshot wounds	INJ
		?	59	Paralysis	NER
		F	11	Consumption	INF
	1885	M	16	Consumption	INF
		F	5	Consumption	INF
		F	0	Bronchitis	RES
		?	8	Burns	INJ
	1886	F	45	Consumption	INF
		M	75	Bronchitis	RES
		M	12	Bronchitis	RES
		M	30	Consumption	INF
	1887	F	54	Dropsy	ILL
	1888	M	1	Bronchitis	RES
	1889	F	1	Diarrhoea	INF
		F	0	Bronchial-pneumonia	RES
		M	8	Acute rheumatism	ILL
		F	1	Diarrhoea	INF
	1890	F	7	Acute rheumatism	ILL
		F	7	Diarrhoea & exhaustion	INF
	1891	M	56	Heart disease	CIR
		M	70	Bronchitis	RES
		F	23	Influenza	RES
	1892	F	60	Senile decay	ILL

Settlement	Date	Sex	Age	Cause of Death	ICD
		F	0	Convulsion fits	NER
		M	0	Bronchitis	RES
	1893	F	51	Heart failure	CIR
		M	50	Spinal caries	ILL
		F	0	Diarrhoea	INF
	1894	M	70	Injury in fall	INJ
		F	50	Chronic bronchitis	RES
	1895	M	2	Bronchial pneumonia	RES
		F	21	Consumption	INF
	1896	M	?	Phthisis	INF
		F	70	General decay	ILL
	1897	F	3	Pneumonia	RES
	1898	F	25	Phthisis	INF
		F	45	Septic poisoning	ILL
		F	50	Cancer	NEO
	1899	F	2	Bronchitis	RES
		F	58	Cancer	NEO
	1900	M	3	Measles	INF
		F	62	Pneumonia	RES
		M	56	Hydatids	INF
Ebenezer	**1876**	F	40	Consumption	INF
(Lake Hindmarsh)		M	40	Heart Disease	CIR
Victoria		F	35	Consumption	INF
		M	10	Drowning	INJ
		F	10	Stomach Complaint	DIG
		M	0	Convulsions	NER
		F	29	Inflammation of the lungs	RES
		M	65	Heart disease	CIR
	1877	F	16	Enteritis	INF
		F	70	Tumor	NEO
		M	40	Inflammation of the lungs	RES
	1878	F	10	Consumption	INF
		F	1	General debility	ILL
	1879	F	16	Consumption	INF
		F	19	Dropsy	ILL
		M	32	Hydatids	INF
		F	28	Lung disease	RES
		M	13	Bronchitis	RES
		F	27	Consumption	INF
		M	36	Consumption	INF
		F	14	Dropsy	ILL
		M	70	Paralysis	NER
	1880	M	4	Marasmus	END
		M	1	Stomach complaint	DIG
		F	6	Consumption	INF
		?	100	Senile decay	ILL
		F	21	Drowning	INJ
		M	7	Marasmus	END
		M	22	Consumption	INF

Settlement	Date	Sex	Age	Cause of Death	ICD
	1881	M	20	Consumption	INF
		?	36	Tumor	NEO
		?	3	Marasmus	END
		F	26	Consumption	INF
		M	45	Disease of brain	NER
	1882	M	0	Teething	ILL
		M	60	Consumption	INF
		F	1	Dentition	ILL
		F	45	Dropsy	ILL
	1883	F	68	Bronchitis	RES
		M	19	Fever	ILL
		F	0	Convulsions	ILL
		F	3	Congestion of brain	NER
		M	40	Liver complaint	DIG
		M	0	Convulsions	ILL
		F	1	Bronchitis	RES
		F	25	Consumption	INF
		F	1	Dentition	ILL
		?	1	Dentition	ILL
	1884	F	60	Liver complaint	DIG
		F	0	Dentition	ILL
		F	14	Consumption	INF
		M	3	Peritonitis	DIG
	1885	M	87	Senile decay	ILL
		F	11	Consumption	INF
		M	0	Bronchitis	RES
		M	0	Dentition	ILL
	1886	F	0	Whooping cough	RES
		M	1	Whooping cough	RES
		F	28	Fever	INF
		M	42	Lung disease	RES
		M	50	Tumor in chest	NEO
	1887	M	65	Bronchitis	RES
		M	80	Bronchitis	RES
		M	50	Bronchitis	RES
		M	65	Bronchitis	RES
		M	1	Dentition	ILL
	1888	M	65	Stomach complaint	DIG
		F	2	Marasmus	END
	1889	F	85	Bronchitis	RES
		M	60	Consumption	INF
		F	20	Consumption	INF
	1890	M	0	Congestion of stomach	DIG
	1891	F	80	Old age	ILL
		F	1	Dysentery	INF
	1892	M	1	Marasmus	END
	1893				
	1894				
	1895				
	1896	M	70	Cancer of leg and exhaustion	NEO
	1897	M	27	Consumption	INF

Settlement	Date	Sex	Age	Cause of Death	ICD
		M	2	Congestion of the lungs	RES
		M	6	Inflammation of stomach	DIG
		M	0	Bronchitis	RES
		M	20	Phthisis	INF
	1898				
	1899	F	0	Dentition	ILL
	1900				
Point McLeay	**1876**	?	75	Old age	ILL
South Australia		?	0	Teething	ILL
		M	0	Diarrhoea	INF
		M	28	Consumption	INF
		F	6	Marasmus	NEO
		F	30	Consumption	INF
		?	60	Old age	ILL
		?	0	?	ILL
		?	75	Old age	ILL
		F	42	Lung disease	RES
	1877	M	1	Atrophy	ILL
		?	65	Old age	ILL
		?	60	Heart disease	CIR
		M	0	Teething	ILL
		M	50	Stomach disease	DIG
		M	4	Pneumonia	RES
		M	24	Throat disease	ILL
		M	2	Diarrhoea	INF
		?	60	Consumption	INF
		M	60	Stomach disease	DIG
		M	60	?	ILL
		F	0	Diarrhoea	INF
		F	2	Lung disease	RES
		?	65	Old age	ILL
		M	37	Bright's Disease	GEN
		F	8	?	ILL
	1878	F	15	Enteritis	INF
		?	0	Dentition	ILL
		?	0	Atrophy	ILL
		M	1	Marasmus	END
		F	19	Consumption	INF
		F	3	Pneumonia	RES
		F	0	Whooping cough	RES
		M	1	Dropsy	ILL
		M	3	Pneumonia	RES
		M	2	Diarrhoea	INF
		F	2	Atrophy	ILL
		F	0	Bronchitis	RES
		F	39	Consumption	INF
		F	0	Atrophy	ILL
		M	35	Consumption	INF
		?	?	Old age	ILL

