

Invisible Invaders

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*Smallpox and Other Diseases
in Aboriginal Australia
1780–1880*

Judy Campbell



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Foreword

The influence of smallpox imported from Africa and Europe on the fate of the indigenous people of the Americas is now widely known, not least because of William McNeill's book *Plagues and Peoples*. Far less is known about smallpox amongst the Aboriginal Australians, and there is a difference of opinion about its origins and its effects. All agree that the Aboriginals were free of the infectious diseases of childhood common in Britain at the time: measles, chicken-pox, smallpox, rubella, diphtheria and pertussis. Some ascribe the early outbreaks of smallpox, the most lethal of these diseases, seen by the settlers in 1789 and 1829–30, to importations by the British; others believe that it may have come 'from the north'. There is little disagreement that later outbreaks, in the 1860s, arose from contacts of Aboriginals living in northern Australia with infected sailors coming from the islands to the north. There are also disagreements about the demographic effects of Aboriginal smallpox and its influence on the resistance of the Aboriginal tribes to extension of the settlement of British settlers into outback areas of New South Wales and Victoria.

This book sets out to assess the scanty historical information available in the light of modern knowledge about the epidemiology of smallpox. Unlike observations of more recent outbreaks in other parts of the world, there is no reliable photographic evidence of sick or pockmarked people. On the other hand, the early settlers and explorers were well aware from personal experience of the symptoms of smallpox and the traces of its presence left for many years among some of the survivors (that is, the characteristic pockmarks), so that their comments on such matters are reliable.

Using all the historical evidence available, reinforced by the researches of Campbell Macknight on the expeditions of trepang fishermen from the Indonesian islands to the north of Australia from the mid-eighteenth century onwards, the writer concludes that the four or five importations into Aboriginal Australia between the 1780s and 1860s came in with Macassan traders operating on the northern

coast of Australia. She dispels the myths that the outbreaks seen by the colonists in 1789 and 1829–30 were associated with the British settlement in New South Wales. The outbreaks among Aboriginal Australians, which moved slowly over great distances, contrast sharply with the importations into the European populations of the port cities of Australia between 1857 and 1903, which originated from ships coming to Australia from the northern hemisphere and remained localized in those cities.

The author also examines the probable demographic effect of smallpox and other introduced diseases, such as tuberculosis, and the way in which population loss due to introduced diseases weakened Aboriginal resistance to the seizure of their lands by European settlers, which occurred from the mid-nineteenth century onwards. Her observations emphasize the fact that infectious diseases, and especially the most lethal of them, smallpox, have played a major role in weakening the resistance of the indigenous inhabitants of the ‘isolated’ continents of the Americas and Australia and the isolated islands of the Pacific to the global European expansion that began in the fifteenth century.

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Preface

Smallpox and tuberculosis were unknown to the indigenous inhabitants of the isolated continents of America and Australia, and they were the most damaging of new infectious diseases to break out among Native Americans and Aboriginal Australians when their isolation ended. There is no doubt that both diseases arrived in North America with passengers on European ships. Smallpox travelled with English settlers and their African slaves on the short Atlantic crossing in the seventeenth century. In the late eighteenth and nineteenth centuries, many migrants leaving for North America and Australia were heavily infected with tuberculosis, because England was then at the height of an epidemic of the disease.

Although members of the First Fleet soon realized that ‘consumption’ had travelled to Australia with them, they were certain smallpox had not. The English knew the disease better than they had on their American voyages in the previous century, and by the time they came to Australia, they had some control over it. Most were immune after recovering from it in childhood or after being variolated. Accidents of infection at ports on the way in 1787 had been avoided, and smallpox had not occurred in any members of the First Fleet. After 1800 most European travellers arriving in Australia had been vaccinated. However, colonists and convicts from England were not the only voyagers to arrive in Australia between 1780 and 1870, and there were widespread outbreaks of smallpox among Aboriginals in those years.

Speculation about the origin of these outbreaks began when it appeared at Sydney in 1789, and continued in the nineteenth century, when explorers and settlers reported unmistakable traces of its recent presence. There was little evidence of the source of infection before the 1870s, when newcomers in the Northern Territory, who were familiar with smallpox, saw pockmarked Aboriginal people. The latter had contact with travellers from islands in the Indonesian archipelago, who regularly visited them. These travellers were Macassan fishermen from South Sulawesi and neighbouring

islands, who sometimes stayed as long as six months, and reliable observers attributed Aboriginal smallpox to contact with the travellers from the north. In the twentieth century, South Sulawesi was an important focus of infection in the archipelago, and the eradication of smallpox there was one of the more formidable tasks undertaken during the smallpox eradication campaign. It now seems certain that travellers from South Sulawesi introduced smallpox to indigenous populations in the region in the past, including populations in northern Australia in the eighteenth and nineteenth centuries.

Although outbreaks in the 1780s occurred over a wide area in eastern Australia, it was only in Sydney in 1789 that Europeans were present, and thus able to provide food and care for a few of the victims. Food was supplied to the sick in the settled districts in some of the later outbreaks, and colonial surgeons vaccinated Aboriginals in some districts. Nevertheless, the indigenous population declined catastrophically. They could not resist the invaders during the epidemics, and they were soon dispossessed. Smallpox was followed by tuberculosis among Aboriginals who had contact with Europeans in newly settled districts in south-eastern Australia. It was not possible to prevent or cure tuberculosis, and it was the main reason, apart from smallpox, why Aboriginals were thought to be a 'Dying Race', an image that persisted into the twentieth century. The presence of these two lethal diseases and others such as measles in the first century of European occupation had long-lasting consequences for the original inhabitants.

The activities of these invisible invaders are no longer as hypothetical as they once seemed. Records of Aboriginal smallpox, tuberculosis and measles were kept by officials who knew enough about the diseases to judge the severity of outbreaks they saw. Modern medical knowledge provides a framework for evaluating the records they left, and, in the case of smallpox, which is now unknown to Australians, data compiled by international teams who eradicated it is now generally available. However, there is so far no consensus of opinion in either immigrant or indigenous communities about its origin and impact in Aboriginal Australia.

The first chapter of the book outlines indigenous and introduced diseases observed in Aboriginal populations after 1788. The

second presents up-to-date information about the main killer, smallpox, and its history in North America. The third chapter presents theories about Aboriginal smallpox put forward in the last two hundred years, which have obscured its history. The next chapter is about smallpox in the Indonesian archipelago at the time of the Aboriginal epidemics, and is followed by chapters based on records of Aboriginal smallpox in eastern Australia in the 1780s, 1820s, early 1830s and 1860s. Tuberculosis and measles, which occurred in the victims of smallpox as well as in many others in those decades, are also included. Aboriginal smallpox occurred in Western Australia as well between 1860 and 1870, and is the subject of the final chapter. The threads are drawn together in the conclusion.

This book was written over a long period, with generous advice from virologist Frank Fenner of The John Curtin School of Medical Research, Australian National University, about records of smallpox and other diseases in Aboriginal populations. When he and international colleagues published *Smallpox and its Eradication* in 1988, a wealth of information about the epidemiology and world history of smallpox became available, and its impact on indigenous populations in other parts of the world suggests how it may have affected Aboriginal populations in different environments in Australia.

Following research in northern Australia by John Mulvaney, Jim Allen and Campbell Macknight in the second half of last century, our understanding of Aboriginal Australia is changing. I am greatly indebted to Macknight, whose publications about economic and social ties between Aboriginal clans and visitors from Macassar suggested new perspectives in Australian history. I am also indebted to Peter Moodie, whose publications on Aboriginal health provided many answers to my questions. Manning and Dymphna Clark, Don Baker, Jill Waterhouse, Geraldine Triffitt, Tamsin Donaldson, Jane Simpson, Luise Hercus, Peter Stanley, Graham Henderson and Peter Dowling read parts of the manuscript and gave information on particular questions. Help from archivists and librarians at the Mitchell Library, the Battye Library, South Australian Archives, the Hancock Library, the National Library of Australia and the British Library, London was invaluable. Kay Walsh at the Australian Defence Force Academy found essential sources and bibliographical details. Karen and Paul Walter found South Australian material. Cartographer

Kevin Cowan provided maps. Members of my family helped from the start. John Trinca and Barbara Cook found sources, Dave Campbell found sites, Barb and David checked drafts.

I particularly wish to thank the institutions that made the pictorial collection possible.

Abbreviations

<i>ADB</i>	<i>Australian Dictionary of Biography</i>
Col. Sec.	Secretary of the Colonial Office, London
<i>HRA</i>	<i>Historical Records of Australia</i>
<i>HRNSW</i>	<i>Historical Records of New South Wales</i>
NSWA	New South Wales Archives
SAA	South Australian Archives
<i>TPRSSA</i>	<i>Transactions and Proceedings of the Royal Society of South Australia</i>
<i>V & P</i>	<i>Votes and Proceedings</i>
WAA	Western Australian Archives

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Aboriginal Australians and Old World Diseases

The brief impressions of the first British observers who saw Aboriginal Australians on the east coast of Australia in the late eighteenth century suggests they were to be envied for their physical well-being. At Botany Bay and Endeavour River, on Cook's *Endeavour* in 1770, Joseph Banks wrote in his journal that

On their bodies we observed very few marks of cutaneous disorders as scurf, scars of sores, etc. Their spare thin bodies indicate a temperance in eating, the consequence either of necessity or inclination, equally productive of health particularly in this respect.

In his first months at Sydney Cove in 1788, the enthusiastic George Worgan, a naval surgeon on Phillip's *Sirius*, similarly found that the inhabitants 'seemingly enjoy uninterrupted Health, and live to a great Age'. These Englishmen knew the worst diseases that surrounded life in Britain, and would never before have seen whole populations without pockmarks. The appearance of local people they saw was a great contrast to the unhealthy appearance of scurvy-ridden arrivals in 1788.¹

Banks' and Worgan's remarks about the health of Aboriginal Australians are like other travellers' tales about peoples who were meeting visitors from the outside world for the first time. The

questionable accuracy of these abundant reports led historian William McNeill to investigate them. He found that medically competent outsiders, such as Banks and Worgan, were among those who thought the hunter-gatherers they had seen did indeed enjoy a high level of general health. The travellers' first fleeting impressions are surprisingly close to the opinions of modern epidemiologists. Some isolated pre-agricultural nomad communities in remote parts of the world who survived into the twentieth century are unusually free of disease. Epidemiologist Francis Black, who observed the limited range of their infectious diseases, reported: 'In large measure modern advances can do no more than return to us the state of health mankind enjoyed 10 000 years ago'.²

At the same time, none of the world's indigenous populations was entirely without disease. The relatively few important and widespread infectious diseases that occurred in pre-contact Australia were caused by bacteria, protozoa and viruses that were established in the region, and Aboriginal people lived with them for thousands of years. Some of these diseases, which varied in different climates, were acute infections, such as those caused by arboviruses. Dengue, for example, is spread by mosquitoes in the tropics. Other diseases were ancient and chronic. These long-established diseases developed slowly and usually affected the elderly, but did not kill them. They included trachoma, non-venereal treponemal infections and hepatitis B. These and other rare or less important infections that harassed isolated people in the pre-European past were unfamiliar to Europeans.³

The most widely known indigenous disease today is the chlamydial eye disease trachoma. This is an eyelid infection that impairs vision, can become chronic, and sometimes causes blindness. Trachoma is common in hot, dry and dusty conditions where there are swarms of flies, which are persistent in temperate mainland districts as well as in the tropics. The lives of bush people, with inevitable scratches and injuries, dust, dirt and smoke, allow early infections to last a lifetime.⁴ The ancient presence of trachoma is best illustrated by linguistic evidence. Isobel White points out that words for old and blind are used interchangeably by the Pitjanjatjara in the Western Desert. In a rare pre-colonial glimpse of an indigenous disease, its ecology and its consequences, English explorer William

Dampier described his experiences on the north-west coast in 1688 and 1699. Plagued by the terrible flies and troubled by the sun, he saw Aboriginals who 'had such bad Eyes, that they could not see us till we came close to them'. In the nineteenth and early twentieth centuries, settlers saw Aboriginal people with trachoma, and some suffered from it themselves (they called it 'sandy blight'). Aboriginal and European sufferers lost the sight of one or both eyes.⁵

Unlike venereal syphilis, the sexually transmitted treponemal disease of European adults, the treponematoses in Aboriginal Australia are caused by ancient bacteria prevalent in the region, and the history of diseases they cause, such as yaws, is obscure. As McNeill and others suggested, yaws is of more ancient origin than syphilis. It has long been endemic in hot, damp rural districts in Indonesia and northern Australia as well as in Africa. Yaws was not well known at the end of the nineteenth century, and the late destructive changes that follow several decades after childhood infection were not recognized until the 1920s. Non-venereal endemic syphilis, also known as treponarid, is caused by a closely related spirochete, and was virtually unknown until the 1930s, when pathological changes in Australian Aboriginal bones were recorded, and comparable evidence of it was observed in the Euphrates River delta in 1942. Its presence in desert lands in the Middle East, tropical Africa and South Africa was then recognized.

The presence of treponematoses in the remote past is suggested by skeletal material, and in Australia it can be accepted that any treponemal changes in bones are due to yaws if they come from the north, or non-venereal endemic syphilis (treponarid) if they come from the centre or south of the continent. These diseases are common in nomad societies, although since the 1940s the use of antibiotics has affected their formerly widespread prevalence. However, laboratory analysis of sera demonstrates the presence of antibodies, and thereby the past presence of disease. There is serological evidence of yaws, *Treponema pallidum pertenue*, on the humid north coast of Arnhem Land, and of non-venereal endemic syphilis, *T. pallidum endemicum*, in the dry, hot interior of the Northern Territory. The most recent evidence of yaws in the northern half of the Northern Territory showed infection continuing, whereas in the southern half, evidence suggests endemic syphilis is disappearing.⁶

The symptoms of these closely related diseases develop in the primary, secondary and tertiary stages of infection during the sufferer's lifetime. In the 1930s South Australian physiologist and physical anthropologist C. J. Hackett described them after a period in Central Australia, studying physical anthropology among Aborigines. He investigated 'boomerang leg', the tertiary stage of endemic syphilis, which is characterized by the bowing of the bones of the lower leg. Hackett showed it was due to a non-venereal treponemal infection, which they called *irkintja*. It was then common in the Northern Territory and Central Australia, and skeletal evidence of it had been observed in south-eastern Australia. More recently, medical historian Peter Moodie said 'boomerang leg' is far from being merely a medical curiosity. During a survey in 1968 in the Northern Territory, it was found to be quite common: 20 per cent of people aged forty-five or more were affected to some extent, and it occurred in a smaller number of younger people. In some cases, it caused serious deformity. Moodie thinks treponemal infection, nutrition, and the function of the lower leg all play a part in causing 'boomerang leg'.⁷

The indigenous experience in Australia is not unique. Yaws was introduced into the Caribbean islands by slaves from French West Africa. Among their descendants in humid tropical Jamaica, where it was endemic in rural districts early in the twentieth century, people in the secondary stage of the disease had painful and tender swellings in their bones, and the bowing of bones in their arms and legs followed. In 1996 medical microbiologist Justin Radolf described destructive changes in long bones of the legs and forearms of people with tertiary yaws.⁸

Hackett became an acknowledged world expert on treponemal diseases, and besides working on programmes to control yaws in many countries, he published some ninety scientific papers on yaws and related questions. In his opinion, Australian Aborigines had suffered treponemal infections for thousands of years. Worldwide, climatic conditions determine the presence of one or the other, with yaws in the humid warm equatorial belt, and non-venereal endemic syphilis in arid, warm areas north and south of it. In favourable conditions, they spread from person to person, and can affect most of a

population by adulthood. Yaws commonly enters the body through an initial lesion on the lower leg; the primary lesion looks like a raspberry, and the disease is known by the name of *Framboesia tropica* (tropical raspberry).

Endemic syphilis enters the body in the mucous membranes of the eyes and mouth. Infection is transmitted easily by contact between children who play and sleep together. Secondary skin lesions, which are infectious, are very numerous in yaws, and are exacerbated by the abrasions and injuries of bush life. The early lesions of endemic syphilis are scanty and less recognizable; then secondary lesions occur in skin, muscles and bone. They are transient in both infections; however, painful and tender joints and bones cause problems in the secondary stage, before being latent for up to fifteen years. In the tertiary stage of both infections, there are sometimes changes in bones, which may be permanently destructive, producing disfiguring bony deformities, especially when lesions of the face and nostrils occur. 'Boomerang leg' is one of the late changes that follow long after childhood infection. Another treponemal disease, *pinta* (*T. carateum*), affects the skin only, and is found only in Central and northern South America. Australian treponematoses, and others discussed by Hackett, and in 1996 by Radolf, usually all protect against venereal syphilis in adults. Recently, a higher incidence of syphilis followed effective campaigns to eradicate yaws, endemic syphilis and *pinta*.⁹

Observers who left written descriptions of these infections in Australia in the nineteenth century were new to indigenous diseases, and often attributed symptoms they saw to another closely related treponematoses, venereal syphilis, *T. pallidum pallidum*. However, in a remote part of Tasmania, French naval explorer Nicolas Baudin saw an indigenous disease in the inhabitants of D'Entrecasteaux Channel in 1802, and remarked on the absence of European diseases such as syphilis among Aboriginal Tasmanians. In his journal, Baudin said Aboriginals: 'seem to be subject to a type of yaws, for several had ulcerated legs. We discern no trace of smallpox on their faces or bodies, and they are possibly fortunate enough as well not to know syphilis.' Hackett, who described possible treponemal changes in a Tasmanian skull, thought the disease Baudin saw was not yaws,

which occurs in hot humid climates, but may have been endemic syphilis (treponarid).¹⁰ Historian Lyndal Ryan found venereal diseases, including syphilis, occurred among stock-keepers, sealers and settlers in Tasmania, and thought the low level of venereal disease in Aboriginal people there was the most puzzling feature of the Tasmanian evidence.¹¹ An indigenous treponemal infection, perhaps endemic syphilis, may have protected the Tasmanians from venereal syphilis, just as before penicillin was discovered yaws protected New Guineans from it.

The presence of another indigenous disease was not discovered until 1967, when B. S. Blumberg was investigating thousands of sera from diverse populations, including Australian Aboriginals, the latter being material sent to him by Dr R. Kirk. His work unexpectedly revealed a novel substance, initially called 'Australian antigen', which turned out to be the surface protein of a previously unknown virus, hepatitis B, of which there was a high incidence in Aboriginal populations. It has now been shown to occur worldwide, especially in less developed countries; there are estimated to be 350 000 000 cases in China. The virus is perpetuated through its transmission from mother to child during the period of intimate contact between them in early life. When it is transmitted at birth, hepatitis B is usually a lifelong infection, and it is sometimes responsible for cirrhosis or cancer of the liver in the later life of its human host. In adult life, it can be transmitted by the exchange of bodily fluids, such as blood. As it can survive in small groups, its ancient presence among Australian hunter-gatherers is likely. In the nineteenth century, a few Europeans reported that 'liver disease' caused sickness and death among Aboriginals. It was not clearly identified at the time, and may have been hepatitis B, which is now more common among Aboriginals than Europeans.¹²

Like his comments about Aboriginal health in 1788, Worgan's remark—that he saw people who had lived to a great age—anticipated the opinions of modern scholars about their life-span. In his study of the nomads, historian Geoffrey Blainey discriminated between the precarious expectation of life at birth in a society of hunter-gatherers, and the expectation of life some years later, when survivors of a nomad childhood might expect a life-span as long as

that of Europeans. Virologist Frank Fenner considers that the relatively long life-span of human beings, after the risks of birth and infancy were surmounted, was a significant feature of nomad life, together with relative freedom from infectious diseases in small isolated bands of hunter-gatherers. The chronic diseases prevalent among them probably interfered less with their potentially long lives than violence or natural disasters did.¹³ Despite those hazards and the new and worse hazards of European settlement, nineteenth-century settlers who got to know local Aboriginals echoed Worgan's opinion, and described some whose longevity seemed exceptional, including some who had survived smallpox.¹⁴

The isolation of geographically remote regions, islands and whole continents, perpetuated the early balance between humans and microparasite that allowed both to survive, and for many centuries the inhabitants of the Americas and Australia were protected from the destruction new diseases caused among humans on the Eurasian-African continents.¹⁵ Gradual changes to modern disease patterns probably evolved on the Eurasian landmass about 10 000–12 000 years ago, when humans tamed domestic animals and cultivated plants, then set up larger settlements. Bacteria and viruses that occurred in their domestic animals evolved to infect humans, and many of these were readily transmitted from person to person by droplet infection in more densely settled regions.

Some of the world's worst diseases became endemic in the growing populations of Eurasia in the last three or four millennia. Some were acute and lethal infectious diseases, such as smallpox, bubonic plague and measles, which killed suddenly. Tuberculosis was insidious, causing early deaths in some, and chronic illness later in life in others. Venereal infections, such as syphilis, were chronic in adults, and gonorrhoea made women sterile.¹⁶ Some were ancient diseases, whose origin was obscure until recently. For instance, smallpox was known for at least 3000 years before it was eradicated in 1977. Yet the international team who eradicated it did not know how, when or where smallpox virus had originated, and there was no answers until recently. Then, in 1998, with data from American physiologist Jared Diamond, Frank Fenner, a senior member of the team, suggested smallpox virus probably evolved from cowpox

virus, a disease of rodents in Asia and Europe, which is readily transmitted to cows. This may have begun in the Fertile Crescent about 6000 BC.¹⁷

The extraordinary twentieth-century discovery of the seemingly pockmarked mummies of several Egyptian royals, preserved in the second millennium BC, confirmed the antiquity of smallpox in the Nile Valley and the Fertile Crescent. These remarkable events, and an account of the Pharaoh Rameses V, who died, apparently of smallpox, in 1157 BC, have been expertly reviewed by Fenner, Hopkins and Diamond.¹⁸ Smallpox was periodically rife in the Ancient World, and it is not surprising to find that early in the first millennium, between AD 165 and AD 180, it reached Rome as the Plague of Antoninus, killing millions of Romans, according to Diamond. Moving on to Europe's late medieval plague, the Black Death of 1346–1352, which he said killed a quarter of Europe's people, Diamond may have underestimated the toll of that frequently lethal disease. Like smallpox, it came from rodents.¹⁹

In the wake of Columbus, over a hundred years later, there were great changes in the incidence of the world's worst diseases, when they attacked previously unexposed populations in the New World. Early in the sixteenth century, when they caught diseases, in particular smallpox, which was prevalent among conquistadors and their African slaves, Native Americans suffered what was possibly the greatest biological catastrophe experienced by humans.²⁰ For fifty years after 1492, the Spaniards had a firm foothold on both American continents, which were already lost to Native American survivors of massive smallpox die-offs.

The isolation of Australia from Eurasia and its plagues was never as complete as the American isolation had been, and it ended less abruptly. However, the rising seas separated the continent from New Guinea and offshore islands when Torres Strait was 'drowned', perhaps 6500 years before the present. So for several thousand years before smallpox was endemic in Eurasia or present in the offshore islands, the region's sailors were unlikely to voyage south, leaving the original Australians isolated, while the seas kept travellers, migrants and invaders away. Accidental contacts with unexpected seafarers, local islanders, and eventually with the first European explorers, were inconsequential in the history of infectious diseases.

The balance between Aboriginal and microparasites was almost certainly undisturbed by contact with other humans, and any alien diseases they carried, until the eighteenth century, when contact with the wider world had far-reaching consequences. While they investigated fishing-grounds on the north coast of Australia in the 1720s, trepang collectors from the Sulawesi port of Macassar became interested visitors, and by mid-century more praus from Sulawesi came on the monsoons more often. Aboriginal clans on the coast shared the islanders' life while they fished for months each season.²¹

Then the British colony on the east coast made a small start in January 1788, when Governor Phillip arrived with over 1400 people. Once again the first serious infectious disease noticed in a population previously unexposed to the diseases of Eurasia was smallpox, which appeared in the late eighteenth century. An old and familiar enemy of the British at Sydney Cove, the virus had been established in the denser populations in south-east Asia and its offshore islands at that time. It is possible that it was not entirely unknown to all the people of the northern coastal fringe of Australia when Macassan trepangers began to visit, but it seems that there were no outbreaks of any consequence until the 1780s. The British first saw Aboriginals suffering from smallpox at Port Jackson in April 1789. None of those who recorded their impressions of sick and dying Aboriginals had any decisive evidence of its past presence among them, and at first there was speculation and uncertainty among Europeans, because Aboriginals and their language were still unknown. However, it looked as though smallpox was a new disease. Returning to the colony in the *Sirius* in May 1789 after a voyage to the Cape, Admiral Hunter expressed surprise 'at not having seen a single native on the shore, or a canoe as we came up in the ship'. When he was told why, the 50-year-old Hunter concluded, after a lifetime's experience of the disease, that: 'As we had never yet seen any of these people who have been in the smallest degree marked with the smallpox, we had reason to suppose they have never before now been affected by it, and consequently are strangers to any method of treating it'.²² Banks and Worgan would have agreed.

The new disease differed from the usual chronic ailments of isolated nomad societies in every way. It was the most lethal and the most frightening of diseases known to humankind. The onset of

sickness was sudden and unpredictable. Unfamiliar symptoms were acute and agonizing: victims were feverish, with a headache and backache, while a searing pustular rash developed. The virus could kill sufferers quickly, sometimes in only a day or two. It also inflicted immediate and permanent damage on survivors, who nearly all found they were scarred, at least on the face, and some found they were blind as well. These painfully disturbing characteristics caused some Native American sufferers to kill themselves. Panic and flight were normal reactions among all those seeing what happened for the first time. Refugees and messengers who subsequently sickened spread infection. In different societies people feared, hated, placated, and even worshipped supernatural powers said to cause the disease. The Australians associated smallpox, more than 'natural' diseases they already knew, with the malevolent powers of their enemies.²³ The resulting loss of morale, as well as the loss of men, women and children in depleted bands of survivors, made it easier for colonists to occupy the seemingly sparsely populated country, just as the Spanish were able to occupy Mexico so easily. Smallpox is said to have killed over 70 or 80 per cent of previously unexposed populations in some parts of North America.

Smallpox was the most recognizable of diseases. There were typical mis-diagnoses, and there are considerable difficulties about identifying other diseases in historical descriptions. But worldwide records of smallpox are rare, like the disease itself. When it was a new infection, onlookers and survivors all described unprecedented experiences rarely repeated in a lifetime. People who saw the active disease did not fail to convey the shock and horror of its many deaths in their accounts. Facial scarring with permanent pockmarks, a characteristic that assisted confirmation of the disease, also ensured evidence of its former prevalence, not only soon after outbreaks, but for as long as survivors lived. Records of smallpox in Australia are no exception. No other disease present in Australia after 1788 can be traced with equal certainty, and evidence of outbreaks accumulated until the early twentieth century. European newcomers, who knew the disease well, were surprised and disturbed when they saw active smallpox, and often noticed Aboriginals who were pockmarked and blind. Later, Aboriginals themselves vividly

described the many deaths of victims. Their recollections were immediately confirmed by visible scars that showed they had survived the outbreaks they described. Australian evidence is more fragmentary than North American evidence, because there were fewer outbreaks and many fewer observers, but it is similar and more recent than most American evidence.

The Australian evidence of Aboriginal smallpox in 1789 is easily found in printed sources, and there is much evidence of later outbreaks in printed official sources and colonial memorabilia. However, the best evidence of later outbreaks is the written evidence of the people on the spot, most of which has not been seen since it was written in the 1830s and 1860s, and which has been preserved in archives in Sydney, Adelaide and Perth. Using dated reports of active disease written by contemporaries who saw outbreaks, and others who later talked to survivors, and estimated the ages of scarred and unscarred people, we can estimate when and where epidemics occurred.²⁴

Apart from periodic localized introductions into Australian ports, summarized by Cumpston, smallpox was prevalent during four periods of Australian history. The disease, which was introduced to mainland indigenous populations from outside sources on more than three occasions, spread extensively in them at least three times. In eastern Australia, active smallpox was seen by colonists in 1789 at Port Jackson, Botany Bay, Broken Bay and on the Hawkesbury; scarred elderly Aboriginals, who were probably infected about the same time, were seen later in the settled districts of New South Wales by several regimental surgeons. They were also observed in Queensland, Victoria and South Australia. Active smallpox was again described in eastern Australia between 1828 and 1832. Its scarred survivors, who were seen a little earlier on the remote north coast, were recognized afterwards throughout the newly settled districts in eastern Australia by well-informed observers, and during colonists' expeditions inland.

Early settlers in Queensland, such as Tom Petrie, left records of pockmarked Aboriginal people they had met in the 1860s, 1870s and 1880s, and active smallpox was also observed in western South Australia in 1867. After 1870, when colonists arrived in the Northern

Territory, scarred survivors of earlier outbreaks were frequently noticed in Arnhem Land and along the overland telegraph line; for instance, by German migrant Paul Foelsche. Active smallpox was also evident each year from 1866 to 1870 in newly settled northern districts in Western Australia by Resident R. J. Sholl and colleagues, and scarred survivors were obvious. The limits of literary evidence of smallpox are the limits of European exploration and settlement when smallpox was prevalent, and while its scarred survivors lived. Written records do not of course reflect the entire geographical incidence of smallpox, or the time it took to spread over vast distances. Finally, the mild form of smallpox, *variola minor*, was introduced in Sydney from the United States in 1913, and spread slowly in the European population of New South Wales between 1913 and 1917, causing over 2000 cases of disease. There is no evidence of smallpox in Aboriginal Tasmania.

Other kinds of evidence might add to present knowledge of its impact. In South Australia early in the twentieth century, Sir Edward Stirling thought archaeological evidence might be relevant, and in 1962 New Zealand scholar C. W. Dixon mentioned inconclusive early reports of burial sites thought to be those of smallpox victims. Historical records and local traditions, which still exist, suggest that most of the burial grounds have not been found.²⁵ Unlike treponemal infections, smallpox left no distinctive skeletal evidence. There might also be evidence of smallpox in nineteenth-century collections of portrait photographs of Aboriginal people. In the 1860s Governor Hunter's nephew, John Hunter Kerr, had the negative of a photograph of a Loddon Aboriginal whose face was covered with pockmarks.²⁶ European portrait painters were often kind to their subjects, but sometimes painted the pockmarks so often seen in earlier centuries. French painter Petit, at Sydney in 1802, may have painted some pockmarked Aboriginal survivors.

Written records include unmistakable descriptions of smallpox in Aboriginal Australia, but it would be about a hundred years before Europeans began to piece together scattered evidence of outbreaks that coincided with their own occupation of the country. In 1789 the newcomers could only guess what might have happened on the other side of the Blue Mountains. After forty or fifty years of

exploration and settlement, people who saw pockmarked elderly Aboriginals a long way from Sydney realized how extensive that 1780s epidemic had been. Others, who had met much younger pockmarked people in newly explored, still sparsely settled districts, realized another epidemic had occurred more recently. In the 1860s smallpox apparently did not occur in New South Wales or Victoria; but in the 1860s, 1870s and 1880s, reliable observers reported another epidemic. In eastern Australia in Queensland, the Northern Territory and western South Australia in the 1860s, these were outbreaks at about the same time as the one that was reported in Western Australia. The later occurrences were not included in Noel Butlin's estimates of the impact of Aboriginal smallpox in eastern Australia, or in estimates using his work.²⁷

During the first century of settlement, familiarity with smallpox and its scars dwindled. Jenner's discovery of vaccination in 1796 had made it less common in Britain, the long voyage, isolated colonists, and ships arriving at Sydney were usually supervised in order to keep smallpox out of the colony. Native-born European Australians had never seen smallpox, and when it appeared among Aboriginals in the late 1820s and early 1830s, younger settlers were less likely to recognize it than those who had been in Sydney in 1789. In 1831, when the havoc it was causing near Bathurst was reported in the press, the *Sydney Gazette* said it might be 'peculiar to the native blacks'.²⁸

By comparison with the reactions of Phillip and others at Sydney Cove in 1789, the response of prominent settlers to the presence of smallpox among Aboriginals in the 1820s and 1830s was low key. It was not seen at Sydney, and Charles Sturt did not identify it on his first expedition in 1829, although he later realized that the disease he had seen was smallpox. In 1836, on his only inland journey in Australia, 23-year-old Charles Darwin visited Bathurst, where he saw no more than 50 Aboriginals, and believed they were rapidly decreasing in numbers, and that European diseases, 'even the milder ones', such as measles, were destructive. Darwin stayed on a Wallerawang farm, where smallpox had killed, scarred and blinded Aboriginals a few years earlier, but did not mention it in his account of the visit.²⁹ There was often disturbing news of violence in frontier

districts in the 1830s. The massacre at Myall Creek, for example, was public knowledge. Deaths of indigenous people caused by the now unfamiliar disease were not newsworthy.

The first official reports, which were also the most reliable and detailed reports of Aboriginal smallpox in the colony of New South Wales, had come from Scottish regimental surgeons Alexander Imlay and John Mair in 1831. They first saw active smallpox in the Bathurst district; then they and several other military personnel observed active Aboriginal smallpox on the east coast the same year. When the European population of New South Wales grew to over 100 000³⁰ in the 1830s, many other migrants who were familiar with smallpox saw pockmarks and described Aboriginal smallpox in their accounts of the colony. A little later, the Scotsman Thomas Mitchell and Peter Snodgrass encountered recent Aboriginal smallpox in remote districts. Decades later, the Scot minister J. D. Lang was told by an escaped convict who had seen the 1830 outbreak.

In the 1840s it was clear that the Aboriginal population had declined catastrophically. A history of violent dispossession and European diseases was like the history of other European colonies, where the extinction of indigenous people seemed inevitable. In 1845 a Select Committee of the New South Wales Legislative Council was appointed to investigate depopulation and its causes among Aboriginals. Leading colonists appeared before the Committee, and 30 more replied to a circular letter.³¹ Archbishop Polding of Sydney and German missionary William Schmidt at Moreton Bay, who had arrived several years after the second smallpox epidemic, seemed curiously unaware of it. The one person who spoke about smallpox was the Aboriginal witness Mahroot, alias the Boatswain. Only three or four replies sent to the Committee mentioned smallpox as a cause of depopulation, although other correspondents knew of it.

When smallpox occurred in Western Australia in the 1860s, there was little public knowledge in that western colony of the new disaster, and even less in the east. Except for those in the settled northern districts of Western Australia, who saw it or read of it in the press, most European Australians did not know that smallpox was again present in the country until after the damage was done. The Western Australians did not know that smallpox was also present in unsettled and newly settled districts in eastern Australia at

that time. However, with new frontiers and a population of over 1 000 000 in the 1860s and 1870s, for the first time there were settlers and officials in the Northern Territory, Queensland, South Australia and Western Australia who knew something about smallpox and identified its Aboriginal victims during outbreaks, or not long after. Some of them suspected that the origin of recent outbreaks was not European, and a few, who thought fishermen in fleets from the islands to the north were the source of the Aboriginals' infection, left records of what they had seen.

From 1789 onwards, most British colonists thought they were responsible for most, if not all, of the diseases they saw in Aboriginal populations, and they soon found that Aboriginals themselves associated the arrival of the British and their ships with the arrival of smallpox. After frequent contacts with the dispossessed survivors of 1789 and 1830, settlers in the south-east became increasingly aware of other familiar diseases among Aboriginal people. They talked about diseases that were endemic in Europe, especially chronic conditions such as consumption, venereal disease, and occasionally the acute infectious diseases of European children. These diseases, which were seen in newly settled districts in New South Wales, soon occurred in uprooted Aboriginal fringe-dwellers in the newer colonies. Their European origin was not in question.

Early in the nineteenth century, 'catarrh', probably the common cold, and influenza, affected Europeans and indigenous people, especially the elderly. These virus infections die out in small isolated communities, but they were so new in Aboriginal communities that complications would have occurred. Virus infections may have been the prelude to infection with tuberculosis, as they had been in Native American populations a century earlier. Tuberculosis was probably the most common cause of disease that settlers attributed to various respiratory tract infections in the indigenous people. It was certainly the most lethal respiratory tract infection known to Europeans. They called it consumption or pthisis, and initially it could be mistaken for bronchitis or even for a cold. The long-lived tubercle bacillus was transmitted from person to person when infectious droplets, usually coughed up by a sufferer, were inhaled by others, infected their lungs and became chronic. The 'white plague' of industrial

Europe increased between about 1780 and 1850 in both Europe and America. It has been suggested that by the mid-nineteenth century approximately half the English population had been infected. It was not always apparent to sufferers or those who saw them, so introductions in colonial ports could not be controlled by quarantine. Once it was introduced, the slowly developing chronic infection survived among the hunter-gatherers.³²

Tuberculosis is generally regarded as one of the most widespread and damaging diseases introduced into North America by the British. In 1692 an observer in New England said that in the case of consumption, 'sundry of these Indian youths died, that were brought up to school among the English . . . this disease is frequent among the Indians, and sundry die of it, that have not been with the English'. In 1795 an onlooker in Massachusetts said more than half that were born were carried off young with consumption, and another observer said: 'Their tender lungs are greatly affected by colds, which bring on consumptive habits'.³³ Modern American records illustrate the impact of tuberculosis as a new disease. When he investigated its history among Native American nomads at Qu'Appelle Valley reservation in Western Canada in the 1890s, bacteriologist René Dubos showed how the extent of prior contact with the disease affected its impact on succeeding generations. More than half the families in the tribe were eliminated in the first three generations. Then its clinical manifestations changed, and in four generations the acute, fulminating disease seen in newly exposed people began to resemble the common chronic condition known to Europeans.³⁴

The destruction of the first generations of other indigenous peoples hit by tuberculosis in the twentieth century has recently confirmed the severity of the disease in naive (previously unexposed) populations. Using recent evidence, Francis Black showed how rapidly tuberculosis spread as a new disease among isolated pre-agricultural South Americans. In 1962 it affected 5 per cent of the Txukahamae; in 1966 12 per cent of them; and in 1967 32 per cent.³⁵ It was new to the peoples of Australia, where there is no skeletal evidence of its pre-European presence. Records of it in eighteenth-century and nineteenth-century Australia lack such detail, but when effective drug treatment became available in the

twentieth century, it was possible to describe differing manifestations of the disease in Aboriginal and European patients. Newcomers suffered a chronic disease of low-grade activity, while many Aborigines suffered an acute, severe disease. All responded equally well to modern treatment.³⁶

However, anecdotal evidence from settlers indicates that first introductions of tuberculosis had unusually severe effects on indigenous Australians, as they did on Native Americans. Evidence of virgin soil tuberculosis in survivors of smallpox follows in later chapters.

In colonial records, venereal diseases among Aborigines who had contact with European settlers were mentioned as often as tuberculosis. In Hackett's opinion, the bacterial infections syphilis and gonorrhoea, which were the two most common sexually transmitted diseases among Europeans arriving after 1788, were not known in pre-European Australia. Syphilis is a venereally transmitted disease of adults, with genital lesions, followed by changes to the skin and bone, and by severe damage to the heart, arteries and brain. It has been estimated that in England, at least 10 per cent of the adult population was infected with it before effective treatment was available. It has long been universal in large urban populations in Europe and America, and only adults and adolescents are likely to have sexual contacts necessary for its transmission. In 1996 Radolf reaffirmed the worldwide modern prevalence of syphilis, pointing out that this highly invasive disease can attack all body tissues, leaving destructive lesions, and unlike other treponemal diseases, it also causes congenital infection.³⁷ Gonorrhoea is not lethal, and in general it is always more common than syphilis. Gonorrhoea is not particularly destructive in populations where it is endemic, although without effective treatment it becomes chronic and causes sterility in women.

Unlike tuberculosis, which was seen in Aborigines in the 1790s, venereal diseases cannot be identified with certainty in historical records. In 1786 celebrated surgeon and physiologist John Hunter published *Treatise on the Venereal Disease*, which included erroneous material about syphilis, and in the late eighteenth century, *lues venerea*, as it was known, included several diseases not yet clearly recognized as separate diseases. They were all described as

‘venereal disease’ until they were decisively identified as distinct diseases in the nineteenth century. For some time, nineteenth-century British migrants would not have been able to distinguish clearly between these and similar conditions when they referred to ‘venereal disease’ in Aboriginals. Early hospital records suggest that gonorrhoea was more common than syphilis among settlers at Sydney in the 1820s; nevertheless, the apparent presence of syphilis in Aboriginals still attracted attention early in the twentieth century.³⁸

One of the early explanations of the presence of venereal disease in Aboriginals in south-eastern Australia was that it had arrived on the remote south coast with visiting whalers and sealers before the colonies of South Australia and Victoria were occupied by settlers. In 1837 early South Australian settler W. Bromley informed the Governor that he had learnt from a European drunkard that sailors at Encounter Bay cohabited with Aboriginal women, and infected them with venereal disease. Another settler who mentioned venereal disease specified syphilis, and a colonial surgeon at Adelaide in 1840 said he supposed Aboriginals had it. In Hackett’s opinion, if sealers introduced venereal disease, it was probably gonorrhoea, which is infectious for longer than syphilis. However, when he investigated causes of Aboriginal depopulation in 1983, economic historian Noel Butlin speculated that venereal diseases had spread ahead of settlers, and if syphilis was new to Aboriginals, it is ‘possible and likely’ that it spread from the Victorian coast northward from sealers and whalers in Bass Strait for thirty years.³⁹

There seems to be little foundation for early rumours about sailors at Encounter Bay. In 1962 Manning Clark found that not much is known of the life of the sealers at sea or on land, where only deserted campsites on offshore islands remain. Records of whaling and sealing between 1800 and 1806 suggest the export of seal skins was a profitable venture for an adventurous minority, and about 100 000 seal skins were exported in colonial vessels in those six years. At the time, perhaps several hundred sealers on the unsettled coasts of south-eastern Australia, armed with clubs and muskets, might have ensured a steady supply of seal skins, but the later history of the industry is uncertain.⁴⁰ About 10 per cent of the sealers were probably syphilitics, rather fewer would have harboured active infection. Whether they were likely to infect newly met Aboriginal

women with syphilis is a hypothetical question. Explorers in South Australia in the 1830s did not see diseased Aboriginals in coastal districts visited by sealers.

Sturt, who described signs of disease in Aboriginal people on the Murray River during his second expedition, believed that the healthiest tribe was that on the lower reaches of the Murray, near the sea. And on the south coast of South Australia in June 1839 Eyre made no mention of venereal disease. He said: 'Many natives were constantly seen but all were friendly and well behaved. They appeared to frequent the whale fisheries at Encounter Bay, spoke a little English . . .' As Lyndal Ryan said, venereal syphilis apparently did not occur among the Tasmanians, who also were visited by sealers. Like the Tasmanians, the mainlanders may have been protected against syphilis by previous exposure to a different, but closely related, treponemal infection.⁴¹

Observers who saw these diseases in the first century of European settlement described symptoms they thought they recognized. In 1796 David Collins, whose occupation in the colony was legal not medical, mentioned venereal disease in Aboriginal people he had seen at Port Jackson, and attributed it to contacts between European men and Aboriginal women. Collins added that the disease he called *lues venerea* was probably not new to them, because they had a name for it, which implied a knowledge of its dreadful effects.⁴² We might disagree with his opinion, because Aboriginals almost certainly did not know the dreadful effects of venereal syphilis. It is likely that the word they used for it referred not to syphilis, but to a closely related indigenous disease, unknown to newcomers, which resembled the Europeans' disease in some way. As we shall see, in 1833 Charles Sturt would say syphilis was the 'loathsome' disease he had seen among Aboriginals on the Murray on his second expedition.⁴³ As well as being unsure of European diseases, colonists were confused by indigenous infections, such as endemic syphilis and yaws.

Aboriginal diseases in the colony of New South Wales were an important issue when members of the Select Committee on the Aborigines assembled in Sydney in 1845. According to Presbyterian minister J. D. Lang, who was on the Committee, Aboriginals had strange and incurable diseases, but tuberculosis and venereal

diseases associated with sexual relations between European men and Aboriginal women, attracted more attention than unknown indigenous diseases in the discussions that followed. J. B. Polding, a Benedictine, and the Catholic Archbishop of Sydney, said the abuse of females at an early period of life, 'mere children, who are thus made incapable of becoming the mothers of healthy offspring', and 'the universal prostitution of the women, caused by the depravity of the whites', had resulted in the decline of the Aboriginal population.

Prostitution has a long history in Sydney. From the first settlement, the predominantly male convict population far outnumbered the female convict population. In 1830 there were about two and a half men for each woman, and prostitution was common. Extensive government-assisted immigration in the 1830s was designed to redress the imbalance, and boatloads of single women arrived at Sydney. Selection procedures in Britain were altogether inadequate, and on some ships, at least a quarter of the new arrivals were common prostitutes.⁴⁴ The Archbishop's remarks suggest that Aboriginal women were drawn into flagrant prostitution then obvious at Sydney. As a result, venereal disease was a likely outcome for women living in the port city. In the case of syphilis, indigenous treponemal infection may have conferred partial protection, but there was no protection from gonorrhoea. What the Archbishop said about girls who were incapable of producing healthy offspring, and the 'paucity of births' mentioned in the Committee's report suggests gonorrhoea was causing sterility in women.

By 1845 there were about 150 000 people at Sydney. It had the largest population of any Australian town prior to the gold rushes. By comparison, the European population of the future colony of Queensland was then under 3000; but even there, missionary William Schmidt at Moreton Bay said the connection of Aboriginal women with European men was the principal cause of declining indigenous numbers. Schmidt was asked if venereal disease was present before Europeans arrived, and he said he found it prevailing among Bunya Bunya tribes in the early days.⁴⁵ It is unlikely that he saw a European venereal disease 'in the early days', but in clans in the tropical climate of Moreton Bay, Schmidt may have seen yaws, the closely related indigenous infection, without knowing what it was.

Protector William Thomas had arrived in the Port Phillip District in 1838, and saw the European population grow to about 30 000 by 1845. He had found Aboriginals in the Yarra and Westernport tribes in 'an awfully diseased state' after contact with Europeans who had venereal disease: 'old and young, even children at the breast' were affected with it, and some infants were 'literally rotten with this disease'. In 1845 Chief Protector George Augustus Robinson, just back in the Port Phillip District after an inland journey in the south-east, said hundreds had fallen victims to European diseases, and that smallpox, influenza, febris and syphilis 'have extended their baneful influence to the remotest parts of the interior; the latter disease is now almost prevalent throughout the land; ophthalmia is in some districts endemic [*sic*]; cutaneous affection is peculiar to the natives, and prevalent'. Fifty years after Europeans arrived, syphilis was more often mentioned than most diseases as a cause of decreasing numbers of indigenous people: ten written replies to a circular letter mentioned venereal disease among Aboriginals, and 'immoral', 'illicit' or 'promiscuous' intercourse with Europeans.⁴⁶

The reports of venereal syphilis in Aboriginals in those years are ambiguous. Syphilis was entrenched in England, and observers such as Thomas and Robinson may have seen symptoms they associated with it, such as lesions of the face and nostrils in adults, or bony deformities in older people. Thomas, who had apparently seen children and infants suffering from the disease he was describing, had also observed people in other age-groups affected by it. So, contrary to his fears, the symptoms were not those of venereal syphilis, the sexually transmitted disease of European adults. Skeletal evidence of 'boomerang leg', which is the tertiary stage of a treponematosiis, strongly suggests the presence of an indigenous treponemal disease in the south-east, and the most likely explanation of the wide range of the sufferers' ages is that symptoms they saw were those of the primary, secondary and tertiary stages of an indigenous treponemal disease, probably non-venereal endemic syphilis, which occurred farther south than yaws.

Endemic syphilis was unknown and unrecognizable to European observers in the 1840s, and it is not surprising that the protectors and other curious observers attributed symptoms they saw in

Aboriginal sufferers to the only treponemal disease they had seen before, venereal syphilis. It is likely that as well as seeing abrasions, burns and injuries common to bush people, the Europeans saw the lesions of a disease that was being maintained in small nomadic bush families, whose children were each potentially infectious with the complaint for as long as five years. It was also transmitted between families that met periodically for social and economic purposes.⁴⁷

In the 1880s the Victorian squatter and ethnographer E. M. Curr, who had a lifetime's experience of indigenous people, enlarged on similar questions. He worked his way through a long list of European ailments in Aboriginals, and said venereal disease, introduced by Europeans long before, had committed terrible ravages, and probably would at length exterminate them; and in Curr's day the incidence of venereal diseases would have increased during the gold rushes in Victoria. Curr continued, saying madness and 'inflammation of the brain' were almost if not quite unknown to Aboriginals, but

They suffer, however, a good deal from a sort of pustular itch, said to be peculiar to them, as it is certainly indigenous, which attacks the whole person . . . Persons of all ages . . . suffer from it. Those affected scratch the pimples with which they are covered (especially on the chest, back and thighs) with shells.⁴⁸

The indigenous disease Curr saw may have been the same as the one Thomas and Robinson had mistaken for a venereal disease. It was almost certainly non-venereal endemic syphilis, and it would have gradually disappeared in the small populations that survived smallpox and European settlement in the south-east, as it is now disappearing in the Northern Territory.