Settlement	Date	Sex	Age	Cause of Death	ICD
		F	8	Malignant stomachitis	ILL
		F	14	Consumption	INF
		F	0	Diarrhoea	INF
		F	0	Diarrhoea	INF
		M	1	Diarrhoea	INF
	1879	M	15	Shot	INJ
		M	60	Old age	ILL
		?	1	Marasmus	END
		F	2	Marasmus	END
		F	50	Consumption	INF
		?	0	?	ILL
		F	21	Puerperal Fever	PRG
	1880	F	11	Malignant stomachitis	ILL
		F	4	Pneumonia	RES
		F	18	Consumption	INF
		F	12	Enteric fever	INF
		F	0	Diarrhoea	INF
		M	40	Enteric fever	INF
		M	32	Hydatids	INF
		M	60	Old age	ILL
		F	45	Consumption	INF
		?	65	Senile decay	ILL
		F	1	Dysentery	INF
		F	0	Dysentery	INF
	1881	M	9	Peritonitis	DIG
		?	60	Senile decay	ILL
		M	50	Paralysis	NER
		?	60	Senile decay	ILL
		F	12	Liver disease	DIG
		M	0	Infantile convulsions	PER
		F	9	Peritonitis	DIG
	1882	M	30	Acute Dysentery	INF
		M	30	Chronic bronchitis	RES
		M	1	Hydrocephalus	NER
		F	1	Diarrhoea	INF
		F	2	Chronic diarrhoea	INF
		M	50	Senile decay	ILL
		?	50	Senile decay	ILL
		F	45	Paralysis	NER
		M		Acute pneumonia	RES
		M	11	Tabes mesenteric	INF
		F	0	Diarrhoea	INF
	1883	M	40	Epilepsy	NER
		?	70	Senile decay	ILL
		F	40	Consumption	INF
		M	1	Convulsions	NER
		M	6	Peritonitis	DIG
		M	1	Diarrhoea	INF
		?	0	Diarrhoea	INF
	1884				
	1885				

Settlement	Date	Sex	Age	Cause of Death	ICD
	1886				
	1887				
	1888	M	2	Consumption	INF
		M	70	Senile decay	ILL
		F	?	Diarrhoea	INF
		F	?	Consumption	INF
		F	?	Diarrhoea	INF
		M	70	Senile decay	ILL
		M	?	?	ILL
		F	?	Senile decay	ILL
	1889	M	70	Senile decay	ILL
		M	43	Consumption	INF
		M	13	Consumption	INF
		F	?	Senile decay	ILL
		M	?	Inflammation of brain	NER
		F	25	?	ILL
		M	?	?	ILL
		M	?	?	ILL
		M	?	?	ILL
	1890	F	?	Senile decay	ILL
		F	0	Dysentery	INF
		M	30	Accident	INJ
		F	0	?	ILL
	1891	?	?	?	ILL
		F	0	Teething	ILL
		M	0	Teething	ILL
		F	27	Hydatids	INF
		F	?	?	ILL
		F	21	Pueperal fever	PRG
		?	0	?	ILL
	1892	F	2	Atrophy	ILL
		?	0	?	ILL
		M	1	Diarrhoea	INF
		F	60	Senile decay	ILL
		?	0	?	ILL
		F	50	Consumption	INF
		F	1	Marasmus	END
		?	2	Consumption	INF
		M	4	Hydatids	INF
		M	0	Infantile convulsions	NER
		M	?	Heart disease	CIR
		F	70	Senile decay	ILL
		F	1	Cerebral affliction	NER
	1893	M	?	?	ILL
		M	?	Senile decay	ILL
		F	?	Senile decay	ILL
		?	1	Bronchitis	RES
		M	15	Inflammation of lungs	RES
		?	75	Senile	ILL
		?	0	Premature birth	PER
		M	90	Senile decay	ILL

Settlement	Date	Sex	Age	Cause of Death	ICD
		M	48	Bronchitis	RES
		M	70	Chronic bronchitis	RES
		F	70	Senile decay	ILL
		F	1	Measles	INF
		M	0	Thrush	INF
		F	0	Thrush	INF
		F	1	Thrush	INF
		F	60	Bronchitis	RES
		?	0	Thrush	INF
		M	0	Dysentery	INF
	1894	M	65	Dysentery	INF
		F	50	Liver inflammation	DIG
		M	11	Spinal disease	ILL
		F	70	Inflammation of bowels	DIG
		F	12	Inflammation of brain	NEO
		F	1	Dysentery	INF
		?	12	Spinal disease	ILL
		M	19	Consumption	INF
	1895	M	18	Diarrhoea	INF
		?	0	Premature birth	PER
		F	5	Intermittent fever	INF
		F	8	Intermittent fever	INF
		M	0	Meningitis	NER
		F	39	Consumption	INF
		M	1	Meningitis	NER
		F	16	Consumption	INF
		?	0	Marasmus	END
		F	12	Consumption	INF
		?	0	Marasmus	END
		F	56	Consumption	INF
		F	75	Senile decay	ILL
		M	54	Bronchitis	RES
		M	65	Senile decay	ILL
		M	1	Infammation of brain	NER
		M	60	Consumption	INF
	1896	F	60	Marasmus	END
		M	0	Scrofula	INF
		M	0	Diarrhoea	INF
		F	0	Diarrhoea	INF
		M	4	Meningitis	NER
	1897	F	60	Senile decay	ILL
		F	0	?	ILL
		M	1	Burns	INJ
		F	30	Heart disease	CIR
		F	30	Inflammation of bladder	GEN
		F	2	?	ILL
		M	4	?	ILL
		M	54	?	ILL
		?	0	Marasmus	END
		F	27	Consumption	INF
		M	8	Brain apoplexy	NER

Settlement	Date	Sex	Age	Cause of Death	ICD
		?	0	Marasmus	END
		F	0	Diarrhoea	INF
		M	65	Senile decay	ILL
		F	40	Cancer	NEO
		F	64	Apoplexy	NER
		M	75	Senile decay	ILL
		F	24	Consumption of bowels	DIG
	1898	F	0	Birth debility	PER
		F	0	Ulcerated throat	DIG
		F	1	Marasmus	END
		M	0	Premature birth	PER
		?	0	Birth debility	PER
		F	65	Acute bronchitis	RES
		F	0	Bronchitis	RES
		F	0	Whooping cough	RES
	1899	M	14	?	ILL
		M	0	Whooping cough	RES
		F	1	?	ILL
		F	50	Cancer	NEO
		?		Stillborn	PER
		F	52	Consumption	INF
		F	0	Dysentery	INF
		M	47	Consumption	INF
		M	0	Debility	PER
	1900	F	1	Dysentery	INF
		F	0	Influenza	RES
		F	0	Influenza	RES
		M	0	Influenza	RES
		F	0	Teething	ILL
		M	1	Teething	ILL
		F	0	Influenza	RES
		M	54	Cancer	NEO
Point Pearce	**1880**	M	34	Phthisis	INF
South Australia		M	19	Phthisis	INF
		F	50	Bronchitis	RES
	1881				
	1882	F	24	Phthisis	INF
		M	28	Phthisis	INF
		F	1	Teething	ILL
	1883	F	13	Hydatids	INF
		F	40	Bronchitis	RES
		M	52	Lung complications	RES
	1884	M	51	Phthisis	INF
		M	0	Bronchitis	RES
		F	2	Bronchitis	RES
		M	70	Senile decay	ILL
		F	22	Phthisis	INF
	1885	M	0	?	ILL
		F	32	Confinement	PRG