Unfamiliar treponematoses were largely ignored in the first century of European occupation; then a decade after Curr's day, an indigenous infection unexpectedly surfaced in the Centre. Writing from Alice Springs in 1897, station master, ethnographer and Protector F. J. Gillen told Baldwin Spencer, a university scientist and an anthropologist, about *erkincha*, a disease that attacked Aborigines only once, generally in childhood, which they called an itch. Gillen's

account of this disease, and how it differed from syphilis, was later known to Hackett. It was not yaws, which occurred not in the interior, but on the humid north coast of Arnhem Land. Nor was it venereal syphilis, as the German traveller Erhard Eylmann told Gillen when he visited the Centre, because its symptoms differed, and were milder. Hackett, who found more evidence of it in the Musgrave Ranges and elsewhere in Central Australia, said it was non-venereal endemic syphilis, and that a childhood attack protected against syphilis in adult life.⁴⁹

In his study of treponemal infections in 1936 Hackett extended his investigation beyond the south-east and the Centre, and interpreted records of treponematoses in Queensland and Western Australia, thus establishing their widespread prevalence. At Townsville, syphilis was uncommon, which was attributed to the prevalence of yaws. From Western Australia in 1844, Governor Hutt said 38 cases of a 'loathsome' venereal disease had occurred in Aboriginal camps beyond York in 1841. The local hospital had treated 17 people and 10 of them improved. A similar disease occurred at King George's Sound.⁵⁰

The last observer to attribute the destruction of traditional Aboriginal societies to the introduction of venereal syphilis was medical practitioner and anthropologist Herbert Basedow, who became Chief Protector and Medical Inspector of Aboriginals at Darwin in 1911. He resigned after only two months in the Northern Territory, returned to Adelaide, and later said venereal syphilis was 'the most formidable in bringing about the speedy decimation of Aboriginal tribes'. Basedow's opinion was influential.⁵¹ The suggestion that syphilis, once introduced, spread rapidly through Aboriginal communities, disregarded the conventions of tribal societies, which discouraged sexual relations between members of certain clans and thereby slowed the spread of disease. Basedow exaggerated the impact of syphilis as a cause of Aboriginal depopulation. Writing some years before Hackett and Chambers, he did not realize that indigenous treponemal infections, which are milder than venereal syphilis, protect against it. By now, it has also been recognized that a higher incidence of venereal syphilis followed the use of antibiotics in the twentieth century to treat indigenous treponemal infections.

For some years after the first settlement, it was the absence of the acute infectious diseases of a European childhood in both European and Aboriginal populations in the colony, not their presence, that attracted comment. A few settlers, such as Worgan, who remarked on the health of Aboriginal populations before contact with Europeans, were surprised by the absence of these diseases. Escaped convict William Buckley reported they were not known in clans he had encountered near Port Phillip. Anthropologist Daisy Bates said the same thing a century later on the west coast. In the nineteenth century, settlers sometimes attributed the decline in Aboriginal numbers to these diseases, which were virus infections, in particular measles, and bacterial infections, such as whooping cough and scarlet fever. Unlike tuberculosis or venereal diseases, those diseases could die out on the voyage from Britain. In the 1820s their absence in the European population seemed extraordinary to a French visitor, who said there was no measles, no scarlet fever and no whooping cough; and puzzled settlers, such as Peter Cunningham and Robert Dawson, remarked on the absence of measles among Aboriginal as well as European children. After 1828, when ships arrived with infectious passengers, introductions in colonial ports were usually prevented by the introduction of quarantine.⁵²

It is now known that the prevalence of infectious diseases is closely related to population size. Without new outside introductions, measles is endemic only in populations of at least 200 000. Colonial populations, even Sydney's, were too small before the gold rushes to support measles and other acute infections. In the 1830s outbreaks of childhood diseases were still infrequent and erratic. However, measles attacked some Aboriginals in a newly settled district in the 1830s, and, as Charles Darwin said and the Mudgee magistrates knew, sporadic outbreaks among Aboriginals were severe.

When the gold rushes brought infectious new arrivals, and there was a larger European population, it was some time before European diseases became endemic. Measles was still not endemic in Sydney in 1860, and when an outbreak occurred, some European children died of it. When the immigrant population of Australia was about 1 000 000, there were some 13 000 cases of measles among Sydney children in 1867, and over 700 died.⁵³ In 1860 Aboriginal

people in the south-west of Western Australia were also exposed to measles, following a shipboard introduction, with similarly severe results. When it became endemic among immigrants, epidemics in settled districts affected Aboriginal people more often, although populations in distant parts of the country remained relatively free of childhood infections for long periods in the nineteenth century. Virgin soil epidemics of measles were occasionally seen in remote modern Aboriginal communities in the twentieth century.⁵⁴

The growing conviction that Aboriginals could not survive the crises of violent dispossession and disease, expressed in the 1830s, was reinforced by the Tasmanian experience and by the startlingly obvious decline of Aboriginal populations in South Australia and Victoria, where in 1858 the *Melbourne Age* told its readers to 'smooth the pillow of a dying race'. To the changing public of the gold rushes and the towns, who knew little of indigenous people or remote frontiers, the sentimental image may have offered a plausible explanation of the small numbers of Aboriginal Australians. The notion that the race was 'disappearing' was still common early in the twentieth century. Charles Rowley and Henry Reynolds thought the Europeans' preoccupation with the impact of disease among Aboriginals in the nineteenth century concealed a history of frontier violence, and the rediscovery of a violent racist past lends weight to that opinion.⁵⁵

Today, it is the lethal damage that Old World diseases did indeed inflict on New World peoples that is being re-appraised. The sometimes fatal effects of diseases that settlers brought diverted attention from the disasters smallpox had already caused in Aboriginal Australia. Half-forgotten, smallpox was the worst disease ever seen among indigenous Australians, and was the first and major single cause of the decline of Aboriginal populations on the mainland between 1780 and 1880. Ancient chains of social connections became chains of infection that caused the strange and terrifying disease, which undermined customary life before Europeans did. Except when it attacked several colonists who had close contact with sick Aboriginals, and a few unvaccinated children, it was the one new disease seen in Aboriginals that was conspicuously absent in settlers, who were free of it until the *Commodore Perry* arrived at Melbourne in 1857.⁵⁶

Recognition of the impact of Aboriginal smallpox was sparked when Europeans themselves were shocked by the incidence of the disease that killed and scarred newcomer victims. The 1857 outbreak occurred when infectious passengers on the *Commodore Perry* landed, and was over after only 4 deaths among 16 sufferers, but the next outbreak in Victoria was more alarming. After the *Avondale* arrived in Melbourne in 1868 there were 43 cases and 10 deaths in the following six months. After the arrival of the *Nebraska* in 1872 there were 10 more cases, including an infected family who went to Sandhurst (Bendigo), where 3 of their children died. A smallpox death at Newcastle that year was also connected with the *Nebraska*, but in 1874 another smallpox death on the Newcastle waterfront was inexplicable.

These unexpected small outbreaks reminded settlers that the disease could still occur, and so did the news from home. Jenner's discovery had made Europeans complacent, and the cost was high. The Franco-Prussian war triggered a smallpox pandemic that killed 500 000 unvaccinated Europeans between 1870 and 1875. Infection crossed the ocean to other continents. In the summer of 1877 a dozen cases of smallpox occurred on the Sydney waterfront. The press reported that 4 or 5 victims died.⁵⁷

The Sydney fatalities prompted the editor of the Melbourne *Argus* to draw attention to the lack of records of the earlier history of smallpox among Aboriginals, and to urge old colonists to preserve all information still available, so the first chapter of the colony's medical history might be written. In January and February 1877 more than twenty letters on the subject were published in the paper's correspondence columns. Most of the letter writers can be identified today. Some, such as politician and medical practitioner Thomas Embling, who arrived in 1851, had not seen Aboriginals until well after the outbreaks, and called the disease 'native pock'. But older settlers remembered Aboriginal smallpox clearly.⁵⁸

During the next thirty years, smallpox occasionally occurred among immigrant Australians. The worst episode was Sydney's nine months in 1881–82, when there were 154 cases, including 40 deaths, and, as Curson relates, unprecedented hysteria and panic in the city. Sydney and Melbourne suffered again in the 1880s, and the public heard of the bizarre case of the victim who walked 150 miles along a

Victorian railway track, became delirious and infectious, and was arrested at Border Town as a lunatic. There were no cases in South Australia or Queensland, and except for unvaccinated children in the 1860s, Europeans in Western Australia escaped until 1893, when 50 cases followed the arrival of infectious passengers on the *Saladin*. Smallpox then horrified Launceston in 1887 and 1903.⁵⁹ Late in the nineteenth century, when settlers had experienced smallpox again, the history of Aboriginal smallpox emerged. Explorers, colonial authors and tellers of tales knew Aboriginals had suffered in outbreaks of an unpredictable and frequently fatal disease. Edward Micklethwaite Curr, who had seen its traces in the 1840s among Aboriginals on his father's Murray River runs, was one of several ethnographers who wrote about it in the 1880s.⁶⁰

Smallpox among Europeans in Australia had presented immediate problems, often urgent ones, for members of the medical profession, and it revived their interest in Aboriginal smallpox. Medical scientists published scholarly and intuitive studies early in the twentieth century. In July 1911 Sir Edward Stirling, a distinguished physiologist, addressed the Royal Society of South Australia on the subject. He was familiar with historical records and with ethnographic work by Curr and others. Investigating a range of sources, he inspected excavations on the Murray, recorded oral history after talking to Aboriginal survivors and European settlers, and corresponded with informants, including F. J. Gillen, who was on the Horn expedition with him in 1896.⁶¹ The pathologist John Burton Cleland (later knighted), wrote a paper on the same subject as Stirling, which he read in Sydney later that year. In a fuller publication in 1928, and again in 1966, Cleland made it clear that he and Stirling, who had worked independently and unknown to each other, had arrived at similar conclusions, which were that Aboriginals had several times suffered extensively from smallpox introduced on the north coast.⁶²

The third scientist who recognized the significance of historical records of Aboriginal smallpox was J. H. L. Cumpston, who published its history in 1914. He was professionally committed to public health, and controlled Australia's Federal quarantine service. He was immediately confronted with cases of mild smallpox, newly introduced by American Mormons. From 1921 to 1945 Cumpston

was Australia's first Director-General of Health. He thought proximity to Asia was a good reason for reliable quarantine, and also for painstaking research about previous introductions of diseases. He thought it curious that there had not been more introductions of smallpox. He found no evidence of it in the First Fleet, or in the French fleet, but, unlike Cleland, Cumpston could not dissociate Aboriginal smallpox in 1789 from the recent arrival of a comparatively large number of Europeans. He had no explanation for it in 1830, but he did attribute smallpox in the 1860s to Asian sources.⁶³

There never was any evidence of the origin of Aboriginal smallpox at Sydney in 1789. The only reliable records of it in New South Wales in 1830 show that it spread from the interior and reached the east coast late in 1831. There were no shipboard introductions in Victoria before 1857. As Cumpston said, it originated on the north coast in the 1860s. By the 1980s failure to connect the arrival of Europeans in 1788 and the arrival of smallpox in 1789 led to wild speculations about the identity of the disease and its origin. In subsequent chapters we shall discuss smallpox itself, then review the many myths about Aboriginal smallpox that have accumulated since the eighteenth century.

2

‘the most dreadful scourge of the human species’

The last recorded outbreak of Aboriginal smallpox occurred in 1870, when there was an epidemic in north-western Australia. The last case of smallpox in Australia occurred during World War I. The last endemic case in Europe, where it was prevalent for centuries, occurred in 1953. The last case in Asia was in 1975, and the last in Africa, and the world, occurred in 1977. Very few Australians have ever seen a case of the disease, and even in its ancient strongholds, India and China, the majority of physicians now practising have never seen a case.

Memory of the disease has been kept alive longer in India, China and populous parts of western Africa because in these places there are gods and goddesses of smallpox, although their role is now being revised. Even in more sparsely populated places where smallpox used to be endemic, memory of the disease soon died out. The last endemic case in Rhodesia (present-day Zimbabwe) occurred in 1970, and when surveys of the population were made in 1978 as part of the global certification programme, it was found that persons under twenty-five years of age could not recognize smallpox when they were shown photographs of its victims, nor did they remember anything about it.¹ Pockmarked faces, once commonplace in the streets of cities in India, Bangladesh and China, are now seen only in a few old people.

The documentation of the global eradication of smallpox has provided an unprecedented legacy of information about it. During the Intensified Smallpox Eradication Programme, for thirteen years from 1967 until 1979, the worldwide incidence of smallpox was monitored more carefully than any other disease has ever been. The way it was behaving in many individual instances and in widely differing societies was observed and recorded by hundreds of physicians and medical scientists who worked with thousands of health workers. Existing knowledge of smallpox was reviewed and expanded, using sophisticated laboratory techniques as well as traditional clinical observation. New knowledge was sought and often won in order to eradicate the disease systematically and successfully, country by country.² After that extraordinary international achievement, a small group of those who had participated in the research consolidated, recorded and in 1988 published their remarkably diverse knowledge of the first disease the world had ever contemplated annihilating.

As Donald Hopkins illustrated in *Princes and Peasants: Smallpox in History*, the comprehensive information gained by this research provided a framework for interpreting evidence of the now-extinct disease. The behaviour of smallpox in unvaccinated modern subjects is the only way of finding out how it affected unvaccinated historic populations. In 1953, before any modern Australian scholars investigated it, J. B. Birdsell, who knew of one Aboriginal epidemic, said the magnitude of its impact cannot now be estimated.³ Difficulties he anticipated are now less daunting: there are many contemporary accounts of Aboriginal smallpox in published sources and in government archives, and recent medical literature makes it possible to understand and evaluate them. Current knowledge of smallpox both enlarges and sets limits to what can be discovered about its history. Comparable historical research is proceeding in the Americas and Africa.

In the 1980s news of the global eradication of the disease sparked a new discussion of Aboriginal smallpox. The Australian evidence and its implications have not yet been fully explored. With some exceptions, such as Kevin Gilbert and Wayne Atkinson, most indigenous and immigrant Australians are unfamiliar with the historical record. In 1983 Noel Butlin published a study of Aboriginal

depopulation 'from the point of view of an economic historian', and rated smallpox, introduced by Europeans, as its main cause in south-eastern Australia between 1788 and 1850. The demographic impact of two epidemics was central to Butlin's speculative and hypothetical study, but he mentioned surprisingly few of many records of smallpox in the south-east. When he constructed demographic models of hypothetical Aboriginal populations, he used a study of smallpox in modern India of unvaccinated subjects to simulate its impact. Like earlier scholars, he assumed that indigenous Australian populations were new to the disease and susceptible—that is, open to infection. In his demographic models, he also assumed that entire populations were exposed to infection, and that all those born before the first epidemic were immune when it recurred forty years later. He chose a case-fatality rate of 45 per cent in both epidemics; that is, he assumed that nearly half of those who caught smallpox died of it. His revisions of pre-contact populations were confined to the south-east, and, he insisted, 'do not imply comparable adjustments for Australia as a whole'.⁴

Since 1983 different interpretations of the evidence have led to differences of opinion about Butlin's work. Peter Curson was not unsympathetic with Butlin's inference about a European origin of smallpox in 1789; however, he considered that at Port Jackson the epidemic was sharp and short-lived, and 'its effects and extent have been exaggerated out of all proportion'. British historian Charles Wilson used medical microbiology to disagree completely with Butlin about a European origin in 1789, and doubted his estimates of its demographic impact. Recently First Fleet historian Alan Frost also disagreed with Butlin's views.⁵

In a summary of former estimates of the Aboriginal population of 1788, J. Peter White and D. J. Mulvaney departed from A. R. Radcliffe-Brown's 1930 estimate of 'probably over 300 000', which, in the last fifty years, has often been accepted. White and Mulvaney's demographic reappraisal used modern archaeological and historical research, and environmental studies, which suggest the land and its resources might have supported significantly larger populations, at least in temperate and coastal regions. They also accommodated Butlin's estimate of the impact of diseases brought into Australia by Europeans from 1788 onwards, acknowledging

that his simulations used 'various probable (though unverifiable) assumptions'. Using Butlin's formula, which emphasized the impact of smallpox in the south-east, the authors arrived at a population of 900 000 for the whole country in 1788. This figure was then reduced to 750 000, a plausible figure, which assumed that the impact of smallpox was less in the north than the south-east.⁶ For various reasons, it is time to review historical evidence in Australia, and the context of recent medical literature is apt.

Smallpox was known as a uniformly severe and dangerous disease wherever and whenever it occurred in human societies. Occasionally there were lucky mild cases, even mild outbreaks; but there was no such thing as endemic mild smallpox. Then, at the end of the nineteenth century, a disease called kaffir-pox or *amaas*, which looked like smallpox, but killed only 1 per cent or less of unvaccinated victims, appeared in South Africa. About the same time a similarly mild disease occurred in North America, spread to South America, where it was known as *alastrim*, then to Europe and Australia. In May 1913, when a ship's steward who had come from Vancouver infected a girl at Sydney, the initial diagnosis was chickenpox. By January 1914 over 1000 people, who had been quarantined, were diagnosed as smallpox cases. It spread for four years in New South Wales, with 2400 cases. Two Aborigines and several families with Aboriginal forebears were among over 1500 people admitted to North Head quarantine station in the first two years. But except for 5 cases in Queensland, it did not spread, and only 2 people died. Introduced to New Zealand in 1913 by a Mormon missionary from the same ship, it lasted about a year, affected 114 Europeans all of whom recovered, but caused at least 1778 cases and 55 deaths among Maoris, who had never previously been exposed to smallpox. In the mid-1950s virological studies established that these outbreaks were indeed mild varieties of smallpox.⁷

It was necessary to identify this new kind of smallpox, and to avoid confusion between the very harmful and the almost harmless varieties. The solution was simply to use a synonym for smallpox. Diseases characterized by pustules or ulcers had always been called pocks, and small pocks were eventually associated with one disease only. When the medical treatises of Muslim physicians Rhazes and Avicenna were translated into Latin about a thousand years ago, the

word *variola* was used to describe the same disease.⁸ To distinguish between the two varieties, mild smallpox with its very low case-fatality rate was called *variola minor*, and 'classical' smallpox, with case-fatality rates that were often 20 per cent or more in the unvaccinated, became known as *variola major*. Only *variola major* was known before 1890. It had caused all the epidemics of smallpox throughout the world until then. Aboriginal epidemics in the eighteenth and nineteenth centuries were *variola major*, not the mild disease.

Knowledge of the smallpox virus and viruses used to control it was central in the science of virology early in the twentieth century, and was crucial in the eradication of smallpox. Other diseases called 'pox' were confused with smallpox. 'The great pox', syphilis, caused by the spirochete *Treponema pallidum*, and chickenpox, caused by a herpes virus, were sometimes mistakenly confused with it. Smallpox was a completely separate disease. It was caused by the *variola* virus, a species of *Orthopoxvirus*, and a specifically human virus, with no natural host other than people.⁹ Detailed research on effects of temperature and humidity on virus in smallpox scabs directly affects the interpretation of evidence of cases in Sydney in 1789.

Recent medical knowledge contributes significantly in other ways to the interpretation of historical records. Contemporary descriptions of Aboriginal smallpox can be evaluated alongside modern medical studies of the disease, its symptoms and usual effects on unvaccinated people. When A. R. Rao classified and described differing clinical types of *variola major* in 1972, the commonest type was what he called ordinary-type smallpox, which occurred in 88.8 per cent of unvaccinated subjects in Madras. Those cases were divided in relation to the density of the rash, which affected the outcome. In the unvaccinated, when the lesions were confluent (running together) on the face and forearms, the case-fatality rate was 62 per cent; when they were confluent on the face and discrete (separate) elsewhere, it was 37 per cent. When they were all discrete, with areas of normal skin between them, even on the face, the case-fatality rate was 9.3 per cent.¹⁰

The first symptoms followed an incubation period that was usually twelve days, but could be as short as seven days or as long as nineteen days, after infection was transmitted. Then seemingly well

victims suddenly suffered a very high fever and often chills. With temperatures of at least 40°C, they usually suffered splitting headaches and severe backaches, and other unpleasant symptoms in the first days: half of them vomited, some had colic, some were delirious, and children had convulsions. By the second or third day, when they might have felt a little better, the rash appeared.¹¹ The fever marked the onset of generalized viral infection and was followed by an eruption on the mucous membranes and the skin, and within twenty-four hours the rash could be seen on all parts of the body. The 'crop' of lesions evolved in about ten days, and in the mouth and throat, which were sore, they broke down quickly, releasing large quantities of virus into the saliva, which became a major source from which virus was transmitted to others. The rash on the skin developed, with raised vesicles that became pustules within a week. The fever returned and the pustules grew larger.

In fatal cases, death usually occurred during the following week, while the lesions of those who had survived began to flatten. Scabs or crusts began to form and started to come off the face. By then the temperature had fallen, and the sufferer felt much better.¹² The rash had a characteristic pattern, and was most dense on the face and more dense on the extremities than on the trunk, especially on the palms of the hands and the soles of the feet. After several weeks most lesions had healed and their scabs had separated, except in the thick-skinned palms of the hands and soles of the feet. They could persist there long after the fourth week, unless they were removed.¹³

Ordinary-type smallpox included cases of widely ranging severity, and the number of pustules varied from a few to several thousand. Over half the unvaccinated cases in Madras were confluent or semiconfluent, and mild cases did not often occur.¹⁴ Several severe types of the disease, which were usually fatal, were not common, even in the unvaccinated. In one exceptionally severe type, pustules were rarely seen; in the very rare haemorrhagic-type, sufferers died before the rash developed. Two-thirds of the Madras cases of that type were women, especially pregnant women. Sometimes these extreme forms were associated with a severe shock-like condition said to be unlike the effects of any other infectious disease.¹⁵ In unusually severe forms of smallpox, death was associated with overwhelming infection by the virus.

The universal perception of smallpox as a dreadful and frequently fatal disease was heightened when very distressing types occurred. However, the likely outcome of ordinary-type smallpox in unvaccinated people provoked tremendous fear. There was no cure, and people had faith in strange remedies, such as the 'red treatment', which were all completely ineffective. Even modern scientific medicine had no effective therapy. More than half of the unvaccinated victims suffered the full force of severe varieties. Many modern sufferers were cared for by immune or vaccinated relatives in hospitals or at home. But there were limits to the relief that even devoted care provided. Feverish and very sick people couldn't breathe or speak properly, or swallow anything, even water, despite agonizing thirst. Suppurating pustules left the skin inflamed, sore and raw, attracting swarms of flies.¹⁶

Restoration of health and well-being did not always follow recovery from the severe acute illness, but was often delayed by complications. Respiratory symptoms, especially bronchitis and coughing, were relatively common symptoms of severe smallpox, and secondary bacterial infection often invaded skin lesions. One observer early in the twentieth century described survivors with 'thousands of boils'. More insidiously, the virus infection caused lesions in the growing bones of children during their severe illness. The resulting bone defects were usually not recognized until years later, when inexplicable limb deformities were seen. However, the hypothesis that smallpox was a significant factor in male infertility was not substantiated.¹⁷ Shortly after recovery, the sites of pustules were usually depigmented in dark-skinned subjects. Most people who recovered from *variola major* had pitted scars, known as pockmarks, on the sites of some pustular skin lesions. These were depressed scars, two millimetres or more in diameter, and usually circular, the number varying between one and several hundred. They were most common on the face, where sebaceous glands were often badly affected by the rash, and 65–80 per cent of survivors had permanent facial pockmarks. Moderately severe pockmarks were still apparent, but sometimes faded in a decade; many people were more severely afflicted and remained badly scarred. When bacterial infection occurred, pockmarks were often worse and more irregular.¹⁸

Traditionally, blindness was also associated with smallpox. At the end of the eighteenth century, in unvaccinated Europe, smallpox

may have caused about one-third of all blindness. A century later, observers in Asia described people who were blinded by smallpox in China, Japan, the Philippines and India, and some high estimates of the blindness were like earlier European estimates of it. It is now clear that corneal ulceration, scarring and blindness, which sometimes occurred after smallpox, were unusual in affluent modern European populations. It was more often a result of smallpox in undernourished and unvaccinated people with poor hygiene.¹⁹

Some people found smallpox and its permanent results were so damaging to their morale that they preferred suicide to survival. Hopkins said sufferers in Japan, Nepal and North America made that choice. Historian Peter Wood related that in the eighteenth-century South, some Cherokee survivors shot themselves, cut their throats or stabbed themselves to avoid the shame of disfigurement, and others threw themselves into the fire in a state of madness. These excesses mirrored the terrible despair and desolation that smallpox caused some victims.²⁰ The only advantage that survivors enjoyed was the high degree of immunity to reinfection. Immunity usually lasted for life, but second attacks were possible, as long as fifteen or twenty years later.²¹

Surveys of case-fatality rates of smallpox in different societies in the twentieth century, undertaken by Frank Fenner, illustrate the known range of its modern demographic impact. Case-fatality rates in unvaccinated people leave no doubt about its frequently fatal outcome, even in societies where it was a familiar menace, and the previous experience of immune survivors mitigated its disastrous effects on new victims. Many fatalities regularly occurred among people of particular ages, as an epidemic in Liverpool, England, indicated in the twentieth century. Nearly half of unvaccinated infants under four and more than half of unvaccinated adults over thirty who caught the disease died. More sophisticated records in yesterday's India provide reliable examples of those trends. Rao's series of smallpox cases in hospitals in Madras, 1961–69, represents 80–90 per cent of all cases there at the time. The age-specific case-fatality rate in unvaccinated persons was slightly greater than it was in Indian states described later by Basu, and used in Butlin's models.²²

Hospitalized patients in Madras (Rao 1972)

Age-groups (years)	Number of cases	Case-fatality rate (%)
0-4	2091	41.7
5-9	708	22.2
10-14	154	11.7
15-19	143	22.4
20-29	260	39.2
30-39	91	44.0
40-44	32	37.0
>45	55	61.5
Total	3544	35.5

As Rao's descriptions of types of *variola major* indicated, case-fatality rates in the unvaccinated were decisively influenced by the sex as well as the age of victims. Smallpox was always more severe in pregnant women, who usually died, than in non-pregnant women or men in the same age-group, and the worst varieties were at least twice as common in pregnant women as in the rest of the population; pregnancies usually ended even if the mother lived. Women who were not pregnant were slightly more susceptible than men, and for all of these reasons the case-fatality rate of smallpox was higher for women in general than it was for men. In Rao's series of unvaccinated cases, the case-fatality rate for all women was 40.8 per cent, made up of 61.1 per cent for pregnant women and 34.7 per cent for non-pregnant women, while the case-fatality rate for men was 30.2 per cent.²³ It seems unlikely that smallpox fatalities went unnoticed by contemporaries when epidemics inflicted personal crises, such as the loss of children and childbearers in small communities. Modern research might explain the impressions of well-informed observers. In the late nineteenth century, when severe smallpox epidemics occurred in Indo-China, the French medical director of Saigon's Pasteur Institute described villages where smallpox not only killed the children, but 'left so disproportionately few females of marriageable age that it had an unusually long-lasting depopulating effect'.²⁴

Smallpox was an infectious disease, and its behaviour in human populations constituted the epidemiology of the disease, which involved examining its incidence (how many people in a specific population caught it), mortality attributable to it (how many died of it), and factors that determined its occurrence and spread.²⁵ This dimension suggests ways of selecting and interpreting historical evidence of smallpox in unvaccinated Aboriginal populations.

In the developing countries of the modern world, the incidence of smallpox was nearly always underestimated, and it was necessary to find reliable ways of calculating the number of cases independently of official reports. An ultimately successful method relied on surveys, by experienced observers, of pockmarks on the faces of people in countries where smallpox was endemic. The centuries-old way of recognizing the effects of smallpox had been put on a scientific basis, and was important in estimating its recent incidence in entire populations.²⁶

The incidence was usually seasonal. It occurred much more often in winter and spring, and was called 'the spring rash' in Bengali, although seasonal differences were less evident in tropical climates. *Variola* virus remained active for longer periods in cold and dry weather than in hot and humid weather, and its human hosts unwittingly collaborated with it. People in traditional settled societies moved around more for social occasions in the dry season, which was a good time for travelling long distances, so infection spread easily.²⁷

Estimates of the mortality caused by smallpox depended on case-fatality rates, which varied considerably in different geographic regions, because strains of *variola* virus in various parts of the world differed in their virulence in people. In unvaccinated individuals, case-fatality rates ranged from 5 to 40 per cent in cases of *variola major*. Asian *variola major* had consistently high case-fatality rates of 20 per cent or more. In Africa case-fatality rates usually varied from 5 to 15 per cent. Some characteristics of case-fatality rates were surprisingly consistent. Pregnancy always affected the outcome, even in cases of *variola minor*. Young children, from infants to four-year-olds, were always singularly susceptible, and their survival was uncertain in African as well as Asian populations. Unvaccinated

people over forty were also more likely to succumb to smallpox and to die, regardless of where they came from. But until the second half of the twentieth century, records of incidence and case-fatality rates were usually too fragmentary to estimate the mortality it caused. In Fenner's opinion, historical documents suggest high case-fatality rates had always prevailed everywhere, and 'Indeed, epidemics in previously unexposed, subsistence-farming populations [in the Americas and Africa] appeared often to have produced much higher mortalities than those seen in Asia, owing to the social disruption and consequent starvation that they caused'.²⁸

Hopkins found evidence of high mortalities attributed to smallpox in parts of Africa in the eighteenth, nineteenth and twentieth centuries, reinforcing Fenner's conclusion that no population was exempt from its devastating impact. The effects were worse in societies that were unfamiliar with it and ill-equipped to deal with it. When ships from India, Ceylon and Denmark introduced smallpox to the new European colony at Capetown in 1713, 1755–56 and 1767, epidemics that occurred in South Africa were an unmitigated disaster for the Hottentots. Smallpox was new to them, entire clans were wiped out in the initial epidemic, and hostile tribes killed those who fled.

The second epidemic, and perhaps also the first, spread inland in south-west Africa and destroyed the identity of the remaining individual Hottentot tribes. Variolation (inoculation with smallpox matter to cause a mild attack and induce immunity) was unknown to them, and they suffered more severely than Bantu tribesmen, who had learnt from the Dutch how to inoculate. Smallpox mortalities ended resistance on expanding frontiers of European settlement by demoralized and depleted Hottentots.²⁹

When inland tribes in east Africa caught smallpox for the first time, mortality rates for the Griqua in 1831 might have reached 80–90 per cent of the total population. In northern Sudan in 1814 only one-third of those attacked recovered. In Kenya at the turn of the century, when the Kikuyu were the victims, 70 per cent of the population died in the Kiambu district during an epidemic.³⁰

If smallpox was present in a population, the way it infected its hosts and its infectivity determined its occurrence and spread, which

seemed relentless in naive populations. It usually entered the body via the respiratory tract, and the source of infection was nearly always virus shed by a recent victim in the highly infectious secretions of the nose and mouth during the first week of illness. When the sick person breathed, talked and coughed, infectious liquid droplets were spread around. Face-to-face contact was the most frequent cause of infection for others, although later there were large amounts of virus in scabs, which could retain infectivity for a long time. But virus in scabs was a much less significant source of infection than oropharyngeal secretions. Virus particles could easily enter the nose or mouth of susceptible contacts, either by inhalation or on contaminated fingers, so there was a high probability that the unvaccinated would be infected if they were exposed to fresh oropharyngeal secretions.

The transmission of infection occurred just after the rash appeared, then infectivity was at its height for about a week. The relative infectiousness of cases varied. The most severe cases, which occurred in unvaccinated people, were also more infectious. Early severe toxæmia virtually segregated the very sick, yet sometimes those who died had been more infectious to family contacts than those who survived. Infection was occasionally associated with handling inanimate objects (fomites), which included bedding. Even less often it seemed to be airborne over distances of several metres. But the vast majority of cases could be traced to close contact with a person who had a rash.³¹ In the overwhelming majority of secondary infections, 'contacts' who were exposed to the risk of direct infection by the victim, or infection by aerosol a short distance away, were members of the family who lived and slept near by. Casual contacts were not usually infected.³²

The effects of infection might have been influenced by genetic factors, but racial differences in susceptibility were never convincingly demonstrated, and genetic selection for resistance to smallpox in countries where it was endemic for a thousand years or more was not definitively established. Individual susceptibility was influenced by physiological factors mentioned above. Smallpox was most severe in the very young and the elderly, consistently much less severe in the age-group 5–14 years than in any other, and pregnancy was associated with the highest susceptibility. However, when

populations were first exposed, people of all ages and both sexes were affected, and when it occurred after some years of freedom from it, most of the susceptible individuals who had accumulated in its absence were affected. At such times, social and economic disruption was more devastating than when smallpox was endemic.³³

The spread of smallpox depended on factors other than biological ones, such as climatic factors, which affected social behaviour and opportunities for infectious contacts. More importantly in some countries, demographic factors, in particular the size and density of the population at risk, affected chances of contact between infectious and susceptible people, and thereby the extent and rapidity of the spread of smallpox. Francis Black showed that in islands of roughly comparable size, the duration of epidemics of measles (and smallpox would be similar) was inversely proportional to the density of the population.

Social factors were also relevant. The more active members of communities were more likely to transmit infection. Males between five and twenty years moved between family groups more frequently than others, and often sustained relatively mild infections, which they introduced to previously unaffected family groups. When smallpox had not occurred previously, or at least not for many years, and a large segment, perhaps all, of the population was susceptible, epidemics were characterized by very high case-fatality rates in all ages and both sexes, and high mortalities. In those circumstances, whole groups perished, as observers said they did after Europeans arrived in the Americas and South Africa. The lethal effects of the new disease itself, in populations that were immunologically and genetically naive, were combined with social disruption and famine following the simultaneous sickness of so many.³⁴

Patterns in the way it spread emerged when the real incidence of smallpox was accurately established. The spatial distribution of cases was unpredictable, because it was never uniformly distributed within a country. When the incidence was high, cases were usually scattered; when it was endemic, there were many small and scattered outbreaks. The distribution of cases by district was patchy, and it was patchy even within affected districts. If smallpox was not recognized, its spatial distribution was even more unpredictable, as the unexpected Yugoslav epidemic of 1972 showed.³⁵ By now, there is

substantial agreement among epidemiologists that the rate of spread in small groups was usually rather slow, yet previously it was believed that smallpox was more infectious than most diseases.

Recent research suggests why differing conclusions are not altogether irreconcilable. When available modern data on first-generation secondary attack rates of several infectious diseases are compared, it appears that *variola major* was nearly always less infectious than measles or chickenpox. Nevertheless, the overall average secondary attack rate of *variola major* in unvaccinated family contacts was 58.4 per cent, well over half of those exposed. In a dry year in the Punjab, the remarkable secondary attack rates of 73.3 per cent and 88.4 per cent, high for any infectious disease, were among those reported. To the last, curious and unusual cases demonstrated that very brief exposure occasionally resulted in unexpected transmission of infection from a completely unsuspected source. In 1970 in Meschede Hospital, someone who had no personal contact with any smallpox case contracted it, probably after being exposed to airborne infection for fifteen minutes or less. In 1972 in Yugoslavia, one index case (that is, the first case in a previously unexposed district) infected nearly 40 people in several different places, who had secondary attacks. In 1977 in Somalia, the last case of endemic smallpox in the world occurred in hospital cook Ali Maow Maalin. He was exposed to infection for only a few minutes while he directed a vehicle carrying two active cases of *variola minor*.³⁶

Family and household life was usually the setting for face-to-face contact and transmission of infection from person to person, but various institutions (for instance, markets and festivals) often harboured visitors who were exposed and went home incubating the disease. Travellers of all sorts, especially adult men, who moved about more than women or children, while still apparently healthy, covered long distances and could introduce the disease in places a long way from where they had acquired it. In rural Pakistan, although there were few cases during the monsoons, outbreaks occurred immediately after the rains, when more people travelled. Prolonged and intimate exposure of susceptible individuals to infection was usually necessary before transmission occurred.³⁷

Perhaps the most surprising feature of the careful surveillance of smallpox during the eradication campaign was its occasional prolonged transmission in small isolated populations of nomads. It seemed unlikely that infection could persist in those circumstances, because opportunities for face-to-face transmission were greatly reduced in open air, and it was expected that such outbreaks would end spontaneously because of breaks in the chain of transmission. This was usually the case, yet in ten outbreaks among nomads in Somalia a different pattern was detected. For instance, there were 14 cases in one nomad group of 55 people in 1977, and six months elapsed between the first case and the last. It was evident that transmission could continue for many weeks, even in very small groups.³⁸

In conditions where transmission of infection was uninterrupted, the *variola* virus usually survived easily by passing from person to person. However, infection did not persist or recur in its human hosts, so the virus was able to survive indefinitely only in populations of a certain size. Smallpox, which spread less rapidly than measles, could become endemic in populations of about 200 000, which produced enough new susceptible individuals to maintain continuous chains of infection. But in small populations on islands or in geographically remote regions, it died out. Then if fresh introductions occurred, smallpox would take root again when there were enough unexposed children or immigrants to supply the virus with the new susceptible hosts it needed to survive.³⁹

The first way to reduce extreme susceptibility to the virus, and to check its continued spread in large human populations where smallpox was endemic, was the practice of variolation, which had been used for centuries in some Asian countries. Variolation was the deliberate inoculation of active *variola* virus taken from smallpox sufferers into the skin of susceptible people, causing a mild attack of it, which induced immunity. The symptoms that followed cutaneous inoculation differed from the usual symptoms, and it is still not clear why variolation caused such mild illness compared with naturally acquired smallpox.

Smallpox was well known in western Europe in the eighteenth century, and was completely uncontrolled until variolation was introduced in Great Britain in 1721, probably by the pockmarked

Lady Mary Wortley Montagu after a visit to Turkey. The practice became popular, and was increasingly common in Britain. Until the end of the eighteenth century it was the only way of avoiding death or the disfiguring scars an attack would cause. If smallpox threatened, the use of variolous matter to inoculate susceptible people was a routine precaution. The skilful inoculation of healthy young people reduced the severity of the disease and the mortality caused. However, occasionally inoculated people developed a generalized rash, and were infectious, and transmitted smallpox in the usual way to contacts who got the normal severe disease, as European experience and recent practices in Afghanistan and Ethiopia demonstrated.⁴⁰

The safer modern practice of vaccination replaced it at the beginning of the nineteenth century. The amazing discovery that cowpox did indeed protect people against smallpox was established by Jenner in 1798. He described the security against smallpox that inoculation with cowpox provided, and predicted that ‘the annihilation of the Small Pox, the most dreadful scourge of the human species, must be the final result of this practice’. He was indefatigable in promoting his discovery, vaccination (as Pasteur later described preventive inoculation) spread quickly and widely, and European enthusiasm knew no bounds. By 1804 the Royal Jennerian Society had sent vaccine to New South Wales, and children were immediately vaccinated.⁴¹

Failures were not unusual in the early history of vaccination. Even in Europe there was not always enough vaccine for the vaccination of many people. It became necessary to increase the stocks of vaccine, to store it and transport it. Threads were impregnated with it, it was spread on glass slides, dried, and kept in glass tubes. Different sorts of vaccine were used, and cowpox virus was gradually replaced by *vaccinia* virus during the nineteenth century. Like cowpox virus, it became inactive when the temperature and humidity were excessively high, and in sunlight.⁴² It proved difficult to maintain a reliable supply of potent vaccine in Australia.

Jenner, who died in 1823, had not yet acknowledged the one serious limitation to the success of vaccination. Sometimes smallpox occurred in previously vaccinated individuals. It was milder, and hard to diagnose. It was eventually realized that immunity to small-

pox conferred by vaccination was neither absolute nor lifelong. The solution was revaccination, which was first recognized in continental Europe, especially in Germany, and revaccination was effectively practised there in the later nineteenth century.⁴³

Much older ways of controlling smallpox were known in Africa, Asia and Europe. In some communities infectious sufferers were isolated, which was an inefficient way of controlling it where it was endemic, unless used together with vaccination. In sparsely settled countries, where it was never endemic, including New Zealand and Australia, quarantine was used to exclude smallpox from port cities in the nineteenth and twentieth centuries.⁴⁴

The biological catastrophes of the Old World may seem irrelevant in Australia today, although some are famous. When Thucydides described a plague in Athens in 430 BC, he recalled the victims' terrible despair, and 'many houses in which all the inhabitants perished through lack of any attention'. Boccaccio described the Black Death of 1346–52, when 'there was no medicine for the disease superior or equal in efficacy to flight', and a recent generation of historians suggested it killed one-third of the total population of late medieval Europe. American historian William H. McNeill concluded that the impact of virgin soil epidemics of diseases, which made history in Europe, has been consistently underestimated in the New World. We will find that except when vaccination became available in parts of Aboriginal Australia, virgin soil epidemics illustrated the combined and simultaneous effects of all the factors that exacerbated the severity of smallpox.⁴⁵

The history of smallpox is central to world history beyond the Eurasian landmass as well as within it. Historians and medical scientists have now recognized its role in European expansion, the colonization of the New World, and the confrontations of Europeans with those they conquered. The seemingly endless destruction of Native American populations began early in the sixteenth century, when introductions in the Caribbean islands by Spaniards and their African slaves, many of whom came from places in Europe and Africa where smallpox was endemic, caused virgin soil epidemics that almost eliminated Native American peoples, who were soon replaced by immigrants. Destruction continued on the mainland in larger urban and horticultural populations in Spanish and Portuguese

Central and South America, while their previous exposure and immunity protected most newcomers. The Europeans' restless travelling and exploration ensured the *variola* virus had access to a new and large supply of susceptible Native American hosts, and the toll of Hispaniola was repeated in Mexico, Central America, Peru and Chile, while the Portuguese and their slaves introduced it in Brazil. In the Aztec and Inca empires, the size of urban and horticultural populations enabled some Native American peoples to survive.⁴⁶

Henry Dobyns recently suggested that after the first introduction in the sixteenth century, smallpox was introduced on more than three dozen occasions before 1900.⁴⁷ Virgin soil epidemics occurred in less densely populated regions on the North American continent, and in so far as the history of smallpox in the New World has relevance for the Aboriginal Australian experience, it is the history of smallpox in North America that best bears comparison. It was introduced as a new disease to previously unexposed North American indigenous populations as a result of seventeenth-century European expansion, often before American Indians had any significant contact with newcomers, and for several centuries it was periodically reintroduced to infrequently exposed populations. The size of populations distributed over the vast continent remains hypothetical. Conservative estimates of the entire population north of Mexico before 1600 vary from 500 000 to over 2 000 000 Native Americans.⁴⁸ Many nomadic groups of hunters, fishers and collectors migrated over large tracts of land in the northern half of the continent, and semi-settled Native American societies developed in more favourable regions. Evidence of literate observers who recognized smallpox when it was introduced is abundant. Their reports of the way it spread among Native Americans, its incidence and mortalities, were widely known.⁴⁹ There is more than enough historical evidence of smallpox in Native American tribes in the seventeenth, eighteenth and nineteenth centuries to perplex modern scholars.

At least one virgin soil epidemic occurred among Native Americans in New England before the Pilgrim Fathers arrived. There was a plague at Massachusetts Bay in 1617, perhaps introduced by shipwrecked French sailors, which lasted until 1619. When Thomas Morton travelled through the Massachusetts forest five years later, 'the bones and skulls . . . made such a spectacle . . . it seemed to mee

a new found Golgotha'.⁵⁰ The diagnosis of the disease that caused the disaster has been questioned: was it bubonic plague, or was it smallpox? Present opinion favours smallpox. It is said to have killed at least one-third of local Native Americans, with mortality as high as 100 per cent in some localities. When a second plague was seen in New England in 1633, it was accepted that smallpox was the cause. Wherever it reappeared in the north-east in following years, observers described the terror of Native Americans, their dismay, their dejection, and always their many deaths.⁵¹

The European and African origins of Native American smallpox are well known. There were shipboard outbreaks and smallpox deaths on the Atlantic crossing in the seventeenth century, and many introductions by settlers and slaves in the ports of North America, with drastic effects on susceptible young colonials as well as Native Americans. There was no effective maritime quarantine, and within the colony quarantine was inadequate. Smallpox was completely uncontrolled in western Europe before the eighteenth century, and epidemics continued as long as trans-Atlantic and West Indian shipping brought smallpox with settlers and slaves to growing urban populations in the port cities. Variolation was not widely practised among Europeans in North America before the mid-eighteenth century, and evidently it was not practised among Native Americans at all.

During the eighteenth century, when Native Americans were generally more vulnerable to smallpox than susceptible colonists were, some Europeans deliberately initiated outbreaks. E. W. and A. E. Stearn said the French, Spaniards, English, then the Americans all wilfully started smallpox epidemics, and described their probable use by military personnel during the Native American wars. 'It would be fully as good as an army', observed one officer who hoped smallpox would break out and spread among rebel tribes in the 1750s. In 1763 the commander-in-chief of the British forces suggested to subordinates that blankets infected with smallpox could be given to disaffected Native Americans, and an officer obeyed him with two blankets and a handkerchief from a smallpox hospital.⁵² In nineteenth-century Europe, the incidence of smallpox changed with vaccination, but there were enough infectious immigrants and slaves arriving on the east coast to start epidemics, and some introductions on the west coast as well. However, smallpox did not become

endemic, and virgin soil outbreaks still occurred periodically in Native American populations.

There is rough but consistent evidence of massive mortalities, and the elimination of from one-half to two-thirds of Native American populations affected by smallpox epidemics has not been questioned by any one of a number of medical historians. North of Lake Ontario, smallpox might have reduced the Hurons by half before it moved inland, and Hopkins thinks that by the end of the seventeenth century it had proved fatal to many of the Native American populations in the eastern third of the continent. In the eighteenth-century South, it attacked the Cherokees, reportedly killing one-half of them. At the beginning of the nineteenth century there were severe outbreaks on the Missouri and Columbia rivers, and elsewhere in the west. The Omaha lost two-thirds of their number in 1801–2. There were said to be 3000 deaths among Pawnees in 1832, and 2000 more Pawnees were said to have perished six years later in one of many outbreaks on the western frontier.⁵³ On the Missouri in North Dakota, smallpox attacked the Mandan twice between 1800 and 1840, and it was said that only 27 of several thousand survived. In 1838 it was claimed that 60 000 western Native Americans had recently died of it. The defence of the frontier became unnecessary, and smallpox was expected to cross the Rocky Mountains.

Accounts of smallpox beyond the northern and western frontiers accumulated. In subarctic country west of Hudson's Bay, smallpox destroyed many hunters and fishermen in 1781–82, and when it returned in 1819 'whole bands' were lost. North-west tribes lost from one-third to one-half of their numbers in the affected area. Destructive outbreaks west of the Rocky Mountains in British Columbia in the 1780s affected coastal tribes in Alaska. Tlingit salmon fishermen in the north-west are thought to have suffered heavy losses in 1835, when about 3000 of them died after refusing vaccination. In 1838–39, Eskimos in western Alaska were attacked and perhaps half of them died.⁵⁴ In California there were far-flung outbreaks among Native Americans in the late eighteenth and nineteenth centuries.

There were various reasons for the great number of deaths among Native Americans. The appalling nature of the disease itself was responsible in the first place, and Hopkins considers that in

virgin Native American populations, smallpox 'often assumed one of its deadly haemorrhagic or confluent forms', which increased mortalities. The extreme susceptibility of Native Americans to infection seemed unusual. In Brazil, the Portuguese said Native American slaves suffered more severely than Negro slaves.⁵⁵ Even after a century or more of contact with it, the susceptibility of many Native Americans to smallpox had not diminished. The Hopi suffered epidemics in 1781, 1853 and the late 1860s. According to the Annual Report of the Commissioner of Indian Affairs in 1899, when smallpox occurred again in 1898, the case-fatality rate was 74 per cent among traditional Hopis, compared with 10 per cent among those who accepted Western medical care.⁵⁶

Using modern evidence of other diseases to interpret smallpox evidence, Alfred W. Crosby depicted the simultaneous sickness of susceptible people, the loss of the will and the means to survive, the ensuing economic crisis, and the abysmal end of sick Native Americans without water, food, fire, or any way of burying their dead.⁵⁷ He drew attention to age incidence in virgin soil epidemics, remarking on the destruction of the very young, and the loss of productive adults responsible for food, defence and procreation. Crosby also attributed mortality to Native Americans' reactions to smallpox. They had no idea of contagion, so the sick and the well were not separated, and close contact caused the frequent transmission of infection. Terror and frenzy were obvious during outbreaks. Many died soon after becoming sick, before the smallpox rash developed, and suicide was a frequent reaction to the first signs of disease. If sufferers sought relief through the practices of traditional medicine, the effects of smallpox were heightened when feverish victims plunged into cold water. Fear and fatalism stopped family and friends from providing drink, food and shelter, and the sick were often abandoned and died of neglect. When the healthy took flight, some might have escaped infection, but others were incubating the virus and took it with them. This may have been the main reason for the appearance of smallpox in some regions.

The demographic impact of smallpox among Native Americans still startles scholars. In his recent enquiry, Wood used a wide range of historical evidence to estimate the population of the American South in the century before the revolution, and found that in the

entire South below Virginia between the 1680s and 1730 ‘The Indian population . . . had dropped catastrophically, from 197,000 to fewer than 68,000 in less than 50 years’. In his opinion there is no reason to dispute contemporaries’ seemingly arbitrary estimates of the inroads of smallpox. In a radical reappraisal of pre-Columbian population estimates, Henry Dobyns acknowledged other and better-known causes of the Indian demise. However, using colonial enumerations of historic populations, and allowing for many epidemic episodes, he proposed a population of 4 500 000 or 5 000 000 non-horticultural Native Americans in North America north of Mexico early in the sixteenth century—twice as many people as earlier estimates had suggested. Recent estimates of the demographic effects of new diseases on Native American populations, especially smallpox, are consistent with McNeill’s opinion, that long-term demographic consequences affected five or six generations after populations were first exposed to the *variola* virus in Mexico and Peru.⁵⁸

As McNeill said, difference in resistance between genetically and immunologically naive Native American populations, and newcomers from the Old World, who were often immune and at least knew about smallpox, affected North American history greatly. Virgin soil epidemics among Native Americans, which were mostly accidental and often inexplicable, were the main reason why Europeans arriving in North America could settle the continent relatively easily, and smallpox even seemed to justify the god-fearing Europeans’ occupation. The new arrivals believed they were biologically superior to the indigenous people, and the conviction was persistent. The impending extinction of depleted and demoralized Native Americans seemed inevitable. Treaties and land rights were the token gestures of a minority.