Settlement	Date	Sex	Age	Cause of Death	ICD
		F	13	Phthisis	INF
	1886	F	0	?	ILL
		F	27	Bronchitis	RES
		M	60	Senile decay	ILL
	1887	M	16	Phthisis	INF
		F	20	Phthisis	INF
	1888	M	0	?	ILL
	1889	M	1	Bronchitis	RES
		F	56	Senile decay	ILL
		M	50	Dropsy	ILL
		M	?	?	ILL
	1890	M	30	Phthisis	INF
	1891	M	1	Teething	ILL
		M	16	Typhoid	INF
		F	62	Paralysis	NER
		F	80	Senile decay	ILL
		F	6	Pulmonary	RES
	1892				
	1893	M	0	Heart complications	CIR
		F	6	Fever	INF
		F	0	Tubercular meningitis	NER
	1894	M	0	Whooping cough	RES
	1895				
	1896				
	1897				
	1898	F	13	Consumption	INF
		F	2	Scarlet fever	INF
	1899	F	11	Pneumonia	RES
		F	2	?	ILL
	1900	F	49	Pneumonia	RES

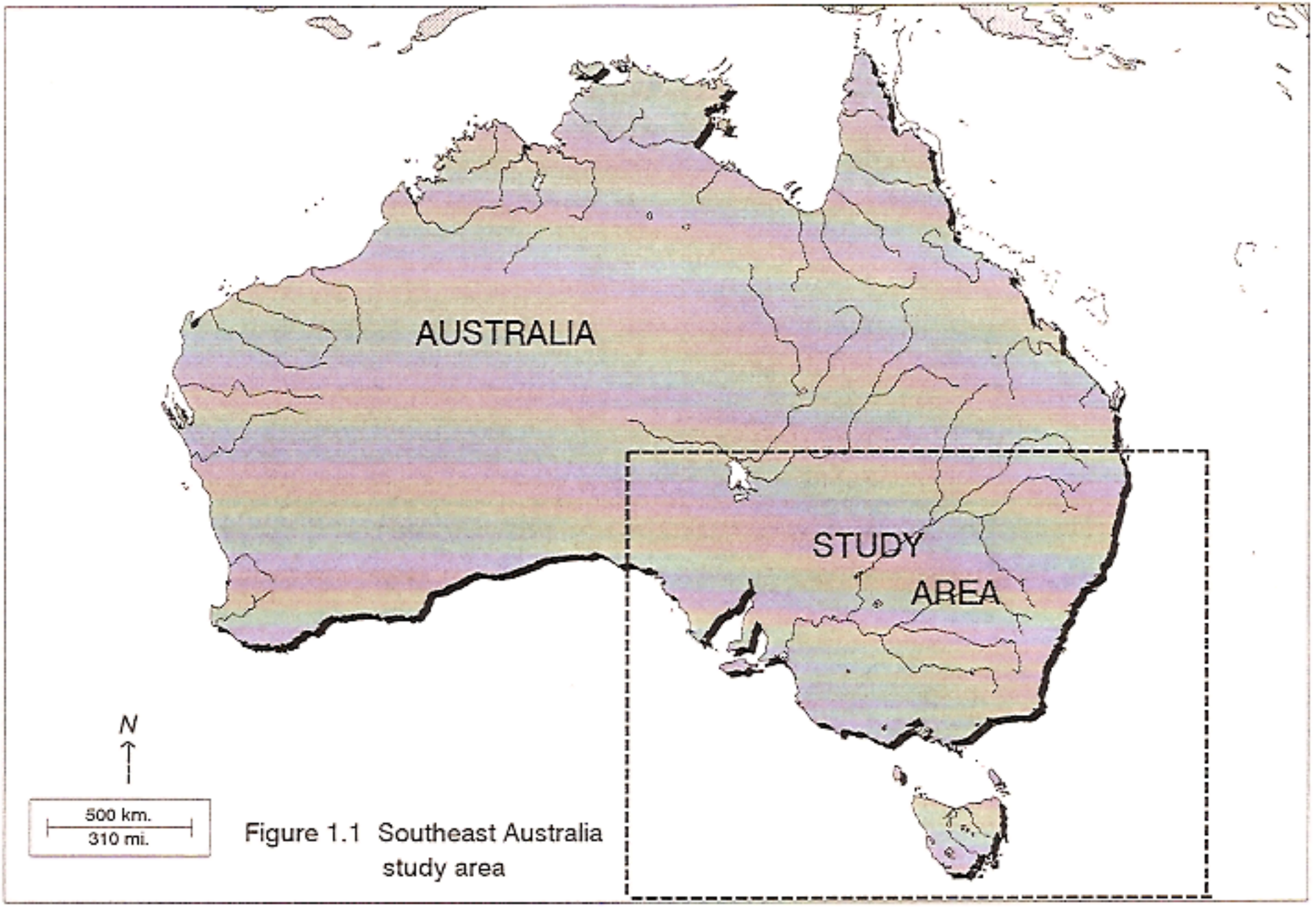


Figure 1.1 Southeast Australia study area

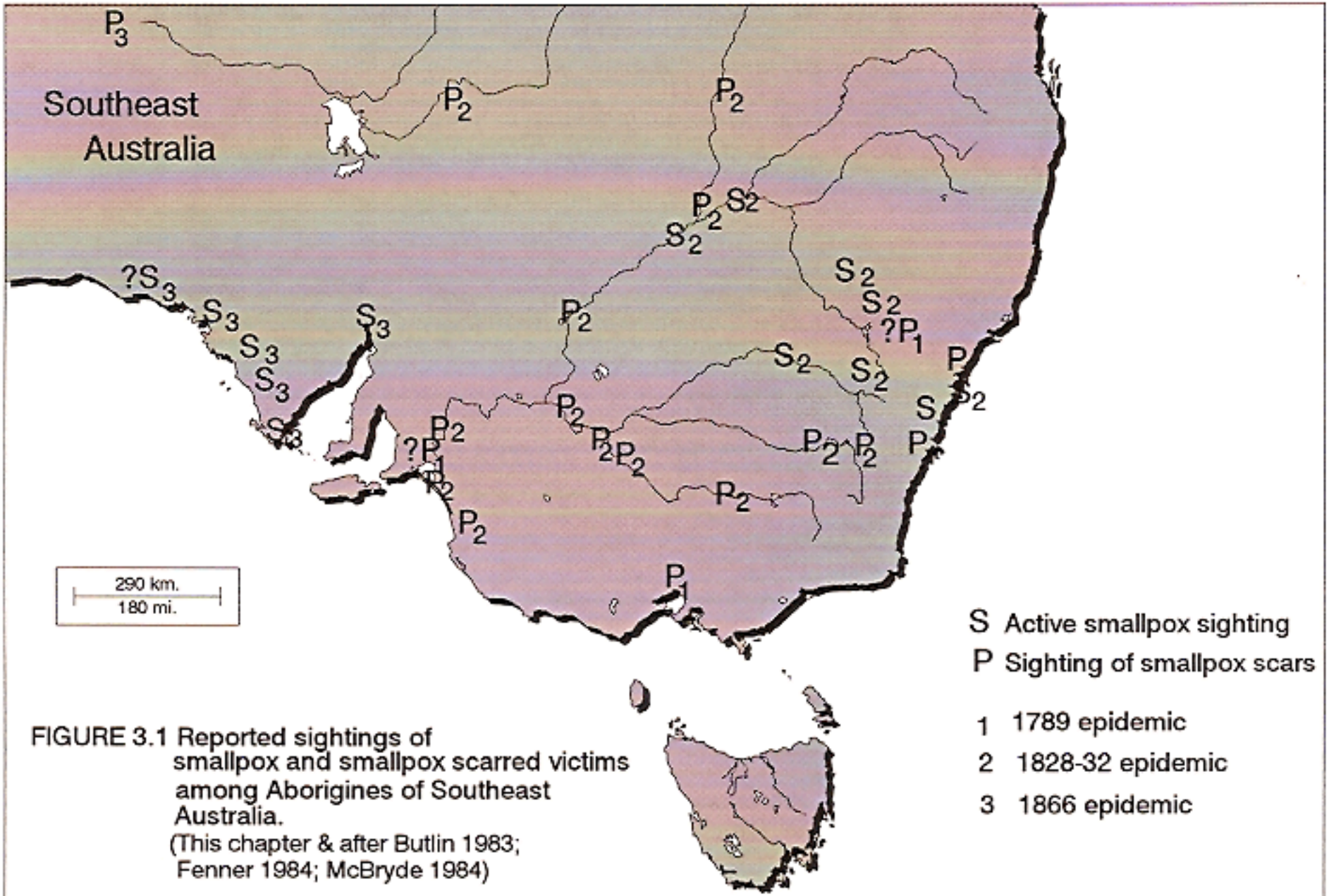


FIGURE 3.1 Reported sightings of smallpox and smallpox scarred victims among Aborigines of Southeast Australia.
(This chapter & after Butlin 1983; Fenner 1984; McBryde 1984)

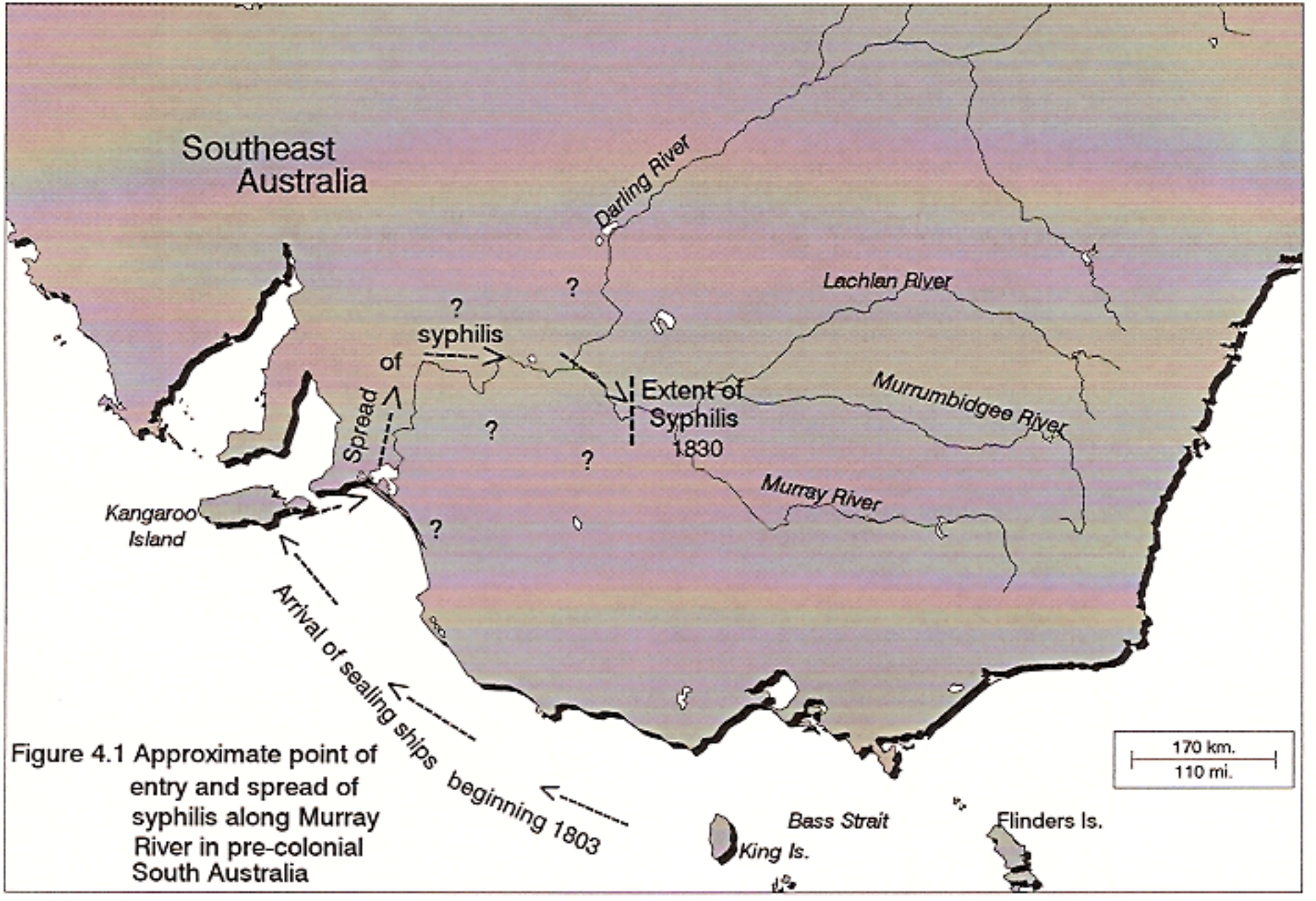


Figure 4.1 Approximate point of entry and spread of syphilis along Murray River in pre-colonial South Australia

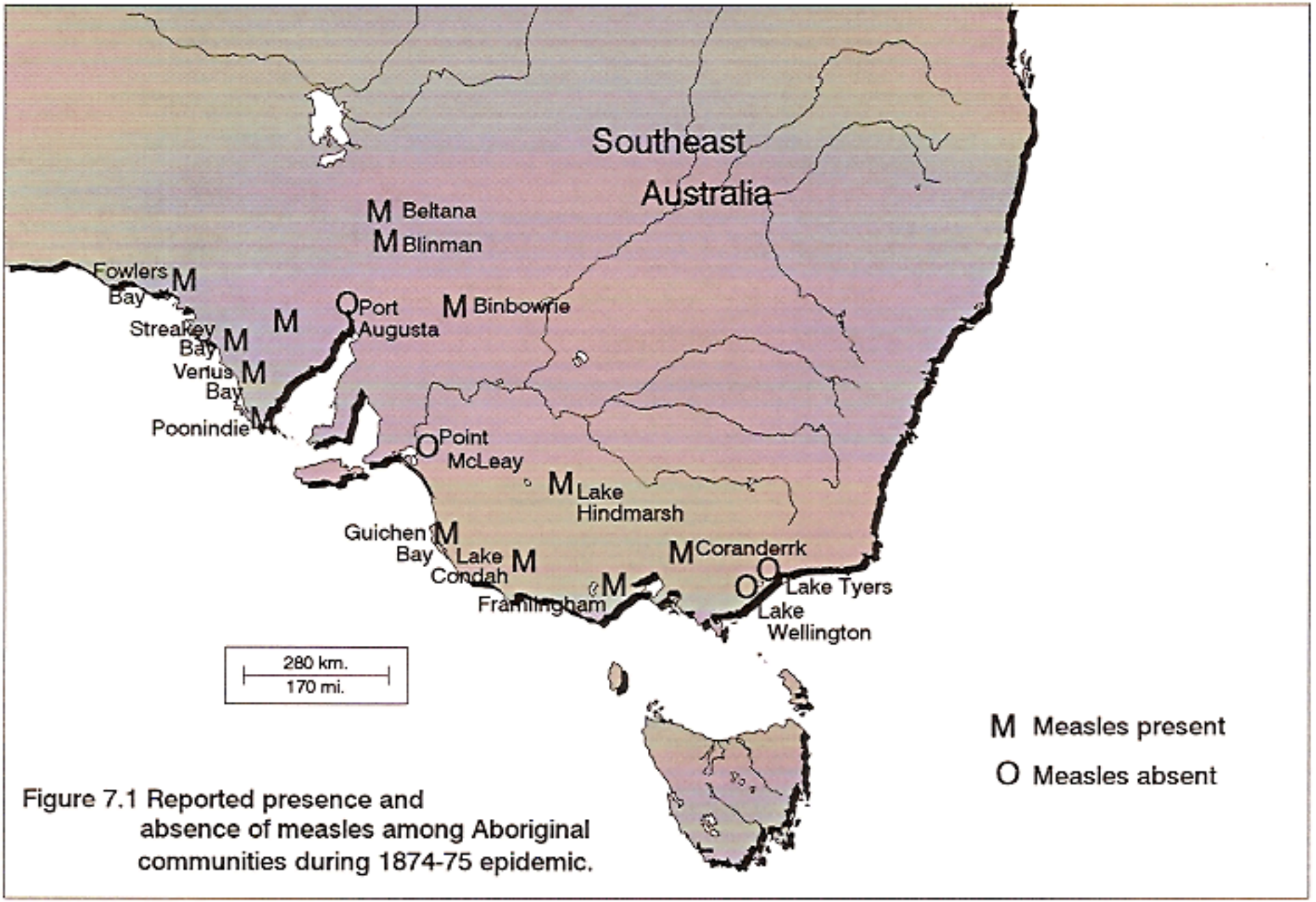


Figure 7.1 Reported presence and absence of measles among Aboriginal communities during 1874-75 epidemic.

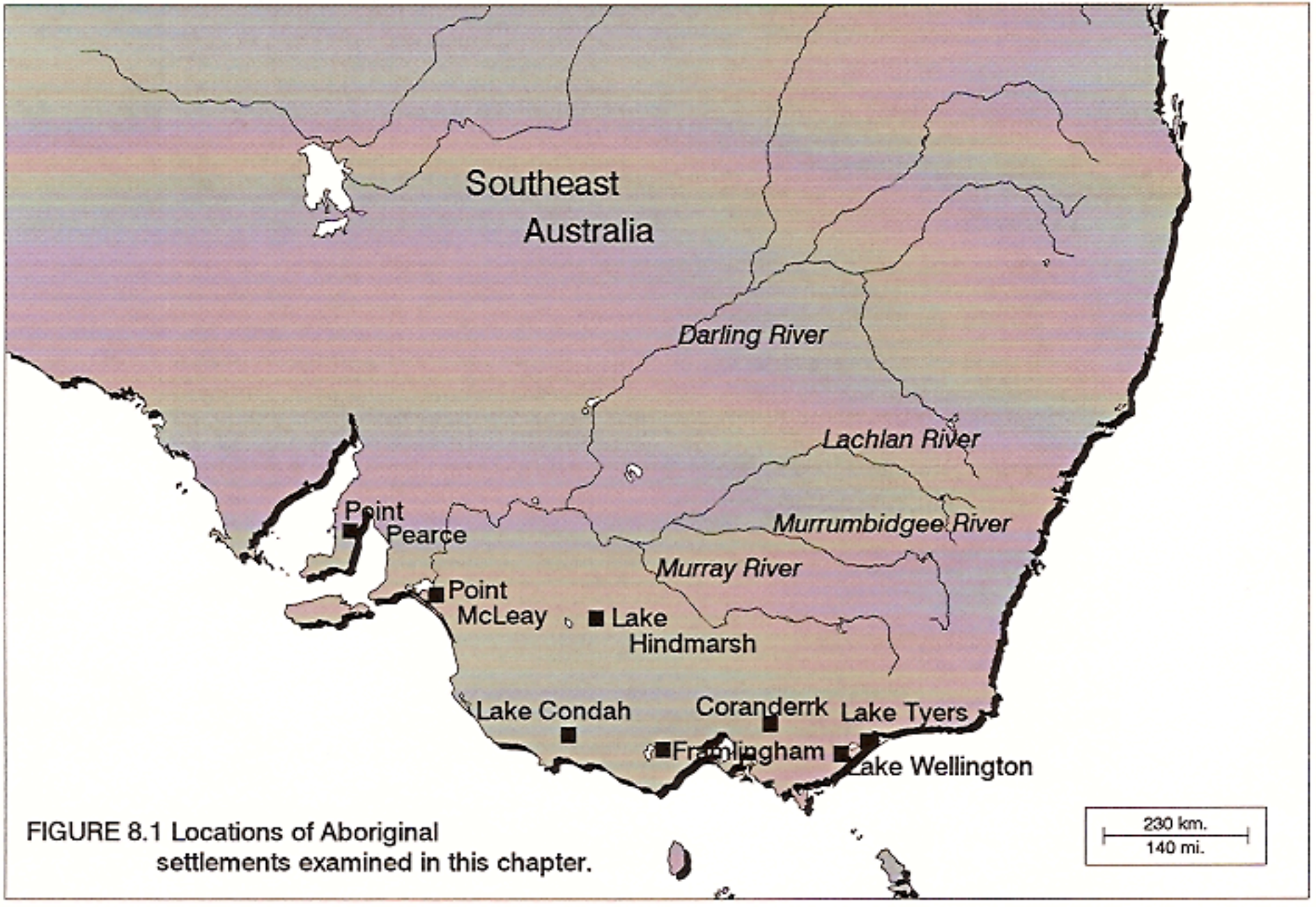


FIGURE 8.1 Locations of Aboriginal settlements examined in this chapter.

Plate 4.1 Adult male cranium (A911 434) from Riverland region of South Australia (Murray River) with frontonasal bone destruction due to treponemal infection.