Recent medical literature informs the history of smallpox in both North America and Aboriginal Australia, and there are similarities in the impact of smallpox in the previously unexposed nomad populations. In both cases, virgin soil outbreaks of the new disease coincided with the arrival of European settlers, occurred before the original inhabitants had sustained contact with them, and on both sides of the frontiers. In both cases, pandemics occurred before Europeans had controlled the disease effectively, and long before

they were able to use Jenner's solution to mitigate its effects in indigenous populations on the formidable, vast, and largely unknown continents. There are, of course, important differences in the history of smallpox in Australia and North America. Australia had smaller populations, introductions were less frequent and epidemics fewer. One significant variation was the world scenario of each continent. The isolation of the island-continent of North America from Eurasia was almost complete until Europeans crossed the Atlantic in the fifteenth century. But Australia is the largest of Eurasia's south-east offshore islands, and Europeans were not the only voyagers to reach the continent during Aboriginal Australia's century of epidemics.

In the decade following the eradication of smallpox, questions about its significance have persisted. In a country where its past history in Aboriginals is little known, Fenner's recent comments about its importance are of special interest to Australians. There is no foundation for the view, put forward in North America but uncommon here, that *variola* virus was evolving towards avirulence. It now seems certain there were numbers of strains of differing virulence, both of *variola major* and *variola minor*, which could have affected the relative severity of Aboriginal and European epidemics. Surviving evidence of Aboriginal smallpox suggests that the strain, or strains, of smallpox, introduced by travellers to the north coast were severe Asian strains. Fenner reminds us of what happened to Native Americans five centuries ago, when smallpox was imported with African slaves, and killed more than half of the population, while survivors, left without families, food, or anything else, died of starvation. The Australian history of this highly lethal disease is the most recent chapter of a history that began in India and China thousands of years ago, continued in England hundreds of years ago, then unexpectedly coincided with the arrival of the English here in the 1780s.⁵⁹

3

Myths

Of the diseases that occurred among Aboriginal Australians in the first century of British occupation, smallpox caused the greatest damage. It has also caused the most confusion among scholars in smallpox-free Australia in the second half of the twentieth century. Interest in Aboriginal smallpox revived during the smallpox eradication campaign, and continued after *Smallpox and its Eradication* was published. There was still no evidence of the origin of its introduction on the east coast in 1789, 1830 or 1860, and old questions about whether the Aboriginals' disease really was smallpox were raised, while speculation about its source continued unabated. Today, using research undertaken in the smallpox eradication campaign, we can discard misleading theories about what the disease was and where it came from, because we are now in a better position to interpret the records.

Chickenpox or smallpox in the colony of New South Wales?

The disease most frequently confused with smallpox in suspected but unconfirmed cases was chickenpox,¹ and although the suggestion that chickenpox was the disease that killed and scarred Aboriginals in 1789 and 1830 was always contrary to mainstream medical

opinion, new knowledge added interest to it for a time. It is now known that chickenpox (*varicella*) and shingles (*herpes zoster*) are caused by the same virus. After causing the acute infectious disease of childhood, the *varicella-zoster* virus persists in nerve ganglia for years. The latent virus can be reactivated, causing the lesions of *herpes zoster*. If the infection is then transmitted to susceptible children, they in turn suffer from chickenpox. In this way, the *varicella-zoster* virus passes from generation to generation, and survives in very small populations of perhaps only one hundred people without a new source of infection. For this reason, unlike measles and smallpox, it can survive in nomad societies, and is prevalent in some isolated pre-agricultural societies today.²

When Peter Moodie described epidemics of chickenpox in modern Aboriginal communities in 1973, he said that, unlike measles, it had not caused serious sickness. Chickenpox is a mild disease, and Moodie's opinion is consistent with more recent evidence of it elsewhere. Research during the smallpox eradication campaign clearly demonstrated that chickenpox has never killed or scarred its victims in the way smallpox did. In one previously unexposed population in Kerala, India, only the elderly suffered particularly severely from chickenpox, and among modern nomads in Somalia, less than 3 per cent of those seen after attacks of it were scarred. Consequently, it is surprising to find chickenpox in a list of principal recorded killers in the Australian chapter of Diamond's remarkable world history, *Guns, Germs and Steel*.³

Chickenpox was possibly previously known to Aboriginals, and Europeans may have introduced it as a new disease in Australia. The latent virus travelled to Australia with most adults after 1788, and chickenpox could have occurred at any time when susceptible children or others were infected by an adult who had shingles, as well as in the more usual way by a child with chickenpox. One of the most infectious of childhood diseases, chickenpox is easily transmitted by contact between children.⁴ While there is no record of chickenpox in 1789, there were probably outbreaks in the children of 5000 or so Europeans who had arrived in the colony by 1800, and perhaps in Aboriginals also. It was certainly present in both races in the settled districts in the nineteenth century. In 1804 the Principal Surgeon, Thomas Jamison, noticed it among Europeans,

and in 1831 John Mair saw unequivocal cases. The same year, James Bowman, Inspector of Colonial Hospitals, said: 'This disease has long prevailed in all parts of the Colony, both among the white and Black Population, and [is] known by the name of Native Pock'.⁵

Smallpox and chickenpox were easily confused when they were present at the same time, and this happened in newly settled districts in the colony of New South Wales in the 1830s. A generation after Jenner, the effects of smallpox were attributed to chickenpox, or in colloquial terms 'native pock', by younger settlers who were no longer familiar with smallpox. Bowman in Sydney and his assistant at Windsor did not see active smallpox in Aboriginals, and in 1831 they assumed that chickenpox was the only eruptive disease among them. That year, George Busby, a young British medical graduate practising at Bathurst, saw several sick Aboriginals, and did not recognize their disease. In a letter to Bowman, his superior in the colonial medical service, he described the symptoms of smallpox in Edward Titman, a surveyor who had been living with them, but diagnosed his disease as chickenpox. Busby's letter about the surveyor with 'chickenpox', who was treated at Bathurst Hospital, was published in 1870 after his death, when interest in the Aboriginal disease had revived. Cumpston republished the letter in 1914, and it may have confused modern readers.⁶

The most persistent modern challenge to the interpretation of smallpox as the disease responsible for massive mortalities in Aboriginals in the colony of New South Wales, and described in contemporary records, was New Zealand author C. W. Dixon's hypothesis that it was not smallpox at all, but 'it could have been chickenpox introduced as an entirely new infection to a completely susceptible population living under very primitive conditions'. He published this in a monograph on smallpox in 1962, when his account of Aboriginal epidemics was limited by a lack of relevant sources. In 1986 American historian A. W. Crosby expressed similar doubts, and speculating uncomfortably about Sydney in 1789, he said: 'perhaps the native Australians, who had been in isolation for thousands of years, lacked any and all immunological defenses to some infection so minor among the Europeans that the settlers never noticed it themselves'.⁷ Dixon's and Crosby's opinions have proved attractive to

some Australian authors. The idea that chickenpox was the disease seen at Sydney in 1789 was explored by James Urry in 1979, Peter Curson in 1985, First Fleet historian Alan Frost in 1994, and surfaced in *Frontier* on ABC television on 5 March 1997, when Henry Reynolds claimed that epidemiologists support the hypothesis.⁸

Not only is it most unlikely that any of the few susceptible white children at Sydney early in 1789 happened to have chickenpox and infect Aboriginals with it, but in First Fleet letters and journals, there was not one dissenting voice. No one even mentioned chickenpox and, without exception, contemporaries who knew smallpox said it was the cause of extensive outbreaks in Aboriginals in the Sydney district. Lay people and professional observers were all too familiar with the disease, which was endemic in Britain at the time, before vaccination altered its prevalence and appearance after Jenner's discovery in 1796. Differences in the records are trivial. When John White and other surgeons examined the victims in April 1789, they found that 'pustules, similar to those occasioned by the small pox, were thickly spread on the bodies'. For the next forty years, members of the colonial establishment, newcomers and visitors, described the scars of 1789 and heard Aboriginal accounts of smallpox. Its effects were still apparent in 1829, when two years after he was appointed as assistant surgeon in the 39th Regiment at Sydney, John Mair said: 'Marks are found upon the Aborigines nearly resembling those left by Small-Pox'.⁹ He offered to vaccinate both colonists and Aboriginals.

Although younger settlers, such as Busby, did not recognize smallpox when they saw it in 1831, those who had seen it before emigrating, such as Imlay and Mair, could recognize both old and more recently acquired pockmarks in Aboriginal victims. After investigating the outbreak in the newly settled districts in 1831, Mair concluded that it really was smallpox, and prepared a twenty-five-page report for the Governor. The report was evidently made available to George Bennett, medical practitioner and naturalist, who visited Australia in 1832, and included some of Mair's findings in *Wanderings in New South Wales*, which he published in London in 1834.¹⁰ Mair's unpublished report is central to the retrospective diagnosis of Aboriginal smallpox. On seeing Mair's report, Fenner said: 'I have not the slightest doubt that the disease described by

Mair was smallpox . . . there are so many features that are characteristic of smallpox and of no other disease'.¹¹

Impetigo or smallpox in the Port Phillip District?

The incidence of smallpox in country south of the Murray River is more difficult to establish, because in 1830 there were no European settlers in the area. In subsequent years there were few well-informed observers to report it when it occurred. But by 1845 several medical practitioners in the Port Phillip District had seen people with pockmarks, which they attributed to outbreaks about 1789. Years later, older settlers still remembered very old pockmarked Aboriginals. Curr's informant, Dr James King, who had been a member of the Medical Board of Victoria for twenty-four years, seven of which he had spent at Swan Hill on the Murray River, had heard of the ravages of smallpox from Aboriginals he had treated. Some were pitted by 'confluent' smallpox after an outbreak some seventy years earlier. He was sure that: 'there is no disease among the Blacks which leaves marks which can be mistaken for small-pox, *nor any pock peculiar to the Blacks*; and what has been called native pock is mere chicken-pox, which is more common among White than Black children'.¹² Other reports later in the century refer to survivors of an outbreak in 1830.

In a discussion about Noel Butlin's references to pockmarked Aboriginals, Diane Barwick suggested in 1984 that 'remembered pockmarks' described in the *Argus* in 1877 were caused not by smallpox, but by 'native pock', which she said was a colloquial expression for *impetigo contagiosa*, a common minor bacterial infection. However, Cumpston, who discussed 'native pock' and impetigo in 1914, said smallpox was probably present in Victoria as well as New South Wales and South Australia in 1830.¹³ Writing before *Smallpox and its Eradication* was published, Barwick was not aware that blemishes left by impetigo and other skin diseases are unlike pockmarks, or that evidence of pockmarks from experienced lay observers was used during the eradication campaign to establish the past presence of smallpox. Her earlier work, which referred to smallpox in 1789 and 1830 as a cause of depopulation in Victoria, was vindicated.

Naturally there were differences in the recollections of Victorians of different ages and from different districts. We will see later that a number of settlers did contribute reliable evidence to the *Argus* debate in 1877, but evidence from a few others, notably the historian G. W. Rusden, was unreliable. Smallpox occurred less often after vaccination was practised, and Rusden may not have seen it before he emigrated in 1834. In the *Argus* and in his *History of Australia* in 1883, he said he had seen ‘scores of natives pitted with what was called in the bush “native-pock”’, which was easily mistaken for smallpox. As a pastoralist near Gundagai and on the Lachlan and Goulburn rivers in the 1840s, he would certainly have seen survivors of smallpox, and his comment about people *pitted* with native pock did not refer to marks left by chickenpox or impetigo. He probably saw pockmarks without knowing what they were. Curr, who had seen pockmarks, disagreed with him about ‘native pock’, and Rusden’s *History* is now considered inaccurate and untrustworthy.¹⁴ In the circumstances, the interpretation of records of smallpox in Victoria has been uncertain. Historian Jan Critchett did find evidence of smallpox in Victoria, but quoted Barwick’s discussion of impetigo as an alternative explanation.¹⁵ Barwick mentioned smallpox in North America, but neither she nor Critchett acknowledged it behaved similarly in Victoria.

Smallpox in South Australia, the Northern Territory and Western Australia

In land that became the colony of South Australia in 1836, there were no literate observers to leave records of Aboriginal smallpox in the 1780s or 1830, but reports of its past presence, including oral history from Aboriginal people, followed later and continued into the twentieth century. On the lower Murray, missionary George Taplin saw impetigo among local Aboriginals, which lessened when he obtained a better supply of soap.¹⁶ British and German settlers knew that smallpox, not impetigo, was the disease that had scarred and killed the original inhabitants of South Australia.

In the Northern Territory early in the twentieth century, Herbert Basedow found that the scars of *acne tropica* were mistaken for pockmarks. He could readily tell the difference himself: ‘I have

myself seen a number of individuals in the Port Darwin district whose faces bore genuine and typical pock-marks'.¹⁷ Common minor ailments do not account for mortality and facial scarring attributed to smallpox by both Aboriginals and newcomers for over a century, and the prejudiced term 'native pock' must go, because it hid a fearful biological disaster.

Unlike the situation in eastern Australia, when smallpox occurred in Western Australia in the 1860s, a number of experienced lay people and professional observers observed the epidemic over a longer period of time as it spread over greater distances. There were a few colonist cases in outlying districts, where most settlers recognized the dangerous and unfamiliar disease for what it was, and both races were vaccinated when it was possible. No other disease was even considered as the cause of the deaths among Aboriginals at the time or the pockmarks seen later. Aboriginals in the Broome district knew smallpox as *walmbing*, the same word they used for chickenpox,¹⁸ but there are no records to support Dixon's hypothesis that the disease of the 1860s might have been chickenpox. Recent medical literature confirms that smallpox caused the lethal, disfiguring disease in indigenous Western Australians.

A European origin of Aboriginal smallpox?

In 1789 new arrivals at Sydney thought the smallpox epidemic inexplicable, and found nothing to suggest its origin was European. The only ship to arrive after the First Fleet left in July 1788, until the *Sirius* returned from the Cape during the epidemic in May 1789, was the *Supply* from Norfolk Island, where there was no smallpox. After he left the country in December 1791 Captain Watkin Tench of the Marines reviewed the possible origins of the disease 'for the ingenuity of others to exercise itself upon':

Is it a disease indigenous to the country? Did the French ships under Monsieur de Peyrouse introduce it? let it be remembered that they had now been departed more than a year; and we had never heard of its existence on board of them.—Had it travelled across the continent from its western shore, where Dampier and other European voyagers had formerly landed?—Was it introduced by Mr Cook?—Did we give

birth to it here? No person among us had been afflicted with the disorder since we had quitted the Cape of Good Hope, seventeen months before. It is true, that our surgeons had brought out variolous matter in bottles; but to infer that it was produced from this cause were a supposition so wild as to be unworthy of consideration.¹⁹

By the 1820s the origin of Aboriginal smallpox in 1789 had not been confirmed, and a younger generation of settlers added only rumours to records left by members of the First and Second fleets. William Charles Wentworth, son of Dr D'Arcy Wentworth, arrived with his parents at Sydney on 28 June 1790, and spent his early years with his parents on Norfolk Island. He subsequently spent much of his life in England and France, obtaining an education and professional qualifications. Wentworth crossed the Blue Mountains with Blaxland and Lawson in 1813, and became a landowner before he published *A Statistical, Historical and Political Description of the Colony of New South Wales* in 1819. He had seen pockmarked Aborigines, but could only speculate about the origin of their disease. In the first edition of his work, which was popular in the colony and among prospective immigrants abroad, he said: 'Some few years, indeed, before the foundation of this colony, the small pox committed the most dreadful ravages among the aborigines. This exterminating scourge is said to have been introduced by Captain Cook.'²⁰ Wentworth's version of Aboriginal smallpox was widely read.

On his voyage to the Antarctic, Russian visitor Captain Bellingshausen called at Sydney in 1820, and asserted that intercourse with Europeans had spread diseases among New Hollanders: 'small-pox, brought by Europeans, has also produced the most terrible results and has considerably reduced the native population. We were told that in the caves near Broken Bay one could see the bones of numbers of people who had died there, far from all help.' He added that consumption and dysentery were very prevalent in New Holland.²¹

Another visitor who had read Wentworth's account was the French traveller R. P. Lesson. Naturalist and surgeon in the French corvette *La Coquille*, he was at Port Jackson from 17 January to 20 March 1824. In his account of the voyage, he described seeing

old Aboriginals with pockmarks but blamed Cook not La Pérouse.²² In a third edition of his work published in 1824, and some time after Lesson's visit, Wentworth repeated his claim that the disease was brought by La Pérouse.²³

Fifty years after Wentworth's publications, E. M. Curr published another explanation of Aboriginal smallpox. He believed that smallpox was introduced 'by ourselves' in 1789, through British passengers in the First Fleet. He mentioned a Portsmouth doctor's remark about a 'malignant' disease on the *Alexander* transport, and claimed Aboriginals were infected when they obtained clothes worn by someone who had smallpox. Curr continued that it swept back and forth across the whole continent south of the tropic of Capricorn at least twice, and died out in 1845. However, reports of sickness and deaths on the *Alexander* transport refer to various causes other than smallpox, and First Fleet historian Alan Frost has confirmed that smallpox did not occur on the voyage. Curr knew of Mair, but it is unlikely that he had seen his official report, and he did not have evidence to support his own statement that smallpox was continuously present somewhere in the country from 1788 to 1845.²⁴ Settlers saw active smallpox among Aboriginals only in 1789, and from 1829 until 1832, and modern knowledge of its epidemiology establishes that it would have been virtually impossible for smallpox to have been endemic from 1788 to 1845 in small populations, unevenly distributed over vast areas. Therefore, Curr's conclusions about Aboriginal smallpox are flawed, although evidence from his correspondents may be used to supplement other evidence that became available after his death.

Twentieth century myths

In 1968 the *Australian Encyclopaedia* said that in 1789 Aboriginal smallpox might have come from either Sydney Cove or Asian contacts on the north-west coast. Its origin remained an open question until 1983, when economic historian Noel Butlin published an innovative demographic study of Aboriginal depopulation in south-eastern Australia. He discounted the possibility of northern introductions of smallpox as the cause of epidemics that depopulated the south-east in 1789 and 1829. His argument against northern

introductions depended on doubts about compelling evidence, from Macknight, of 'deaths on the passage' among Macassan fishermen on the north coast (see chapter 4). He considered it unlikely that they transmitted smallpox to Australians in the wet season, because it limited local contacts. But some infectious contacts often persisted through the rainy season in monsoon countries. Australia was no exception. Life in the trepang season ensured close relations between Aboriginals and Macassans, and infection of naive Australians was inevitable. Nevertheless, Butlin emphasized only the 'low probability' of introductions of smallpox by Macassans and its spread into the south-east.²⁵ In 1983 he did not recognize that Aboriginals were 'great travellers', who spread infection over long distances: he had no explanation for widespread outbreaks, other than infection in some way by Europeans. His hypotheses about British introductions of smallpox in the south-east in 1789 and 1829 are mentioned below. Evidence of the spread of smallpox from monsoonal north Australia to the temperate south-east is presented in later chapters.

Variolous matter

In 1914 Cumpston could not dismiss the surgeons' variolous matter as a possible source of smallpox in Australia. His opinion was influential, and in the late twentieth century, when Australians rediscovered the country's Aboriginal past, the history of European indifference, violent dispossession and discrimination affected discussions of the origin of Aboriginal smallpox, which seemed to be the first major disaster of the white invasion. Some writers thought the smallpox epidemic seen in the Sydney district after Europeans arrived was caused by them. Alan Moorehead, who saw it as part of the fatal impact of Europeans in the south Pacific, attributed it to the arrival of the Europeans, and another author said infectious passengers had arrived on a British ship.

A comment likely to damage prospects of reconciliation in modern Australia was Noel Butlin's explicitly speculative and hypothetical statement that: 'Although the origins of the main killer, smallpox, are obscure, it is possible and, in 1789, likely that the infection of the aborigines was a deliberate exterminating act'. He suggested that the origin of smallpox in 1789 was 'variolous matter

in bottles', perhaps long-lasting smallpox scabs, brought by First Fleet surgeons. Inoculation with live virus matter was a common practice in Britain at the time and the deliberate or accidental infection of Aboriginals were 'logical possibilities'. Perhaps Phillip was 'pressured into action': American colonists had given blankets infected with smallpox to Native Americans. In 1985 Curson also favoured the variolous matter hypothesis.²⁶ The issue of Aboriginal smallpox re-emerged on Australia Day 1988 on ABC TV, when a reporter asked an indigenous Australian how he liked the Bicentenary events. He replied: 'You gave us smallpox'.

Tench was the only source of the circumstantial evidence that Butlin used when he suggested the deliberate or accidental infection of Aboriginals were possibilities. The surgeons did have a supply of variolous matter, because the only precaution they could have taken if smallpox threatened their charges on the passage in 1787 or after their arrival was inoculation with live virus matter to induce immunity. Writing after the eradication of smallpox, Butlin was aware that in the absence of active smallpox among Europeans at Sydney, variolous matter, or potentially infectious virus on fomites, were the only possible sources of infection. Smallpox virus was more heat resistant than most other viruses, and virus within scabs was better protected than that in oropharyngeal secretions. However, Butlin wrote five years before the World Health Organization published *Smallpox and its Eradication*. He did not know that investigations during the eradication campaign showed that even virus protected in scabs retained infectivity for only three weeks at temperatures of 35°C (95°F) and relative humidity of 65–68 per cent, and for very little longer even when temperatures and humidity were lower.²⁷

A voyage through the tropics would therefore affect the viability of the virus, and it was probably inactive soon after the arrival of the First Fleet at the height of the Sydney summer in 1788. Worgan recorded temperatures of 85–90°F in the early months, and the Europeans soon discovered that summer were hot, sultry and stormy. It was hot again in spring, with the temperature 81°F in October and 93°F in November. It was 102°F in December, and 112°F in January 1789. There was rain and hazy, cloudy, blustery weather, and Tench found the humid heat intolerable.²⁸ After using freshly available variolous matter in Britain's cool climate, the surgeons were not to

know that it would have been adversely affected by Sydney's summer weather. Tench obviously did not know that by April 1789 it was no longer a likely source of infection. In 1983 Butlin found his remark about it unduly significant.

The *Bussorah Merchant*

The second smallpox epidemic in south-eastern Australia was also attributed to contact with Europeans. One author thought smallpox subsided after 1789 and flared up about 1830. But the second epidemic came from a new source, not from the 1789 introduction, because the smallpox virus was never latent. Moorehead, who thought that smallpox and tuberculosis swept through the tribes whenever they came into contact with outlying farms,²⁹ did not acknowledge that smallpox was seen on the Darling in February 1829 during Sturt's expedition, over a year before there were any European cases. As we shall see, the only cases of smallpox in 1830 among colonists were a few unvaccinated children in newly settled districts, where there had been contact with infectious Aboriginals. Most settlers in the colony at that time were immune after previous attacks of smallpox, or after variolation or vaccination, so could not have infected Aboriginals. There are no records of Aboriginals infected by Europeans.

Noel Butlin also speculated about exotic sources of indigenous smallpox in 1829–31. In his opinion, a possible source of the disease observed by explorers on the Darling River in 1829 was a European ship that arrived in Australia after an outbreak of smallpox at sea, the first of many such occurrences. The ship was the convict ship *Bussorah Merchant*, which reached Sydney in July 1828, several months after the outbreak. On that occasion Governor Darling's prompt and thorough measures stopped infection spreading to people on shore.³⁰ However, Butlin speculated that: 'the time of arrival and the knowledge that its infective contents were landed together with the geography of the location to which its convicts were sent seem to qualify it as the prime suspect'. With no evidence to support his hypothesis, Butlin suggested infection originated in the possessions of convicts on the *Bussorah Merchant* who were assigned on the upper Hunter in late 1828, that it was then transmitted to

Aboriginals, and then moved up the Namoi to the Darling in 1829. Alternatively, he thought American whalers frequenting Bass Strait were potential sources of infection at mouth of the Murray, and went on to say that: 'American awareness of deliberate infection and the conflicts with blacks' meant that he could not rule out possible deliberate action.³¹

In the case of the *Bussorah Merchant*, Butlin discounted the official investigation, which established that the last of four cases of smallpox, an infant, was reported on 9 May 1828, over ten weeks before the ship reached Sydney, and that by 29 July 1828 the ship was quarantined, the isolation of its passengers on North Head was arranged, and contact with the colony's inhabitants was prohibited. The crew and the ship were cleaned, clothing and bedding were destroyed and timber fittings of berths burnt. Quarantine ended on 17 August, but the crew were still not allowed on shore. Even if there were infectious virus on fomites when the *Bussorah Merchant* arrived, it is unlikely that it survived the measures taken to get rid of it on the ship, on articles in use, and on passengers. Historian Jean Foley considers that these measures set an important precedent for routine quarantine at Port Jackson.³²

With regard to the possessions of convicts from the ship, indirect transmission of virus on fomites was possible, but infection almost always involved face-to-face contact of a susceptible person with a person who had a rash, and once it was introduced to a population, it spread through chains of person-to-person infection that occurred with close contact. As its history among Native Americans illustrates, smallpox could be transmitted by strings of personal contacts over surprisingly long distances.³³

However, in the case of convicts on the *Bussorah Merchant*, the continuing survival of infectious virus on fomites in their possession, its transmission to an Aboriginal living in the country, and subsequent infection of Darling River Aboriginals, cannot be substantiated. The precautions taken when the ship arrived were evidently effective, because no cases of smallpox were reported among convicts assigned to work in the country. The alternative suggestion about Bass Strait whalers is also no more than speculation. As we shall see, reliable contemporary records establish that smallpox spread east from the Darling River early in 1829 to the coast late in

1831, not west from Port Jackson in 1828, or north from Bass Strait and the mouth of the Murray River, as Butlin proposed. There is no more evidence of a European introduction of the second epidemic of Aboriginal smallpox than there was of the first.