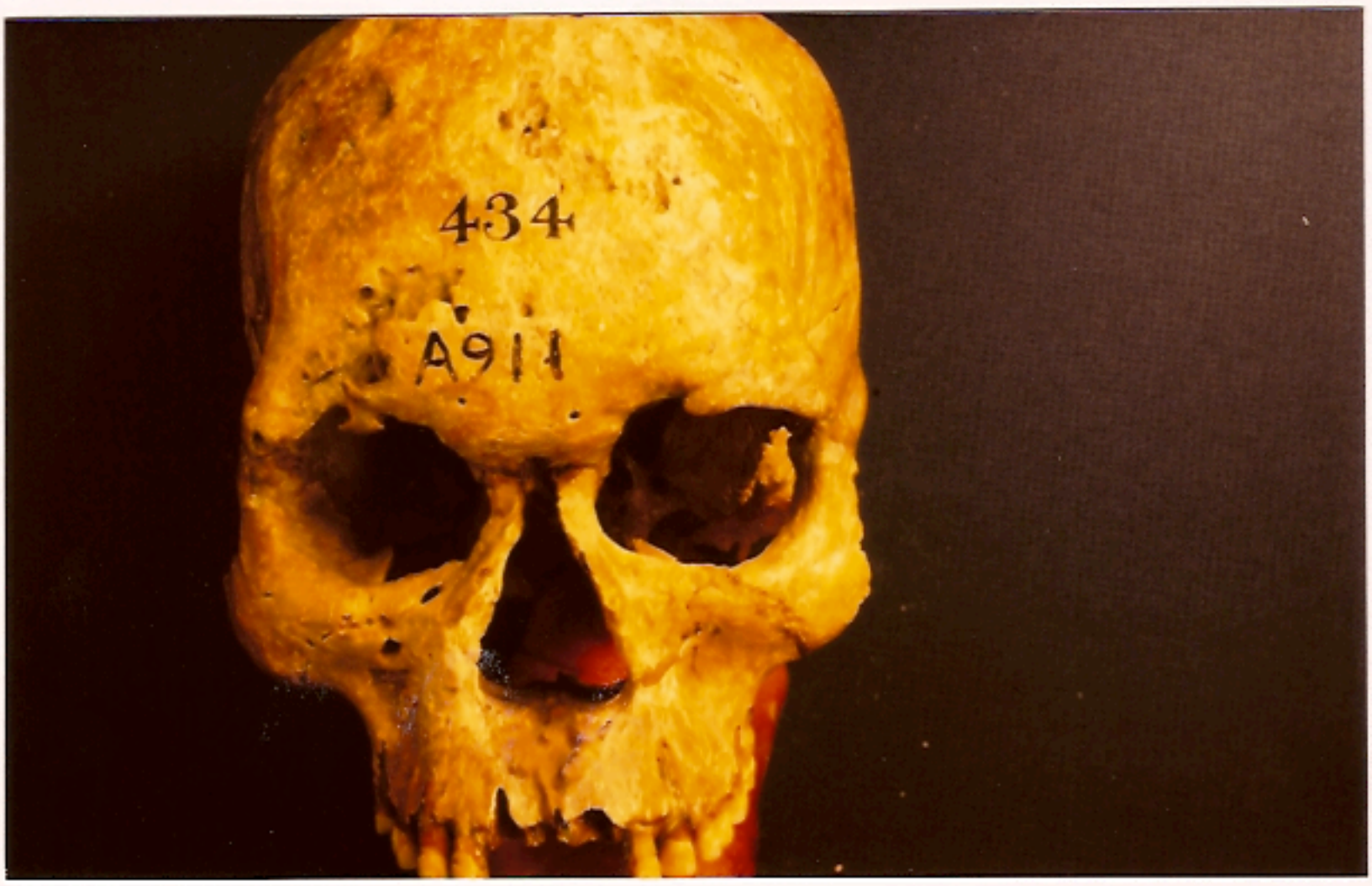


Plate 4.2 Adult male cranium (A911 434) from Riverland region of South Australia (Murray River), with destruction of nasal, frontal and right parietal bones due to treponemal infection.

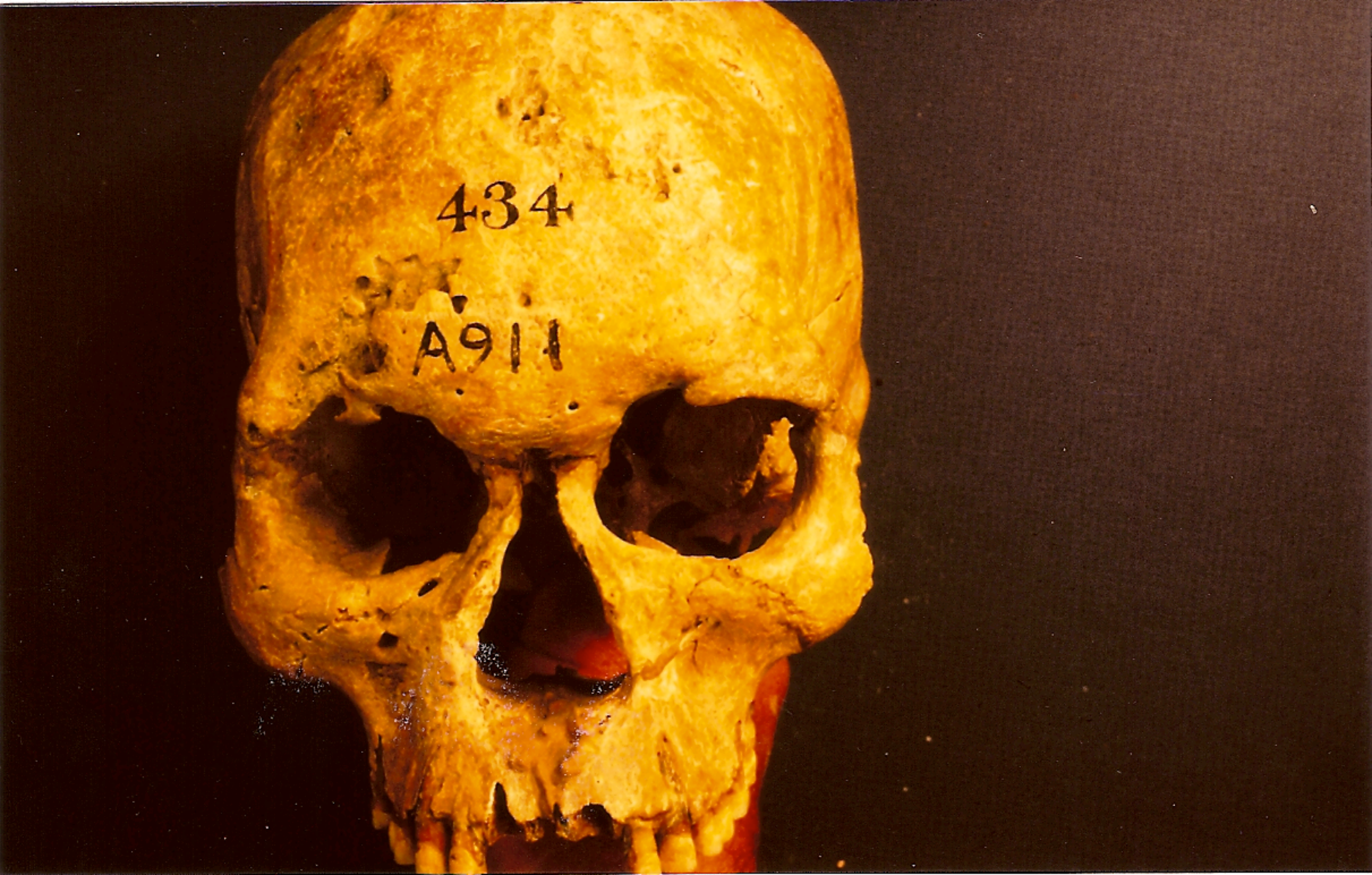

434
A911

Plate 4.3 Adult male cranium (A911 434) from Riverland region of South Australia (Murray River) with areas of destruction on right parietal and frontal bones due to treponemal infection.