Nevertheless, many scholars have continued to associate outbreaks of smallpox, as well as other Old World diseases in Australia, like those in the Americas, with colonization in the New World by Europeans. Indigenous populations were depleted in the massive migrations that followed the voyages of the great navigators. In the case of smallpox in North America, there is no need to question traditional accounts of its many European introductions on the east coast, either directly or through African slaves, and its remarkable spread across the continent. However, in the case of Aboriginal smallpox after 1788, none of the outbreaks was as closely related to the arrival of ships from Europe as outbreaks in the Americas were. Furthermore, as Tench suspected in 1789, and Mair realized in 1831, there was a different explanation of its presence in eastern Australia. In Mair's opinion: 'the disease has not arisen from any peculiar constitution of the air in this part of the world but has been communicated by contagion from the inhabitants of some other country: or . . . if the disease originated in this continent it most probably arose in a very distant part of it.'³⁴

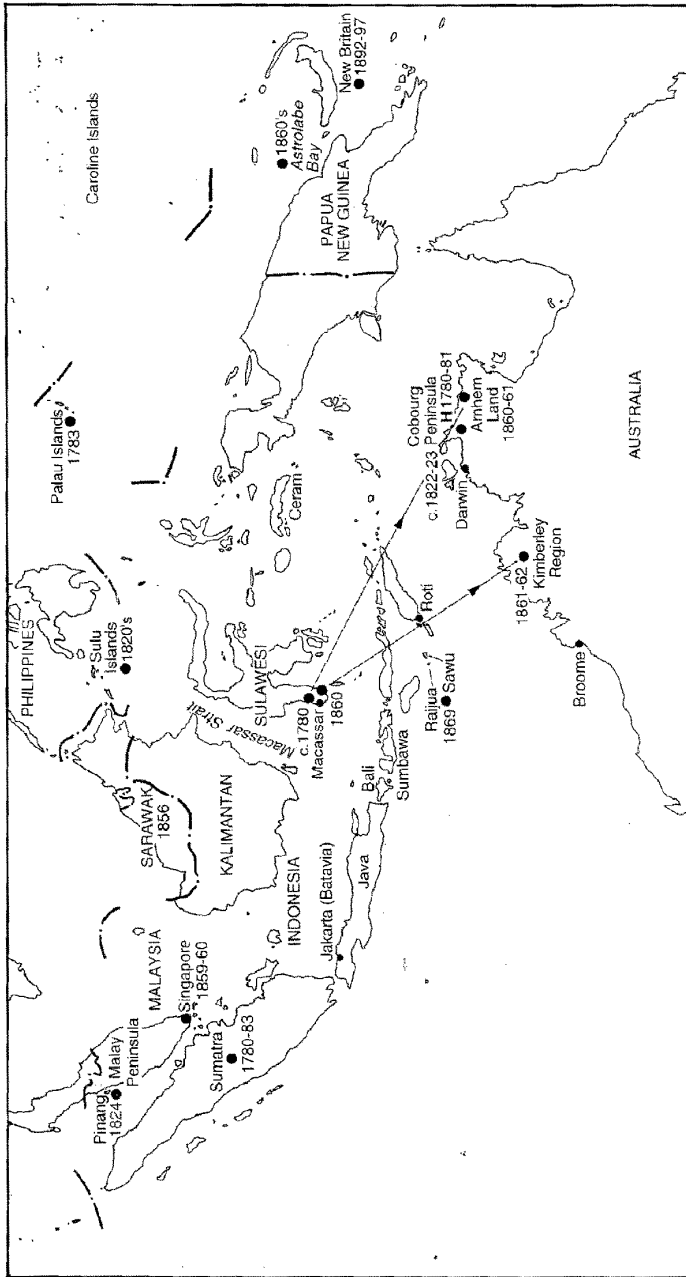
In the early years of settlement, newcomers in Western Australia realized that they had transmitted 'consumption' to Aboriginal people, but they also knew they had not transmitted smallpox to them, because Aboriginals were free of it until the *Saladin* brought it to Perth in 1893.³⁵ Aboriginal smallpox in Western Australia has provoked neither the curiosity nor the controversy that epidemics of it in eastern Australia caused, and its occurrence in the Northern Territory is also uncontroversial. Earlier opinions—that these outbreaks originated after contact with South-East Asians, before and after remote new tropical settlements were established by European settlers—have not been questioned. There are no myths about the identity or origin of Aboriginal smallpox in Western Australia or the Northern Territory in the 1860s.

4

The Indonesian Archipelago 1780–1880

In the absence of any reliable evidence that smallpox was introduced into the Aboriginal population of eastern Australia by the British in 1789 or between 1829 and 1831, other sources must be explored. The most likely, in view of the evidence of later introductions into northern and north-western Australia, is that it came from the islands of the Indonesian archipelago. In this chapter, we shall review the history of smallpox and its spread in the region, which was investigated before it was eradicated, and trace Australian connections with the archipelago.

The ancient presence of smallpox on the Eurasian continent is indisputable. It was in India and China at the beginning of the Christian era, and by AD 1000 it was entrenched in dense populations in river basins in India and China, which were fertile soil for endemic smallpox, so there were always people suffering from it in those countries. Early in the twentieth century, Europeans who visited Asia noticed that adults without pockmarks were unusual, and blindness caused by smallpox was common. India and adjacent countries remained the major focus of smallpox in the world until it was eradicated in the region in 1975, and various beliefs about smallpox had evolved over a long period. According to one persistent and curious Indian myth, smallpox pustules were caused by lentils or pulse the victim had eaten, and, as we shall see, some Aboriginal



The Indonesian Archipelago: smallpox outbreaks 1780–1870

Smallpox spread out from the Asian mainland to island south-east Asia in the late eighteenth century, and was endemic in dense populations on larger islands in the nineteenth century. Introductions into northern Australia may have followed outbreaks in the 1780s, 1820s and 1860s.

Australians had similar ideas. Supernatural beliefs about smallpox were common everywhere.¹

Neighbouring countries in mainland South-East Asia had close geographical, economic and cultural ties with India and China, and, by the nineteenth century, smallpox was endemic in larger populations in Burma, Siam and Indo-China. A European who lived in Indo-China for fifty years said that in the 1860s in Siam and Laos, 'hardly a household escaped, and many had no children left'. Towards the end of the nineteenth century, the French director of Saigon's Pasteur Institute was equally appalled by what he saw of smallpox among Laotians and Vietnamese, and he noticed its long-lasting depopulating effect. It also caused most cases of blindness in Indo-China at that time.²

Close connections between mainland Asia and the Indonesian archipelago, and occasionally infectious contacts with people from endemic centres of smallpox in Europe as well, ensured that smallpox spread eastwards to the islands in the last 500 years. Virgin soil epidemics followed introductions to island populations that had never been exposed to it before. Then, without enough susceptible subjects to maintain the disease indefinitely, and without more outside introductions, it would die out after explosive, distressing, but short-lived outbreaks. The better-known larger islands already had populations of 200 000 or more, which were large enough to maintain endemic smallpox. Epidemic exacerbations characteristic of it in very large populations occurred every few years in the most densely populated islands.³

Sumatra, the westernmost of the larger islands, is connected with the Malay Peninsula and neighbouring Java by constant boat traffic, which linked it to mainland centres of endemic smallpox. A historian of Sumatra described a disastrous epidemic in the early 1780s, and it has been said that by 1783 one-third of the Sumatran population had died of smallpox in the previous three years.⁴ The smaller and more crowded island of Java also had close connections with centres of endemic smallpox in mainland Asia. In the eighteenth and nineteenth centuries, and probably earlier, smallpox was a long-standing endemic infection in Java and Bali. Variolation was not practised extensively in mainland or island South-East Asia; in Java it did not affect the overall occurrence of smallpox significantly, and

vaccination was not effectively practised there until the second half of the nineteenth century. Smallpox spread more readily in Indonesia than in most parts of the world.⁵

Five hundred kilometres across the sea to the north-east, Kalimantan (Borneo), with an estimated population of 1 000 000 in 1800, was within easy reach for seafarers from centres of endemic smallpox in South-East Asia. Dyaks, Dusuns, Muruts, Malays and Chinese who inhabited the island might have suffered from smallpox whenever it was introduced, and early attempts to control it were unsuccessful. When unmistakable symptoms appeared, victims were abandoned, and it proved fatal in almost every case. The smallpox spirits killed indiscriminately; it was beyond the powers of medicine men; and religious ceremonies made no difference. Dyaks ‘fled their villages, and hid in the forest, never speaking above a whisper or using the name of the smallpox spirit, for fear of attracting its attention’. Some Queensland Aboriginals also spoke of smallpox only in whispers and with bated breath.⁶ An observer in the Sulu Islands in the early nineteenth century noted: ‘It is held in the greatest fear and dread . . . if it rages extensively . . . the people fly to the other islands. One-half of the whole that get the infection die with it.’⁷

Several hundred kilometres farther east, Sulawesi (Celebes) with four peninsulas and adjacent islands is distinguished by its mountainous character. ‘Nowhere in the archipelago have I seen such gorges, chasms and precipices as abound in the district of Maros’, wrote the naturalist A. R. Wallace in 1869 about the southern peninsula. Rugged terrain was interspersed with stretches of level ground, but most rivers were navigable for only a few miles. In the nineteenth century, there were four Bugis kingdoms, and the fifth kingdom, Macassar, had been under European domination for some time. The total population of the southern peninsula was then 1 000 000 or less. The towns were small: in 1838 the largest was the port of Macassar, with a population of 23 575. In 1910 one estimate of the population of Sulawesi and adjacent islands was 2 000 000.⁸

Available nineteenth-century records of smallpox in Sulawesi describe its presence where Europeans observed its results in coastal districts on the southern peninsula. In 1840, when the English soldier and traveller Sir James Brooke visited Palopo at the head of the Gulf

of Bone in the kingdom of Luwu, he found 'a miserable town, consisting of about 300 houses, scattered and dilapidated'; the mountainous country could not support a large population; 'wild tribes' peopled the hills and communication with other parts of the archipelago was limited. There had been struggles over the succession of the kingdom of Luwu in 1838, smallpox had followed, and was even more destructive than the civil war. A Dutch official recorded an outbreak on the island of Selayar, south of Sulawesi, in the 1860s, some twenty years before vaccination was available there.⁹

The eradication campaign in Sulawesi in the twentieth century showed that most outbreaks of smallpox occurred in coastal areas on the southern peninsula. A number of different dialects were still spoken in separate centres of population concentrated along the coast, which were often inaccessible because the country was so rugged. Many outbreaks undoubtedly terminated spontaneously, but sometimes infection persisted in isolated settlements. Epidemics were always more frequent in South Sulawesi, where there was a population of about 5 000 000 in the 1960s. Cases of smallpox were still being regularly reported there, when only a few were being reported in the other three provinces. In 1969 endemic smallpox was found only in South Sulawesi, and in 1970 and 1971 the island's last cases occurred in South Sulawesi.¹⁰

South-west of Sulawesi, Sumbawa in Nusa Tenggara had regular contact with Macassar. Smallpox was prevalent in 1847, and vaccination met with opposition. Farther east, on the very small islands of Sawu and Raijua, Dutch missionary Donselaar described destruction caused by smallpox in 1869. His account is hard to believe, except in the context of the damage the disease inflicted on isolated island populations elsewhere. The sudden shattering effect of smallpox was a measure of the islands' isolation. Donselaar estimated that over 12 300 died on Sawu, almost one-half or at least one-third of its population, in a few months. He subsequently saw traces of smallpox and numerous abandoned houses. Eye-witnesses told how at the height of the epidemic, entire households suffered, and when those still alive were too sick to help others, very sick victims died from lack of water. Some took flight and hid in caves, where groups of four or five were found dead. Raijua was also severely affected. About a hundred kilometres farther east is Roti,

the island immediately south of Timor and its port Kupang. Roti also suffered an epidemic of smallpox in 1869. However, according to the same population estimates, Roti lost only one-twelfth of its population. Lower mortality there was evidently a result of its inhabitants' immunity: some might have previously recovered from smallpox, others were probably vaccinated or variolated.¹¹

On the coast at Astrolabe Bay in New Guinea in 1872 the Russian scientist Nikolai Miklouho-Maclay discovered that smallpox was known there, and wrote in his diary that: 'Digu's face bore traces of smallpox. He explained to me that the illness came from the north-west and that many died of it. When it happened and whether it happened more than once, I was unable to find out.' A few months later, he found that smallpox had also left its mark on people who lived in the mountains behind Astrolabe Bay.¹²

In the 1890s there was at least one introduction of smallpox, probably several, in German New Guinea. In 1893 colonial officials reported that 'a particularly severe misfortune struck Kaiser Wilhelmsland when smallpox broke out in Stephansort in June of last year'. It was brought by a 'Malay' stoker on a German ship, attacked the Melanesians with great severity and spread in waves through the native villages. Vaccine was obtained from Batavia, and vaccination was carried out; nevertheless the disease claimed many victims. Years later, Governor Albert Hahl recollected the first outbreak in 1892, several months before vaccine arrived. In coastal districts he saw only 'the wretched remnant' of the former population. The survivors greeted the colonists with lamentations and showed them the mass graves of those claimed by the epidemic. Profound hostility between coast and mountain peoples cut contact between them and limited the spread of smallpox.¹³

Other small islands in Oceania north of New Guinea occasionally suffered epidemics when smallpox was introduced. There was an outbreak in the Palau Islands in 1783. Of the 5000 islanders in the Carolines, 2000 died in 1854. On Guam about 5000 people, one-third of the island's population, died of smallpox in 1856. Hawai'i escaped until it arrived from San Francisco in 1853 to kill 8 per cent of native Hawaiians. Fiji, unbelievably, escaped it altogether.¹⁴

The commercial activities of both indigenous populations and colonists enabled smallpox to spread through expanding and mobile

island populations after 1750, and it was always active somewhere in the Indonesian archipelago in the eighteenth and nineteenth centuries. It was a well-known menace on the close and crowded islands of Java and Bali, and any traders from those islands would have been immune after childhood exposure, so could not have infected Aboriginals. However, travellers from islands farther east in the Indonesian archipelago were less familiar with smallpox, most were susceptible and were inadequately protected by variolation or vaccination. Dutch colonists in the Netherlands East Indies could not maintain a supply of vaccine, and disturbing outbreaks occurred when smallpox visited larger islands more often. Seafarers from the eastern archipelago were victims of smallpox in their home ports or when they travelled, and the fishing-fleets were a major source of smallpox.

Campbell Macknight investigated the voyages of travellers from Macassar who collected marine harvests in the archipelago and regularly visited Australia. Sailors in fleets from Macassar were skilled navigators with highly mobile fast craft. They had trading contacts in old endemic centres of smallpox in India and China, and made long voyages to meet economic and financial obligations. When smallpox travelled with them, they were likely to transmit it to previously unexposed populations. They had made voyages to Australia increasingly often for nearly a century before British colonists, who called them 'Malays', saw numbers of them on the north coast in the 1820s. They sailed here in their praus on the north-west monsoon to collect trepang, or sea slugs, which was their most important harvest, and a highly valued resource in the archipelago. A delicacy and a reputed aphrodisiac in China, the slugs were abundant in shallow waters along the tropical Australian coast. This visiting labour force processed the trepang on the spot, and took it with them when they left the coast on the south-east monsoon at the end of the season. The Chinese market was rewarding, and Chinese merchants in Macassar exported Australian trepang to Canton and Amoy in south China.¹⁵

Using Dutch and British colonial records, Macknight enlarged the previously fragmentary history of the Arnhem Land visitors. The fishermen were more appropriately known as Macassans, a term used to describe trepang fishermen in fleets from Macassar. Most

were from South Sulawesi, but some travelled from islands as distant as New Guinea, and a few were indigenous Australians who visited Macassar. It is likely that Timorese, with a shorter voyage than Macassans, also visited Australia. The Macassans described their destination on the Arnhem Land coast as *Marege'*, and called the north-west coast *Kayu Jawa*. The high quality of trepang on the Kimberley coast was some compensation for less frequent and more dangerous voyages. Macknight thinks that explorations for new trepang grounds might have started in the 1720s, and that Macassan interest in Australian resources was well established by the mid-eighteenth century.¹⁶

For generations of fishermen who travelled in Macassan fleets, life changed when they assembled for the voyage. Groups of thirty or more men lived together on cramped Macassan praus, and crewmen were in close contact with each other during the passage to Australia, which took at least ten days. The fleet left Macassar with the monsoon between late November and mid-January. Sailing south past the island of Selayar, the praus made for Timor in their first week at sea. Sometimes they stopped there for water. They obtained equipment on the nearby island of Kisar. Continuing on their way, with or without these delays, landfall at Melville Island or the Cobourg Peninsula was about four more days' sail. They usually arrived in December or January, and within the incubation period of smallpox, after being at sea for ten to fifteen days. Most praus then spread eastwards along the coast, looking for trepang as far away as the Wellesley Islands in the Gulf of Carpentaria.

Estimates of the numbers of praus and their crews trepanging in northern Australia have been conservative. Macknight's estimate of between thirty and sixty praus annually on the Northern Territory–west Queensland coast in the first half of the nineteenth century suggests that between 1000 and 2000 men made the voyage to *Marege'* each year.¹⁷ If an unknown and changing influx of islanders from the north on the coast of Western Australia is added, it is likely that as many as 3000 islanders lived on the beaches of tropical Australia while the north-west monsoon was blowing. They were dispersed in small groups over hundreds of kilometres of the coastline. Crews of as many as half a dozen praus collaborated, making elaborate preparations and processing the haul of trepang efficiently. They moved

on and began again on another site, leaving their stone hearths for another season. In April or May the wind changed, and the south-east monsoon blew them back to South Sulawesi after absences of up to six months.

Local Aboriginals anticipated the arrival of praus each year and met the fishermen when they landed. There was day-to-day contact with the islanders. Aboriginals visited the praus and the camps the visitors set up on shore, they talked and traded, occasionally collected trepang with the Macassans, and co-operated while they boiled and smoked it. Relations between them were often familiar and friendly, and sexual relations between Aboriginal women and the visitors may have caused some violent altercations between tribesmen and Macassans. Relatively frequent connections with the islanders influenced Aboriginal language, legends, art and ritual. Contacts between islanders and Aboriginals were depicted in Aboriginal paintings, also in drawings by curious Europeans. Artists in north-east Arnhem Land drew praus in sand, and adorned them with masts and flags. Their art had ritual significance in mortuary ceremonies.¹⁸

One of the earliest records of the presence of smallpox among these northern visitors is in an account by the explorer Matthew Flinders of an unexpected meeting on the coast of north-eastern Arnhem Land during his voyage in the *Investigator*. On 17 February 1803 he saw six of the praus that collected trepang, and using his 'Malay' cook as interpreter, he found out that the commander of this small fleet was a short, elderly man from Macassar named Pobassoo, and that it was part of a much larger fleet of sixty praus carrying 1000 men. Pobassoo said he had made six or seven voyages to the coast of northern Australia in the preceding twenty years. He mentioned skirmishes with people on the coast, and told Flinders to beware of the indigenous people.¹⁹

The botanist Robert Brown was with Flinders, and in his diary on 18 February 1803, he said he accompanied him on board the prau of the commander, where there were 26 people, including several boys. There was talk of smallpox, and Brown was told that the treatment used for it was water affusion, that is, cool water was poured on the patient, and unless this was done regularly, 'the patient commonly dies'. Brown added: 'Among the people we saw very few

were pitted by the small pox and these very slightly'.²⁰ The Indonesian fishermen Brown saw had probably come from South Sulawesi and other islands, where introductions of smallpox were infrequent and most adults would have been susceptible. It had evidently occurred in the recent past, perhaps on Pobassoo's first voyage to Australia in the early 1780s.

Other indications that trepangers from Macassar were suffering from a lethal disease came when some of the British colonists in Australia settled at Raffles Bay in the trepang seasons of 1827–28 and 1828–29. A record of visiting praus referred to a surprisingly large number of deaths among Macassans on their short Australian voyages. On 21 February 1828 Captain Smyth of Fort Wellington, Raffles Bay, interviewed Daeng Riolo, the master of a Macassan prau. Through an interpreter, he learnt that the prau had left Macassar with forty-one others on 22 December 1827, and that 3 of the 14 crewmen died after leaving Macassar. Nothing more is known of that season's events.

However, a record of praus that arrived at Raffles Bay the following season shows that the loss of Daeng Riolo's crewmen was not unusual. In December 1828 and January 1829, 1046 crewmen left Macassar on thirty-four praus. By June 1829 they had returned with 1005 crewmen. During voyages that lasted between three and six months, 41 men had died. Three were speared by Aborigines, but in 38 cases no cause of death was specified. The record states only that the men 'died on the passage', and there are no dates of death, except for 2 men who died on 8 May and 17 May 1829, about the time their companions were sailing back. The distribution of deaths between praus was uneven; there were no 'deaths on the passage' on sixteen of thirty-four praus, eighteen recorded deaths, and on thirteen of those, between 5 and 10 per cent of the crewmen 'died on the passage'. The total number of deaths on the voyage was high by contemporary standards. There were twice as many deaths among Macassans that year as there were on convict ships sailing from Britain to Australia at the time.²¹

The evidence suggests a severe infection in susceptible crewmen was responsible, and smallpox was by far the most likely acute infection to be associated with such a high mortality. It is likely that many other crewmen, especially those who lived with the very sick

on stricken praus, and others on praus that had no deaths, also got the disease, and survived to complete the voyage after recovering. If the cause of deaths was indeed smallpox, it would have killed about one-third of its victims, perhaps nearly 40 crewmen, who were infectious and died sometime after arriving on the coast. Another two-thirds of its victims, perhaps over 70 crewmen, would have continued on their way while incubating the disease, or while infectious. There might have been infectious fishermen on the Northern Territory–west Queensland coast for six months before the fleet departed.

It might be expected that infectious visitors, who were no strangers to Aboriginal clans, would have transmitted infection to susceptible people in the first half of 1829, and that British newcomers living at Raffles Bay from 1827 to 1829 would have left records of the lethal disease their older members knew so well. They did not see active smallpox among Aboriginal people while they were there, but medical officers reported that local people were pock-marked. Nor were there reports of active smallpox in the records of the military establishment at Port Essington in 1838–49, but pock-marked people were seen there also. Smallpox had evidently occurred before settlement. There are few records of Indonesian visitors on the Kimberley coast, but when adventurous settlers in Western Australia explored Kimberley and the north-west in the 1860s and 1870s, one observer said ‘Malays’ had infected Kimberley clans in 1866, and others connected pockmarks with trepangers’ visits.

The first unequivocal evidence of smallpox in fishermen from the archipelago was recorded in Western Australia in the 1870s, when ‘Malays’ (mostly Indonesians) from islands in the eastern archipelago were employed as divers on pearling vessels on the north-west coast. The colonists’ quest for pearls and pearl-shell began in 1861 near Cossack, when hopeful and impecunious beach-combers and settlers explored rocks and beaches at low tide, and within ten years pearling became popular and profitable. Small boats and teams of Aboriginals multiplied, but the more successful the pearl-ers were, the harder it was to recruit Aboriginal labour. Neither Aboriginals nor colonial governments liked the industry, and in 1872 perhaps 150 or more divers, some from Kupang, were introduced to supplement local labour. In 1873 there were over eighty boats pearling out of Cossack. They worked farther west, farther

east, and farther north along the coast, and spread to Torres Strait. According to historian Kathy de la Rue, in 1875 there were nearly 1000 ‘Malays’ and nearly 500 Aborigines employed in vessels licensed at Cossack. When R. J. Sholl, Government Resident at Roebourne, included other vessels licensed at Fremantle or Shark Bay, he estimated that some 1800 ‘Malay’ divers were on the boats that year.²²

In 1875 their arrival on the north-west coast prompted frequent inspections under the Quarantine Act, and a Government Medical Officer, Dr C. S. Bompas, visited numerous vessels off Roebourne. In December he reported to Sholl that twenty-five pearling vessels had been inspected during the previous three months. There were nearly 1000 people on those vessels. They were a varied company: about 150 were British or Chinese, and they included owners, masters, mates, seamen, staff, boys, and a few women and children. Except for Arab divers on one schooner and Port Essington divers on another, the vast majority of divers and crews were called ‘Malays’ by Dr Bompas. Four schooners and a cutter came from Singapore with 142 Malay divers. The Arab divers and 173 more Malay divers came on three schooners, a brigantine and two cutters from Macassar. Three schooners from Macassar and Kupang brought 109 Malay divers and crew, and the *Azelia*, another schooner from Macassar and Kupang, had 81 Malay and Port Essington Aboriginal divers on board. Seven more schooners and three cutters from Kupang brought 323 more Malay divers and crew. A brigantine from Macassar, the *Fairy Queen*, had been shipwrecked on the north-west coast, and a cutter from Fremantle with an Australian native crew had rescued the master, 11 Arabs and 21 Malays. Dr Bompas had little to report about sickness on the vessels; a Malay on the *Azelia* died in hospital at Kupang, and on other vessels, one died of a chest complaint, one had syphilis, and several others had minor complaints. A blind Port Essington native was accompanying his son on the *Azelia*. But there were few reports of ill health, and no reports of smallpox.²³

The influx of islanders from Singapore, Macassar, and Kupang in 1875 was exceptional, and they were an unsuccessful labour force. De la Rue points out that the cost of employing Malays was greater than the cost of employing Aborigines, and they had to be

repatriated at the end of the season. They were said to be sickly, and less fit for the work than Aborigines. Cases of ill-treatment of Malays by pearlers surfaced, and the use of 'Malay' labour was one reason for unwelcome regulation of the industry. In 1875 the Dutch Governor-General in Batavia also introduced regulations about the employment of 'Malay' divers from Timor. The humanitarian legislation by the two governments discouraged pearlers from recruiting divers in Macassar and Kupang.²⁴

Not long afterwards, on 7 March 1876, the *Western Australian Times* reported news of smallpox from the port of Fremantle:

It has just become known that 'Small-Pox' is very prevalent on board most of the vessels at the North West Coast, and the '*Azelia*' reeking with the disease, has had, it is reported, several deaths on board. Many of the recent arrivals from the pearling ground actually visited the infected vessel, and now state they did not really know the disease they saw among the divers was small-pox.

The Government officials were also ignorant of the existence of this contagious disease, and the result is we have been mingling with persons who have come direct from the most infected spot.

I now hear the Government have issued orders that no pearling vessel is to be allowed within a particular boundary, and no one off such boats will be allowed to communicate with the shore at Roebourne. This of course will affect all vessels that have visited the infected boats.²⁵

Reports of smallpox were alarming. Smallpox in the pearling fleet was an immediate hazard for all concerned. It was another reason why the number of 'Malay' divers dropped to 13 in 1876.

Smallpox outbreaks probably occurred in all the ports of departure frequented by divers and crews of pearling vessels, and some who contracted it succumbed, as outbreaks on the *Azelia* and other vessels demonstrated. Divers and crewmen who were infectious when they arrived on the north-west coast could have transmitted smallpox to Aborigines who worked as divers on the same vessels. However, those who had caught it in the 1860s were immune, and there is no evidence of it among Aborigines in the 1870s. There is little evidence of smallpox in pearling fleets when the industry reached its

peak after Broome was founded in 1883. Quarantine was a routine precaution, and there were no cases among Aborigines.

The recent occurrence of Aboriginal smallpox on the unsettled north coast was apparent to well-informed newcomers years after South Australia acquired the Northern Territory in 1863. In the 1870s people at Palmerston (Darwin) who had seen pockmarked Aborigines in the district and inland realized the damage smallpox had inflicted before their own arrival, and they associated the incidence with the visitors from Macassar. In 1878 David Morgan, a settler at Port Essington, told the Government Resident that: 'On one or two occasions smallpox has broken out among the natives which can only be accounted for through being brought here by these Malays'.²⁶

Another colonist who realized what had happened was Paul Foelsche, the German-born Inspector of Police in the Northern Territory. He immigrated to South Australia in 1856, and had lived at Palmerston since 1870. Born near Hamburg in Germany, he enlisted in a Hussar Regiment in 1849, when revaccination was compulsory for military recruits in Prussia. More knowledgeable about the scourge than most colonists, he might have known about the savage return of European smallpox with the Franco-Prussian War, 1870–72. In 1881 he addressed the Royal Society of South Australia about the Aborigines of North Australia, and attributed the introduction of smallpox to 'Malays' who collected trepang.

Malay prahus, about 30 in number, visit the coast eastward from Port Essington to Blue Mud Bay in the Gulf of Carpentaria every year in search of 'beche de mer,' and have done so in all probability for centuries past. They arrive from Macassar the beginning of January, and leave again the end of May. During the time they are here they employ all the coast tribes trepanging for them, and they all live together; and I think there can be no doubt as to smallpox having been brought to these shores by them.²⁷

The introduction of smallpox at Palmerston in 1887 attracted rather more attention. Chinese immigrants worked on goldfields and the railway in the Northern Territory, and on 31 July that year, nearly two months after the arrival of the *Port Victor* with Chinese

passengers from Hong Kong, a Chinese covered with smallpox was found in the street at Palmerston. Another Chinese, who escaped quarantine officials, was found in the mangroves—he was well. In August, after the *Tsinan* arrived from Hong Kong, several more Chinese and a customs officer developed smallpox. The officer survived and was badly disfigured. Known contacts were quarantined, but the Chinese community was reticent. According to customs officer Alfred Searcy, the Acting Government Resident ordered the police to set fire to some twenty-six ‘humpys’ in Chinatown. In August a man from the *Tsinan* developed the disease after starting work at a railway camp fifty kilometres inland. After that, all Chinese arrivals from Hong Kong were quarantined, and at one time there were 300 or 400 Chinese at the quarantine station at Darwin Harbour. P. M. Wood, Colonial Surgeon and Protector from 1885 to 1889, undertook the mass vaccination of the native population of Palmerston, ‘assuring their protection from smallpox introduced to the port from time to time by Chinese immigrants’.²⁸

In fact, Chinese immigrants were never the source of Aboriginal smallpox in the Northern Territory. In 1906, in a Report on Hygiene in the Northern Territory, Dr Ramsay Smith concluded that:

There is no doubt that small-pox has caused extensive ravages among the blacks in many parts of the Territory, as in other parts of Australia. Its introduction has been ascribed to the Malays and people from various East Indian islands, who have for the past 300 years visited the river mouths and other parts of the coast when engaged in pearling and fishing for beche-de-mer. A good deal of blindness among natives is attributed to this disease.²⁹

So in the opinion of most nineteenth-century lay people and medical officers, smallpox was occasionally introduced to clans in northern Australia by travellers from South Sulawesi and neighbouring islands. Modern knowledge of the epidemiology and history of smallpox in island South-East Asia substantiates the opinion of contemporaries. Outbreaks followed close personal contacts between Aboriginals and sojourners who lived with them in trepang seasons, and the extreme susceptibility of Aboriginals accounts for the destruction it caused. Introductions of smallpox to Australia by travellers from infected islands over a century ago were like introductions

to other susceptible populations in the region, and foreshadowed introductions into Europe by air travellers from infected countries in the twentieth century, which occurred frequently with fearful results until travel was rigorously controlled.³⁰ In both situations, introductions and spread can be traced to face-to-face transmission in chance meetings between infectious and susceptible persons.

In the last fifty years, depopulation and diseases seen in northern Aboriginals have been attributed to their contacts with Macassan visitors, for instance by the Berndts in 1951. In 1972 Macknight suggested the Macassan connection may have gradually increased resistance to diseases introduced by Europeans, although there was no evidence for this. Similarly, in 1989 Mulvaney stated that in the case of smallpox the arrival of Macassans favoured the development of immunity along the coastal strip. Mentioning Macassan visitors in 1994, mammalogist Tim Flannery said they transferred germs and genes to Arnhem Landers over centuries. He believed the Macassans provided gradual resistance to infections, and their genes also helped to protect the Aboriginals against lethal diseases such as smallpox, tuberculosis, leprosy and malaria. The indigenous population was therefore large and relatively inured to several major diseases that proved fatal to neighbouring indigenous peoples when the Top End was settled.³¹

However, in extensive worldwide investigations during the smallpox eradication campaign, genetic selection for resistance was not definitively established. Nor were there opportunities for gradual inoculation over centuries in northern Australia, because there were no infectious Macassans until smallpox spread out from mainland South-East Asia in the late eighteenth century. There is no evidence that Macassans gave Arnhem Landers tuberculosis, but some Aboriginals in the Northern Territory suffered from it after Chinese and other migrants fraternized with them in the 1870s.³² Leprosy is not lethal, and apart from the severe physical handicaps it occasionally causes, its social consequences were more damaging than mortality and morbidity attributable to it.

The Macassan connection continued into the nineteenth century, when Chinese and Pacific Island labour introduced diseases into the Northern Territory and Western Australia. In the twentieth century such diseases were endemic in Arnhem Land.³³ In the case of

malaria, Arnhem Landers may have benefited, as Flannery thinks. But the damage caused by Macassan introductions of smallpox, and Chinese and European introductions of tuberculosis was greater than the biological benefit of Macassans genes. The impact of these two lethal new diseases was unquestionably severe. Damage caused by smallpox is not well known internationally, judging by Diamond's comments on the limited importance of Macassan visits.³⁴

Smallpox was introduced a number of times to German New Guinea after 1860 by Macassans, and was contained to some extent by vaccination performed by colonial officials in the 1890s. There are no reports of active smallpox among Aboriginals in northern Australia after the 1860s, and by 1900 it is unlikely that immunity conferred by previous attacks, or vaccination at Darwin, extended to many northern Aboriginal families. The incidence of smallpox increased in South Sulawesi, where it was endemic until the 1960s. The Macassan voyages ended in 1906, when the South Australian Government stopped issuing them with fishing licences.³⁵ There were no recorded outbreaks of Aboriginal smallpox after that.

We shall now trace epidemics of smallpox that occurred not long after memorable outbreaks in the archipelago in the 1780s, 1820s and 1860s, and investigate outbreaks of diseases introduced to Aboriginals by Europeans in those years.

5

Hidden History

Like Curr in the nineteenth century and Butlin in the twentieth, in the Australian chapter of his book *Guns, Germs and Steel* Diamond claimed that European germs, introduced soon after the British landed, caused the demise of Aboriginals in the colony of New South Wales. Displaying unfamiliarity with First Fleet sources in his description of the arrival of European germs in Australia, Diamond said: ‘Within a year of the first European settlers’ arrival at Sydney in 1788, corpses of Aborigines who had died in epidemics became a common sight’, and listed nine diseases he said were the principal recorded killers.¹

The one killer included in all the First Fleet sources was smallpox, the deadliest killer of all, but it was not seen until April 1789, over a year after Europeans arrived. Smallpox was the only epidemic recorded in 1789, and was the only known cause of Aboriginal corpses seen at that time. Venereal disease was mentioned in the records, but did not cause corpses. Tuberculosis was the only disease related to Aboriginal deaths in the 1790s. Evidence of smallpox and tuberculosis in Aboriginals follows; other killers were unrecorded.

Members of the First Fleet who explored the district around Sydney in 1789 saw signs of recent smallpox among Aboriginal people wherever they went, and could not understand where it had come from. Supposition about its origin began with Tench’s guesses,

and others have exercised their speculative ingenuity ever since. One way or another, their suggestions have now been debated for two centuries.

Contrary to the opinions of Cumpston, Butlin and Curson, we now know that variolous matter, mentioned by Tench, was not a likely source of Aboriginal smallpox at Sydney in 1789, because during the eradication campaign, it was established that smallpox virus was adversely affected by high temperatures and high relative humidity. Hence variolous matter would almost certainly have been inactivated by the voyage through the tropics in 1787, followed by fifteen months of Sydney's frequently hot and humid weather before April 1789, when Aboriginal deaths first occurred. Cambridge University historian Charles Wilson came to the same conclusion for similar reasons in 1987.²

Early in the twentieth century, when leading members of the medical profession debated the origin of Aboriginal smallpox, Cumpston knew there was no sign of it in the British or French ships in 1788, and said there was an unduly long gap between the arrival of the British in January 1788 and the arrival of smallpox in April 1789. At the time, he was aware that variolous matter was not the only current explanation of the epidemic. Cleland did not think an introduction of smallpox virus on the eastern seaboard in 1788 was the explanation of the appearance of smallpox there in 1789, and suggested it was introduced by trepangers on the north coast. Cumpston suspended judgement on Cleland's hypothesis pending more satisfactory information. His own cautious statement rested entirely on circumstantial evidence of the presence of the First Fleet and the presence of variolous matter. Tench, the only person who mentioned that the surgeons had a supply of it, clearly thought there had been no reason for it to be used.

Since 1914 new information, unknown to Cumpston, has tipped the balance in favour of Cleland's hypothesis. Research in recent decades lends weight to Cleland's opinion about the origin of Aboriginal smallpox. By the late eighteenth century, there were close economic and social ties between Aboriginal people and visiting Indonesian fishermen in trepang seasons, when smallpox could have spread to the Australians. As Cleland knew from contacts with Aboriginal people, and his interest in medical anthropology, chains



Key: ● outbreak H hypothetical outbreak

Eastern Australia: smallpox outbreaks 1780–90

Active smallpox and pockmarked Aboriginal survivors were reported in the Sydney district from April 1789 to February 1790. Elderly pockmarked people who had caught smallpox about that time were later identified by settlers in South Australia, New South Wales and Victoria, and Aboriginal informants in the Northern Territory and Queensland mentioned very old survivors.

of connection would have linked infectious northern Aboriginals to relatives in clans across Australia.

We remember how smallpox in South Sulawesi in the nineteenth century attracted the attention of European visitors, and there is historical evidence that a few outbreaks occurred here in the late eighteenth century, perhaps in the 1780s, when there were outbreaks on other islands in the archipelago. We already know that contacts between Macassan trepang fishermen from South Sulawesi and Aboriginal people may have initiated outbreaks on the coast of northern Australia during the fishing season on several occasions in the nineteenth century. There is no historical evidence of this in the late eighteenth century. However, records kept by Flinders and Brown suggest there could well have been infectious encounters at that time; that is, some years before the newcomers at Sydney saw local Aboriginals suffering severely from smallpox.

Meetings with infectious visitors were likely to trigger outbreaks in wholly susceptible Aboriginal populations between Arnhem Land and the Gulf, and, when it happened, smallpox could be expected to spread rapidly through northern indigenous communities. Jack Davis, an Aboriginal man from Port Essington, was a well-known local identity, and was one of Foelsche's informants in the 1870s. He said smallpox was on the Cobourg Peninsula a long time ago, and remembered very old people who had it when they were children. They called it *Meeha-meeha*, and said it had killed 'plenty black-fellows'.³ They might have been among the first recorded victims of smallpox in Australia. There is no doubt that it spread extensively when it was introduced in the 1780s. It probably travelled in chains of connection between the north coast and southern and eastern Australia, following the same paths of infection it would follow in the 1820s, when, as we shall see, it spread from northern and north-eastern Australia, not only around the coast, but through the interior to the Murray–Darling Basin and the south coast in 1830, and to the Great Dividing Range and the east coast late in 1831. It would travel in the same directions in sparsely settled parts of eastern Australia in the 1860s.

By the mid-nineteenth century, it was clear to older migrants in south-eastern and southern Australia that smallpox had occurred

before settlement. They had known it well in the late eighteenth and early nineteenth centuries, in Britain, as an acute disease and as the cause of pockmarks.⁴ As we shall discover, when the country was settled in the 1830s, 1840s and 1850s, well-informed British and German migrants saw elderly pockmarked Aboriginal people in districts they occupied. Curson, who doubted the connection of their reports of facial pockmarks with outbreaks at Sydney, said evidence for smallpox extending well beyond Sydney was 'highly suspect'.⁵ He could not come to terms with substantial evidence of people who acquired pockmarks in 1789 or 1830, and who were seen inland as well as on the coast. In the smallpox eradication campaign, comparable evidence from experienced lay observers about where and when smallpox had last occurred proved reliable, and was used effectively.

In South Australia, pockmarked people over fifty were seen by well-informed lay observers, who included painter and ethnologist G. F. Angas, settler James Hawker, missionary George Taplin and Governor Hunter's nephew, John Hunter Kerr. In New South Wales, they were observed by the surgeon John Mair and many settlers in the interior and in coastal districts. Among observers in Victoria were pioneer and overlander Joseph Hawdon, Gannawarra settler A. M. Campbell, Dr James King of the Medical Board, Peter Beveridge the pastoralist and ethnographer and other settlers on the Murray and its tributaries. Farther south, while in the Port Phillip District, pastoralist Peter Snodgrass, Dr David Thomas, Kerr and others also met elderly survivors. These old people reported that they had smallpox when they were young, so settlers realized outbreaks had occurred about the time smallpox had broken out at Sydney. Because settlers arrived in Queensland later than in the older colonies, they were less familiar with smallpox and pockmarks, and by then, not many very old victims were left; so there are fewer records of old pockmarked people there. Later records of Aboriginal smallpox in eastern Australia at that time, written by well-informed lay people and several medical practitioners, will be included with other evidence of it in South Australia and New South Wales in chapters 6 and 7. Records of Aboriginal smallpox at Sydney in 1789 are presented below.

Aboriginal smallpox in Sydney 1789–90

These records, which afford unusual evidence of virgin soil outbreaks of smallpox, are more recent by over two centuries than early records relating to indigenous peoples on the isolated American continents. Letters and journals written by literate members of the First Fleet go beyond the formal report by Governor Phillip. Collins, Hunter, Tench, Bradley, Clark and Mrs Macarthur realized they had witnessed an extraordinary and tragic event. Their relations with 2 previously unknown young victims continued for several years.

Governor Arthur Phillip reported the 1789 outbreak in official despatches.

In the beginning of the following April [1789] numbers of the natives were found dead with the small-pox in different parts of the harbour; and an old man and a boy of about eight years of age were brought to the hospital. The man died, but the boy recovered, and now lives with the surgeon. An elderly man and a girl of about ten or eleven years of age were found soon after and brought up; of the man there was no hope of recovery, and he died the third day, but the girl recovered, and lives with the clergyman's wife. I brought these people up with the hopes that being cured and sent away with the many little necessaries we could give them would be the means of reconciling them to live near us; but unfortunately both the men died, and the children are too young to have weight with the natives with whom since they have frequently conversed, and what was more unfortunate our native [Arabadoo] caught the disorder and died. It is not possible to determine the number of natives who were carried off by this fatal disease. It must be great; and judging from the information of the native now living with us [Bennelong], and who had recovered from the disorder before he was taken, one half of those who inhabit this part of the country died; and as the natives always retired from where the disorder appeared, and which some must have carried with them, it must have been spread to a considerable distance, as well inland as along the coast. We have seen traces of it wherever we have been.⁶

David Collins, Deputy Judge-Advocate and Lieutenant-Governor in 1789, also described the outbreak.

Early in the month [April], 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 . . . the bodies of many . . . natives . . . The cause . . . remained unknown until a family was brought up, and the disorder pronounced to have been the small-pox. It was not a desirable circumstance to introduce a disorder into the colony which was raging with such fatal violence among the natives; but saving the lives of any of these people was an object of no small importance, as the knowledge of our humanity, and the benefits which we might render them, would, it was hoped, do away with the evil impressions they had received of us. Two elderly men, a boy and a girl were brought up, and placed in a separate hut at the hospital. The men were too far overcome . . . but the children did well from the moment of their coming among us . . .

May Of the native boy and girl . . . on their recovery from the small-pox, the latter was taken to live with the clergyman's wife, and the boy with Mr White, the surgeon, to whom for his attention during the cure, he seemed to be much attached. While the eruptions . . . continued upon the children, a seaman belonging to the *Supply*, a native of North America, having been to see them, was seized with it and soon after died; but its baneful effects were not experienced by any white person of the settlement, although there were several very young children in it . . . From the first hour of the introduction of the boy and girl . . . it was feared that the native who had been so instrumental in bringing them in, and whose attention to them . . . excited the admiration of every one . . . would be attacked by the same disorder; as on his person were found none of those traces of its ravages which are frequently left behind. It happened as the fears of every one predicted; he fell a victim . . . in eight days after he was seized with it, to the great regret of every one . . .⁷

Collins left Sydney in August 1796. He completed and published his *Account* in 1798. In an Appendix, he added:

The number that it swept off, by their own account, was incredible. At that time a native was living with us, and on our taking him down the harbour to look for his former companions, those who witnessed

his expression and agony can never forget either. He looked anxiously around him . . . 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, which he preferred during the remainder of our excursion. Some days after he learned that the few of his companions who survived had fled up the harbour to avoid the pestilence that so dreadfully raged . . . He fell a victim to his own humanity when . . . others were brought into the town covered with the eruption of the disorder. On visiting Broken Bay we found that it had not confined its effects to Port Jackson, for in many places our path was covered with skeletons, and the same spectacles were to be met with in the hollows of most of the rocks of that harbour.

Notwithstanding the town of Sydney was at this time filled with children, many of whom visited the natives who were ill of this disorder, not one of them caught it . . . that it was the small-pox there was scarcely a doubt; for the person seized with it was affected exactly as Europeans are who have that disorder . . . on many that had recovered . . . we saw the traces, in some the ravages of it on the face. As proof of the numbers . . . carried off . . . Ben-nil-long told us that his friend Cole-be's tribe being reduced . . . to three persons, Cole-be, the boy Nan-bar-ray, and some one else, they found themselves compelled to unite with some other tribe, not only for their personal protection, but to prevent the extinction of their tribe. Whether this incorporation ever took place, I cannot say.⁸

James Scott, Sergeant of the Marines, wrote that on 15 April 1789:

I Went With a party to Cut Grass tree for Lt Johnstone. found three Natives Under A Rock, Vis. A Man and two Boys (of Which One Boy Was Dead) the Governor being Aquented With it. Ordered the Man & Boy to the Hospital Under Care of the surgeon the having the Small pox. the Man died. ye. Next day—the Boy contineus to get Better.⁹

In April and May 1789 Watkin Tench wrote that:

An extraordinary calamity was now observed among the natives. Repeated accounts brought by our boats of finding the bodies of the Indians in all the coves and inlets of the harbour caused the gentlemen of our hospital to procure some . . . for examination. 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; but how a disease, to which our former observations had led us to suppose them strangers, could at once have introduced itself, and have spread so widely, seemed inexplicable. Whatever might be the cause, the existence of the malady could no longer be doubted. Intelligence was brought that an Indian family lay sick in a neighbouring cove: the governor, attended by Arabanoo, and a surgeon, went in a boat immediately to the spot. Here they found an old man stretched before a few lighted sticks, and a boy of nine . . . pouring water on his head, from a shell . . . near them lay a female child—dead, and a little further off, its unfortunate mother: the body of the woman shewed that famine, superadded to disease, had occasioned her death: 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. Their situation rendered them incapable of escape, and they quietly submitted to be led away.

Arabanoo . . . seemed at first unwilling to render them any assistance; but his shyness soon wore off, and he treated them with the kindest attention. Nor would he leave the place until he had buried the corpse of the child: that of the woman he did not see . . . and as his countrymen did not point it out, the governor ordered that it should not be shewn to him . . .

April 1789. An uninhabited house, near the hospital, was allotted for their reception, and a cradle prepared for each of them. By the encouragement of Arabanoo, who assured them of protection, and the soothing behaviour of our medical gentlemen, they became at once reconciled to us, and looked happy . . . 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 to him; but the obstructed tender state of the part rendered it impracticable. *Bado, bado* (water), was his cry: when brought to him, he drank largely at intervals of it. He

was equally importunate for fire, being seized with shivering fits; and one was kindled. Fish were produced, to tempt him to eat; but he turned away his head with sighs of loathing. Nan-bar-ee, on the contrary, no sooner saw them than he leaped from his cradle, and eagerly seizing them, began to cook them. A warm bath being prepared, they were immersed in it . . . had clean shirts put on them, and were again laid in bed. The old man lived but a few hours. He bore the pangs of dissolution with patient composure; and though he was sensible to the last moment, expired almost without a groan. Nanbaree appeared quite unmoved . . . simply exclaiming, bo-ee (dead) . . . Although barely able to raise his head, while so much strength was left to him, [the old man] kept looking into his child's cradle; he patted him gently . . . and, with dying eyes, seemed to recommend him to our humanity and protection. Nanbaree was adopted by Mr White and became . . . one of his family.