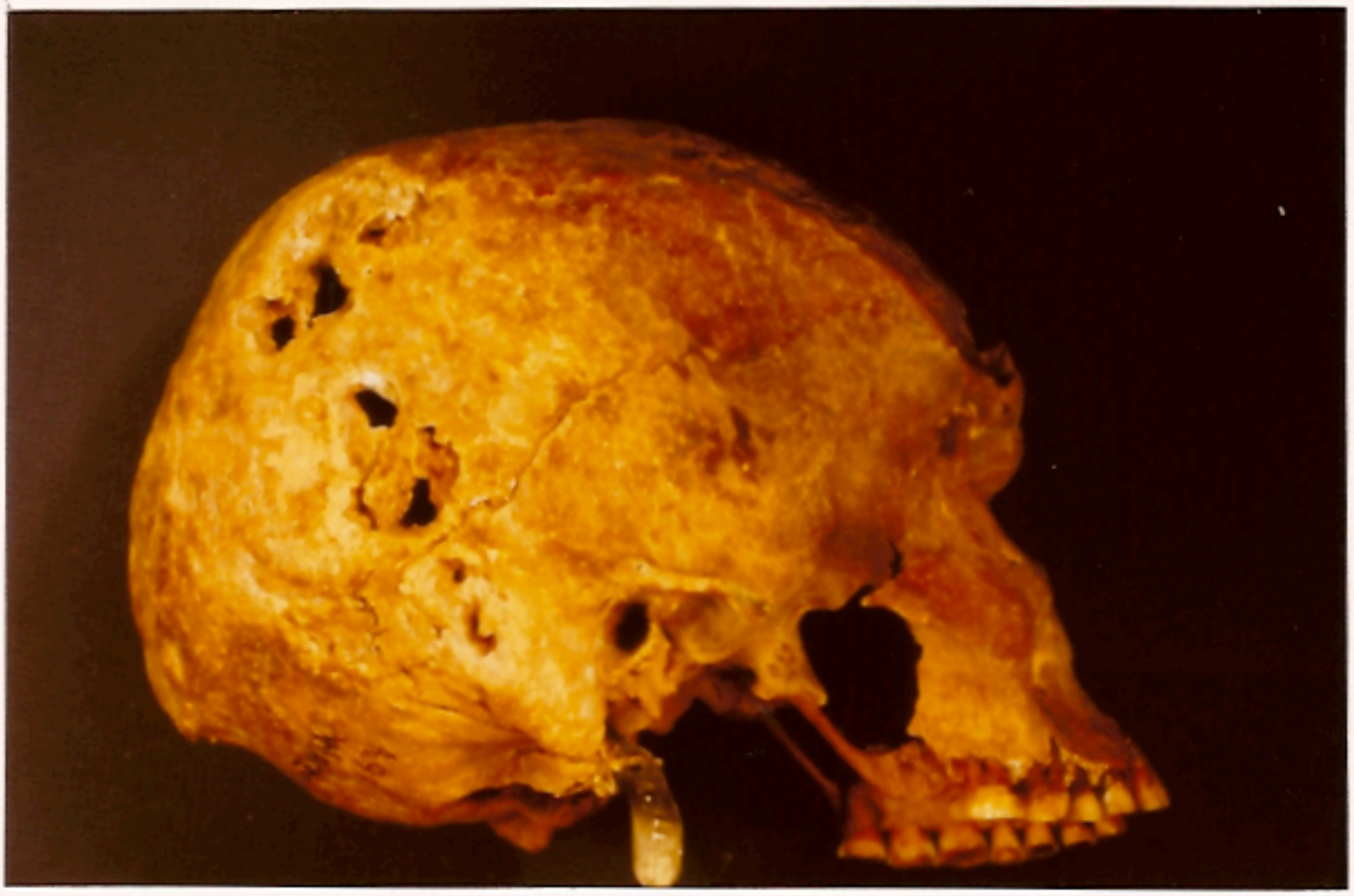
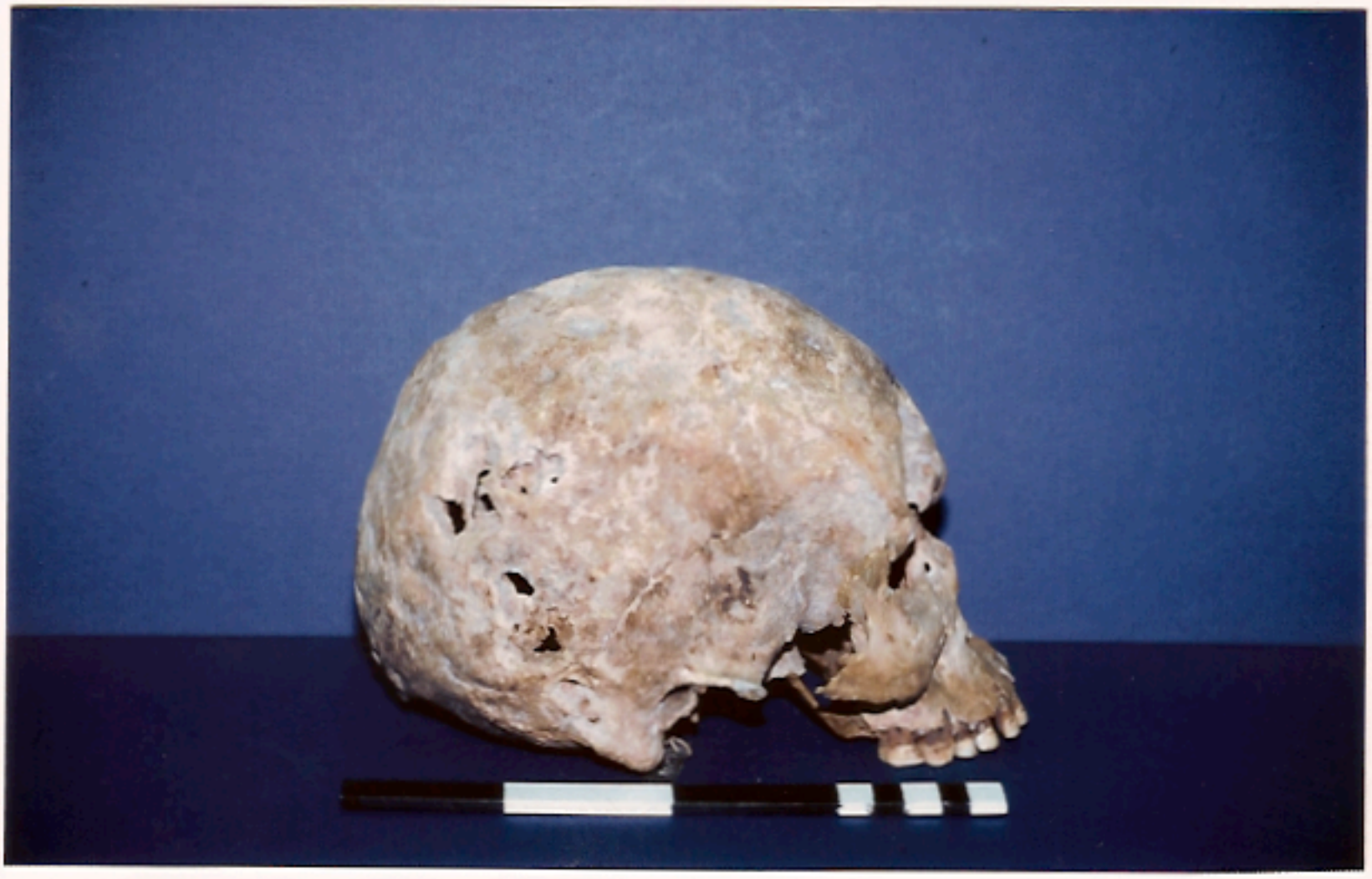


Plate 4.4 Adult male cranium (A351) from Lower Murray River region of South Australia, with frontonasal bone destruction and focal areas of destruction on right parietal bone, due to treponemal infection.



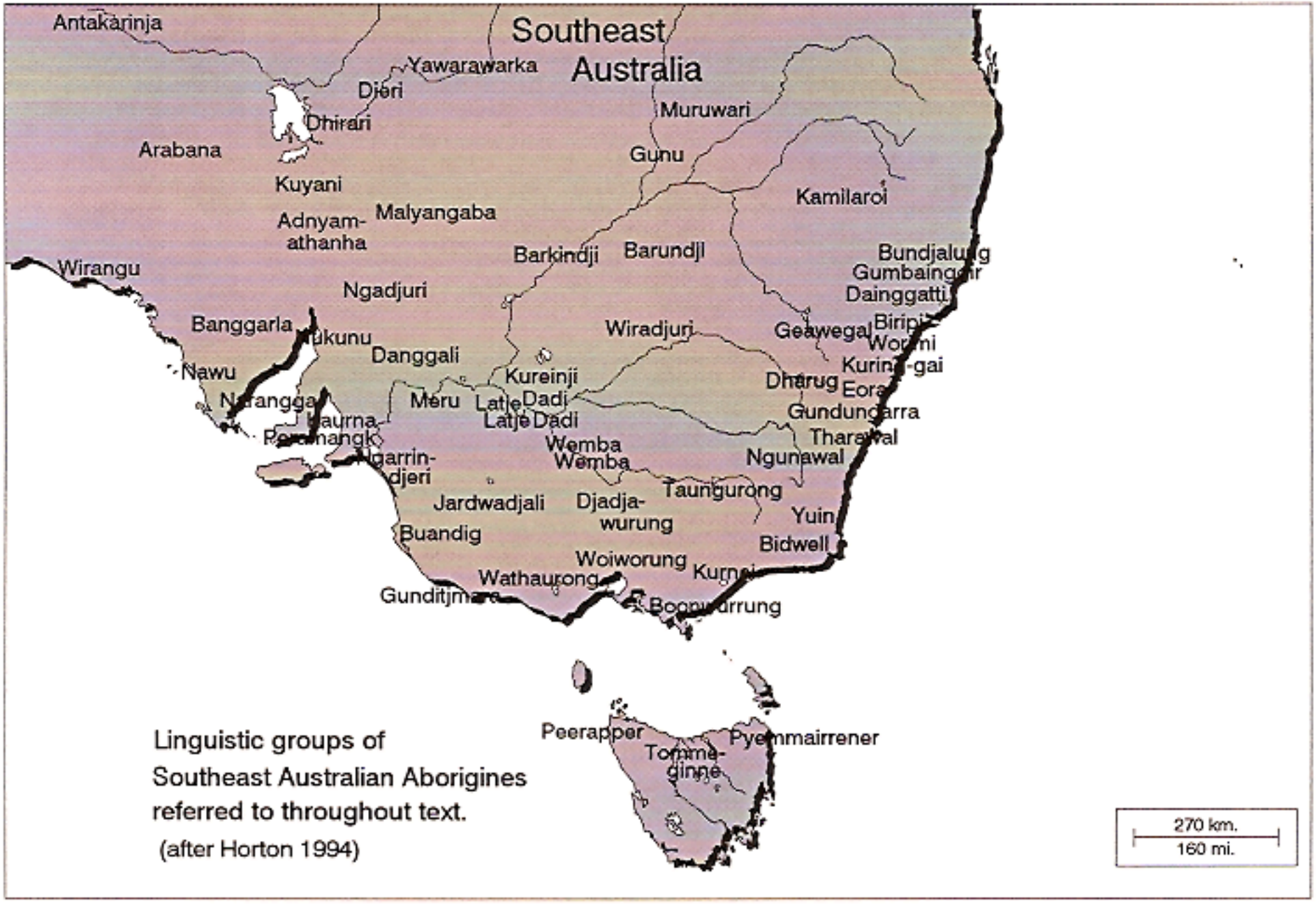

Southeast Australia
Antakarinja
Yawarawarka
Djeri
Dhirari
Arabana
Kuyani
Adnyam-athanha
Malyangaba
Muruwari
Gunu
Kamilaroi
Barkindji
Barundji
Wirangu
Ngadjuri
Banggarla
Wiradjuri
Danggali
Nawu
Kureinji
Meru
Latje
Dadi
Wemba
Ngunawal
Taungurong
Jardwadjali
Djadja-wurung
Buandig
Yuin
Bidwell
Woiworung
Wathaurong
Kurnai
Peerapper
Tommeginne
Linguistic groups of Southeast Australian Aborigines referred to throughout text.
(after Horton 1994)
270 km.
160 mi.

Plate 8.1 Lake Tyers Aboriginal settlement. A posed photograph of Aboriginal residents outside their cottages (La Trobe Picture Collection, State Library of Victoria).

Plate 8.2 Residents of Coranderrk Aboriginal settlement. The picture depicts a mixture of institutionalisation and traditional culture. European clothing is worn by two and a government issued blanket is worn by the elder of the two children. The woman carries a spear in one hand and collected leaves in the other. The elder child carries a steel axe (La Trobe Picture Collection, State Library of Victoria).

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Abbreviations

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AO	Archives Office
BPA	Central Board Appointed to watch over the Interests of the Aborigines in the Colony of Victoria
HRA	Historical Records of Australia
ML	Mitchell Library, Sydney
NLA	National Library of Australia, Canberra
NSW	New South Wales
NSWPP	New South Wales Parliamentary Papers
PRO	Public Records Office
SA	South Australia
SAPP	South Australian Parliamentary Papers
Vic	Victoria
VPP	Victorian Parliamentary Papers
WHO	World Health Organisation

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