April, 1789. Arabanoo had no sooner heard of the death of his countryman, than he hastened to inter him . . . It differed, by the accounts of those who were present at the funeral of the girl, in no respect . . . except that the grave was dug by a convict . . . when intelligence of the death reached Arabanoo, he expressed . . . doubt whether he should bury, or burn the body. Indeed, Arabanoo's behaviour, during this day was so strongly . . . marked by affection to his countryman, and by confidence in us, that the governor decided to free him from all further restraint . . .

Distress continued to drive them in upon us. Two more natives . . . a young man and . . . his sister, a girl of fourteen years old, were brought in by the governor's boat, in a most deplorable state of wretchedness from the small-pox. The sympathy and affection of Arabanoo, which had appeared languid in the instance of Nanbaree and his father, here manifested themselves immediately. We conjectured that a difference of the tribes to which they belonged might cause the preference. The young man died at the end of three days: the girl recovered and was received as an inmate, with great kindness, in the family of the clergyman's wife. Her name was Boo-ron . . . she acquired that of Abaroo, by which she was generally known.

May 1789 . . . the premature loss Arabanoo, who died . . . on the 18th instant, after languishing six days. From some imperfect marks and indents on his face, we were inclined to believe that he had passed

this dreaded disorder. Even when the first symptoms of sickness seized him, we continued willing to hope that they proceeded from a different cause. But at length the disease burst forth with irresistible fury. It were superfluous to say, that nothing which medical skill and unremitting attention could perform, were left unexerted to mitigate his sufferings, and prolong his life, which humanity and affectionate concern towards his sick companions, unfortunately shortened. During his sickness he reposed entire confidence in us . . . The governor, who particularly regarded him, caused him to be buried in his own garden, and attended the funeral in person.¹⁰

From time to time, Tench mentioned pockmarked people who had recently suffered smallpox. In September 1790 he described an 'Indian' who stood aloof from his fellows, but shook hands willingly with the visitors: 'He seemed to be between 30 and 40 years old, was jolly, and had a thoughtful countenance, much marked by the small-pox'. Tench also saw pockmarked survivors on excursions into the interior, where he met people who lived on small animals and roots rather than mullet from rivers. In April 1791 he met Gomberee, 'a man of middle age, with an open cheerful countenance, marked with the *small pox*, and distinguished by a nose of uncommon magnitude and dignity'. By then, he thought the Cameragal people were the most powerful community in the country. He concluded that: 'The tribe of Cameragal is of all the most numerous and powerful. Their superiority probably arose from possessing the best fishing grounds; and perhaps from their having suffered less from the ravages of the small-pox.'¹¹

When Admiral John Hunter returned to Sydney on the *Sirius* in May 1789, he heard why he had seen no natives or signs of life on his way to the town.

it was truly shocking to go around the coves of this harbour, where, in the caves of the rocks, which used to shelter whole families in bad weather, were now to be seen men, women and children, lying dead . . . and, if we consider the various attitudes, which the different dead bodies have been found in, we may easily believe that when any of them are taken ill, and the malady assumes the appearance of the small-pox, (having already experienced its fatality to whole families,) they are immediately deserted by their friends, and left to perish, in

their helpless situation, for want [of] sustenance. Some have been found . . . with their heads reclined between their knees; others were leaning against a rock, with their head resting upon it.

On an excursion to Broken Bay early in June, Hunter saw a party of women fishing; they soon 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 therefore crept off, and concealed herself . . . information was immediately brought to the governor, and we all went to see this unhappy girl . . . just recovered from the small-pox, and lame: she appeared to be about 17 or 18 years of age, and had covered her debilitated body with grass . . . she was very much frightened on our approaching her, and shed many tears . . . and with the assistance of a few expressions which had been collected from poor Arabanoo when he was alive, we soothed her distress a little . . . then we shot some birds . . . skinned them, and laid them on the fire to broil, together with some fish, which she eat; we then gave her water, of which she seemed to be much in want . . . and indeed . . . she had a considerable degree of fever on her. Before we retired for the night, we saw her again, and got some fire-wood laid within her reach . . . and left her to her repose . . . Next morning we visited her again; she now got pretty much the better of her fears, and frequently called to her friends . . . we were no sooner gone from the beach, than we saw some of them come out of the wood . . .¹²

Lieutenant William Bradley had returned with Hunter on the *Sirius* and heard about the sick at the hospital, whom, to the surprise of the onlookers, Arabanoo had met without fear. Whether they were strangers to it was not known. He described the many dead, and the dreadful havoc.

we did not see a canoe or a Native the whole way coming up the Harbour and were told that scarce any had been seen lately except laying dead . . . whence it appears that they are deserted by their companions as soon as the disorder comes out on them and . . . left to shift for themselves. We judge this from their having been found not buried in every part of the Harbour . . . some who have apparently used their utmost exertions to get at water having been found laying dead between a Cave and a run of water . . .

On 2 June 1789 Bradley noted that twenty canoes had passed Sydney Cove going down the Harbour, which was the first time any number had been seen together since smallpox broke out. On 11 June he commented: 'A Native Man was met with at Botany Bay who had just recovered from the small pox. He had a child with him and made signs that the mother of it had died of that disease.' On 19 June he mentioned that 70 Aborigines were seen in Spring Cove. On 30 September, during a survey of Botany Bay, he remarked on skeletons, and saw few people. On 25 November, he saw a great number on both sides of a north arm of the harbour, where Bennelong and Colbee were captured.¹³

Elizabeth Macarthur had arrived at Sydney on the Second Fleet early in June 1789, and wrote to London in 1791.

In the winter of 1789 a dreadful case of smallpox was discovered among the natives . . . Nor is it to be wondered at that this disorder should in general be so fatal to them, when we consider they are not in possession of a single palliative, nor have any means of procuring nourishment . . . when their strength no longer permits them to pursue their usual avocations of fishing, hunting the kangaroo and other animals. Amongst the unhappy [victims] were a boy and girl . . . these two children were very useful, and led to a better understanding with the blacks . . .¹⁴

On 15 February 1790 Lieutenant Ralph Clark wrote in his diary that at Lane Cove he had spoken with Tirriwan, the father of an Aboriginal child, 'and that his woman the mother of his child was (poc) dead of the (mittayon) Small Pox'. At the time, Tirriwan's child had not quite recovered. Lieutenant Fowell told his father in July 1790 that when his party had arrived, not a canoe was to be seen, because they had all left the harbour and fled northward. Boats were often sent down the harbour for no other purpose than to bury the dead found on the beaches. Fowell too described the fury of smallpox and the loss of life, although, according to him, Aboriginal people were generally found with the remains of a small fire on each side of them and some water left within reach.¹⁵

Others who arrived at Sydney soon after outbreaks, as George Barrington did, mentioned it in accounts of the colony, and for forty years or more visitors and migrants who saw pockmarked Aboriginal people at Sydney realized smallpox had occurred there. British

surgeon John Washington Price, who arrived at Port Jackson in January 1800, described the dreadful devastation caused some time ago by a disease 'exactly resembling the small pox'. He heard about the children treated at the settlement, and said closer relations between Aboriginal people and colonists had resulted. Soon afterwards, French explorer M. F. Peron was at Sydney. In Tasmania, where there was no Aboriginal smallpox, his contacts with indigenous people were rare, difficult and dangerous. He did not mention Aboriginal smallpox on the mainland. However, when he described the Blue Mountains, he said:

the savages of these parts have a sort of religious terror for the Blue Mountains; they think them the residence of a kind of evil spirit . . . However incredulous that belief may be in itself, it . . . has its cause in their observations . . . it is in fact from the tops of these mountains that proceed all the plagues which infest the country.¹⁶

Thirty years after the catastrophe, newcomers and visitors saw pockmarks and acknowledged that smallpox and other diseases had occurred before their own arrival. W. C. Wentworth said:

many of those who fell victims to it, are still living; and the deep furrows that remain in some of their countenances, shew how narrowly they escaped the same premature destiny. The recollection of this dreadful malady will long survive in the traditionary songs . . . The consternation which it excited is still as fresh in their minds as if it had been but an occurrence of yesterday, although the generation which witnessed its horrors, has almost past away. The moment one of them was seized with it, it was the signal for abandoning him . . . and in some of the caves on the coast . . . sufferers were left to expire . . . from the want of sustenance.¹⁷

One of the generation to live through smallpox was a man from Cook's River at Botany Bay. He was Mahroot, alias the Bo'sun. In 1845 he was asked what Aboriginals meant by devil devil? He replied: 'Devil devil is all over small pox like'.¹⁸

Commentary

From its first appearance, the British speculated about the origin of the epidemic. Collins doubted that smallpox was a new disease among Sydney Aboriginals in 1789, because they had a name for it.

But horrified victims always found a name for smallpox, and, more to the point, Aboriginals with pockmarks had not been noticed by newcomers before April 1789. As some at the settlement suspected from Arabanoo's fearless attitude to sick victims, the infectious nature of the new disease was unknown to him. His symptoms and his death establish that marks on his face were not pockmarks, as Tench hoped. It was soon clear that the local clans had not been exposed to smallpox before, because they were wholly susceptible. Tench was the only one to raise the possibility that smallpox came with the First Fleet, and he soon dismissed it out of hand. It was only one of several possibilities he raised, and there is no evidence of it in passengers or crew on any of the ships in the First Fleet. Nor is there any evidence that it was introduced after the First Fleet arrived at Sydney as a result of virus on fomites, as Curr later proposed. There is no evidence that the French introduced smallpox during the visit of La Pérouse in 1788.

Present knowledge of smallpox in the archipelago and South Sulawesi, Macassan visitors and their trade in northern Australia, and communication networks within Australia, make it seem highly likely that outbreaks of smallpox swept south from the north coast of Australia over a period of a few years, and from centres of infection in the interior in the 1780s, moved towards the coastal range. Peron thought that the plagues that infested the country seemed to come from the tops of the Blue Mountains; and despite differences in social and economic mores and linguistic patterns between inland and coastal clans, unpredictable chance contacts with travellers and neighbours could have caused infection to spread until smallpox reached the coast at Sydney in April 1789. Meanwhile, it continued to spread farther south, as evidenced by the sighting of a few old pockmarked Aboriginal people in country occupied by newcomers in the 1830s and 1840s.

Contemporary accounts provide ample evidence that the disease the British saw at Sydney in 1789 was indeed smallpox, which killed and scarred its victims as no other disease did, and we can tell from modern knowledge of smallpox that lay people and members of the medical profession, who saw it at close quarters in 6 victims at the settlement's hospital, described smallpox accurately. It occurred in 2 Aboriginal children and 3 adults, and, as Collins said, in a Native American seaman who had visited the sick children. All 4 adults

died in less than a week, and the 2 children recovered, as children often did. Already well established at Sydney in April, smallpox was rampant at Port Jackson in May. The few who survived while it raged around them fled up the harbour or inland, and by June it spread to Broken Bay. On a north arm of the harbour, Bennelong recovered before his capture in November. At Lane Cove, Tirriwan's child was still unwell in February 1790. The epidemic lasted nearly a year in Aboriginal communities in the Sydney district.

The incidence of smallpox in the entirely susceptible indigenous population was high, except, according to Tench, in Cameragal people, whom he described as numerous some time after the outbreak. However, as the observations of Hunter and Bradley, who returned on the *Sirius* at the height of the epidemic, suggest, the mortalities of smallpox in Aboriginal populations were very high. This was typical in families infected with smallpox who slept together; and ate together, and in 1789 there were no immune survivors from earlier outbreaks to procure food and water, and to look after the sick. Accordingly, prostrated victims may sometimes have died of starvation. In the circumstances, the impact of what may have been the first epidemic of acute and lethal infectious disease in previously isolated Australian hunter-gatherers was especially severe, because of shock, flight and fear, as well as the virus itself.

Some of the women who died of smallpox were mentioned in the records. On the day Nanbaree was brought in, Arabanoo buried a child, and did not see a dead woman near by. Abaroo's male relative died, and apparently there was no woman with them. At Botany Bay, Bradley saw a man who had recovered and had a child with him whose mother had died. Similarly, Tirriwan told Clark that his child's mother had died of smallpox. The few cases that were reported might reflect the usually higher toll of smallpox in women than in men. The disproportionate numbers of men and women in enumerations of Aboriginal populations in the colony of New South Wales before 1850 has never been accounted for, and may be related to higher smallpox mortalities in women. For some time the loss of women food-gatherers and childbearers may have delayed a return to customary life and demographic recovery. Smallpox mortalities in eastern Australia in the 1780s were probably greater than those in later epidemics. There may have been more local outbreaks

and higher mortalities in larger and wholly susceptible populations between 1780 and 1790 than there were in later outbreaks in smaller and partly immune bands in newly settled districts, where there was settler support and, in some cases, vaccination.

Although the evidence of virgin soil smallpox is compelling, the interpretation of some features of the records was recently questioned. Looking at Aboriginal cases, Frost was surprised that observers were still commenting on Nanbaree's pustules several weeks after he was brought to the settlement covered with them. However, they would have persisted a long time, because heat, sand, smoke and dirt would have caused bacterial infection, which exacerbated them and was not uncommon in cases of smallpox. Frost also said Arabanoo was from the first at risk of contracting smallpox 'via both air and touch', and mentioned his burial of the dead child, his part in bringing the sick Nanbaree and his dying father to the settlement, and his contact with them. In the circumstances, he thought the onset of Arabanoo's illness on 10 May was too long after his contact with these sick victims on 15 April.¹⁹ But while indirect transmission by airborne infection did occur, it was very rare. It was also established in the course of smallpox eradication that virus on contaminated objects was rapidly inactivated and fomites were of little importance. Heavily contaminated corpses were sometimes an occupational hazard for undertakers and pathologists; however, by far the most important cause of infection was inhalation of droplets at close range, which was likely to occur if there was face-to-face contact with a person who had a rash.²⁰ Arabanoo had no such contact with the dead child, and perhaps not with Nanbaree's prostrated father, who soon died, and his first contact with Nanbaree was in open air. He may have visited him soon after, when he was under the surgeon's care in a separate hut at the hospital, although, according to Tench, Arabanoo's sympathy for Nanbaree was 'languid'. It seems more likely that Arabanoo was infected by Abaroo. She arrived later in April, and he displayed greater sympathy and affection for her. The record reflects only minor differences between cases, which were not unusual, and do not affect the retrospective diagnosis of smallpox.

The fact that there were no European cases at all, even when smallpox was raging at Sydney early in May 1789, surprised Curson

and Frost. At the time, Collins had remarked on it because there were several very young children in the settlement. Collins returned to England in 1796 and in 1798 published an account of the colony. He said Sydney was at that time 'filled with children', many of whom visited the sick natives, but none caught smallpox. Enlarging on this, Curson and Frost estimated that there were 40 or 50 young children, born after the First Fleet left England in 1787, who were present during the epidemic. Curson said if symptoms of smallpox had appeared among them, they were probably accepted as 'commonplace and not worth recording'. But sickness among Europeans usually was reported, and smallpox was never commonplace. Frost found the absence of smallpox among the newcomers the most puzzling feature of the evidence.²¹

It may be less of a problem than it seems. In the late eighteenth century, most people from Britain were immune after previous attacks, or after variolation, and most infants were protected by immunity conferred by immune mothers.²² Smallpox was not usually transmitted in casual contacts, and it is most unlikely that any white toddlers and two-year-olds (or any older children) at the settlement had face-to-face contact with the sick Nanbaree or Abaroo, or their dying relatives, who were in a separate hut at the hospital. Arabanoo did play with the white children, but he would have been prostrated by severe illness at the end of the incubation period, which would have stopped the games, and he would not have been infectious until he had a rash a day later.

It is also unlikely that there were infectious contacts outside the settlement between Aboriginals and any of the few susceptible Europeans, because as early as March 1789 relations between them were often hostile. Interference with Aboriginal food supplies by the Governor's game-keeper, convicts and others wanting sweet tea and vegetables was constant, and was followed by news of the dead rushcutters' fate. Abrasive meetings between Aboriginals and convict stragglers continued, and outright clashes that occurred at the brick kilns were all described by Collins.²³ Face-to-face contacts were always unusual, and after smallpox broke out in April 1789, severely ill and highly infectious Aboriginal victims could not move about. In June 1789, before the epidemic ended, 1000 more convicts arrived in the *Lady Juliana* and the Second Fleet.²⁴ They, too, were

mostly immune, had little or no contact with sick Aboriginals, and none caught smallpox.

In 1789 newcomers did not know the size of local populations, and Phillip's conservative estimate of 1500 people around Port Jackson was never revised. Large groups were still occasionally seen; however the presence of 200 at a whale feast does not support Curson's suggestion that 'by 1790, the Sydney Aboriginal community seems to have made a remarkable recovery'.²⁵ Bennelong's estimate, that half of those in the Sydney district had died, is the only contemporary estimate of smallpox mortality, and it is a low estimate among mortalities attributed to smallpox in naive populations. Contemporaries were unanimous when they reported scenes of disaster, and the scale of the catastrophe was acknowledged in the nineteenth century. In the twentieth century, when archaeologists were mystified by unusual skeletal remains at Port Hacking, Cleland told them about the smallpox epidemic.²⁶

Smallpox reduced Aboriginal numbers around Sydney to such an extent that newcomers never saw Aboriginal communities anywhere near full strength, and a small number of survivors of smallpox were prominent in early accounts of the colony. Pockmarked people who had recovered from smallpox as children and young adults included Nanbaree and Abaroo, the girl whom Hunter helped, Bennelong, Colbee, and Mahroot, while Gomberee and the aloof Aboriginal were 2 of the older victims. The pockmarked survivors of 1789 were among Sydney people whom colonists and visitors painted and wrote about. French artist N. M. Petit painted them in 1802, Wentworth knew them in 1819, Bellingshausen, Lesson and Mair saw them in the 1820s. Members of the Select Committee knew of them in 1845. Today's knowledge of virgin soil smallpox and how it damaged indigenous populations in North America goes a long way towards explaining the near demise of Sydney's indigenous population.

Conclusion

The mysterious epidemic of smallpox that occurred only among Aboriginal people at Sydney in 1789 was recorded by people who knew more about the disease than most of us do now. However,

today we not only know more about the continent and indigenous Australians than members of the First Fleet did in 1789, we also know much more about the history of smallpox in the region. The most likely explanation of its unexpected appearance at Sydney seems to be that it was behaving as it did in the 1820s, when historical evidence shows that it entered Australia on the north coast, and spread through the interior to the south-east, to reach the east coast of New South Wales a few years later. As international experiences demonstrate, susceptible populations were often affected more than once by contacts with travellers from heavily infected neighbouring countries, and it happened again in Australia in the 1860s.

The Aftermath

The smallpox epidemic led to unexpected contacts between Aborigines and the intruders at a time when there was very little other communication between them. The Surgeon John White's 'adoption' of Nanbaree, which was the first adoption by a European of an Aboriginal child in Australia was initially more successful than many of those that followed, and Abaroo was treated as a member of clergyman Johnson's family. The admiration and liking the settlers expressed for Arabanoo were exceptional, so was the decision to bury him in the Governor's garden. But Phillip's hope that help at the time of the great sickness would be the means of reconciling the two races was short-lived. After Arabanoo's death, White, William Dawes and the clergyman still had friendly relations with Nanbaree and Abaroo, but there was little contact with other Aborigines, and before long the unabated hostility that Tench talked about in 1788 intensified. Relations between the indigenous people and newcomers worsened, and Tench talked about the Aborigines' suspicious dread of the Europeans, and attacks on convict stragglers. In the following year, Governor Phillip was speared by a visitor from a Broken Bay clan.²⁷

The British newcomers never found out where smallpox came from, and whatever Aboriginal people thought about it at the time is not clear in written records. Dispossession and disease were closely linked, and they probably thought smallpox arrived with the British.

Smallpox was one of the reasons why they disliked the English flag, and, according to Lesson, their dislike was why the British claimed that the French had caused Aboriginal smallpox, as Wentworth did in 1824. The old tribal chief's fear of getting the disease that killed if he boarded *La Coquille* suggests they were successful. The long-remembered disease was recalled by puzzled European authors as well as indigenous authors in the late twentieth century.

Aboriginal deaths caused by smallpox in 1789 were the first of many deaths due to disease to occur in the colony, in particular deaths from tuberculosis, including some in people who had survived smallpox. The capture of Colbee and Bennelong at the end of 1789 made little immediate difference to contact between the two races, but by 1791 Bennelong's relations with Phillip were cordial. He brought many friends to the Governor's house and other houses, where they often stayed for long visits. From that time on, it was likely that colonists transmitted the diseases of Europe to Aboriginal people who had contact with them. The incidence of tuberculosis was certainly increasing in England, and recent research confirms its presence in passengers in the First, Second and Third Fleets, who arrived at Sydney between 1788 and 1791.²⁸

Like smallpox, tuberculosis was readily transmitted by face-to-face contact between infectious and susceptible persons. Tench and Collins knew Aboriginals at Sydney by name in the early years of settlement, and mentioned a number who had personal contact with the invaders in the 1790s. When Collins left the colony in 1796, he recorded the death of Bennelong's wife, Barangaroo, and went on to say that Colbee's wife, whose name was the same, did not survive her for long, but died of consumption. After her death, the funeral procession passed the door of every hut and house she had visited during the latter days of her illness.²⁹ It is likely that although there were only about 5000 Europeans in the settlement by 1800,³⁰ tuberculosis was already established in the small Aboriginal population. Some thirty years later, Lesson found that although measles and scarlet fever, so common in Europe, were altogether absent at Sydney, 'pulmonary troubles' were prevalent in Europeans, who suffered in winter weather; and he said most indigenous Australians had what he called 'chronic catarrh', while some Aboriginal women were

consumptive.³¹ By then, tuberculosis was almost certainly the greatest hazard for indigenous people who had close contact with settlers, and it would have spread farther when neighbouring clans met.

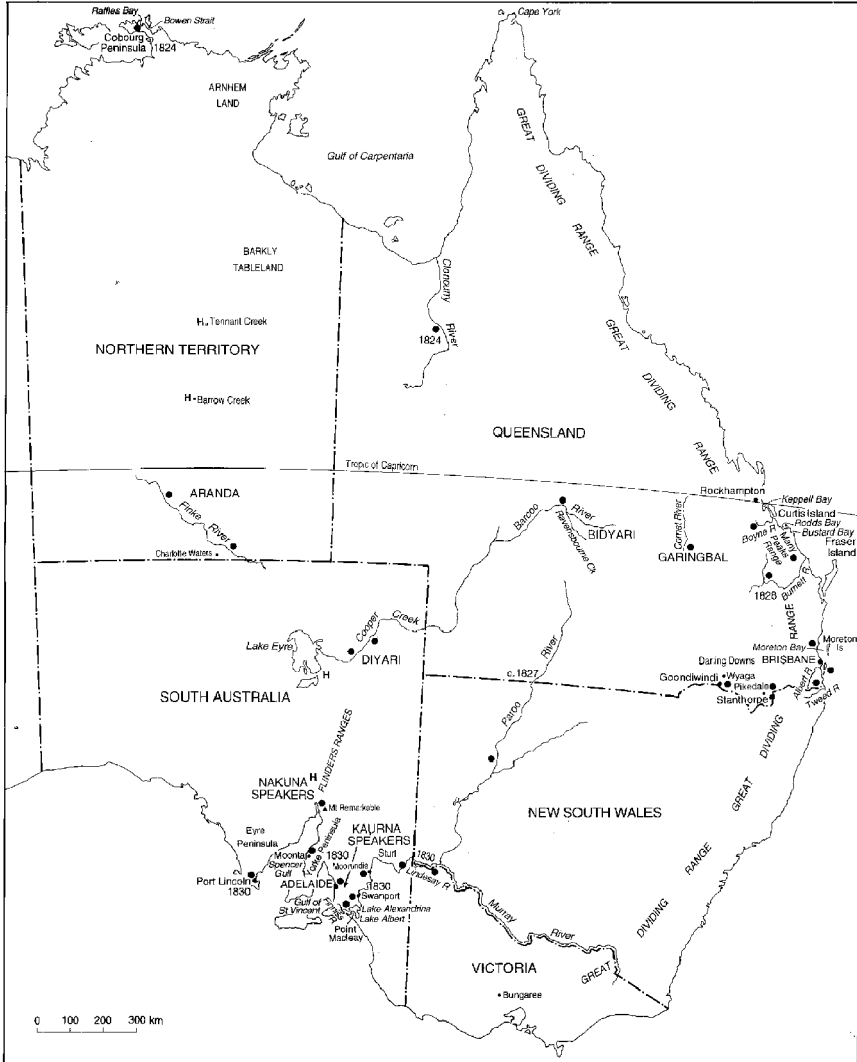
As we know, Dubos found that first outbreaks of tuberculosis among Native North Americans were remarkably destructive, and Francis Black has attributed a major role to tuberculosis in the deaths of 600 of 800 newly contacted South American Surui between 1980 and 1986.³² However, the history of these diseases in indigenous populations in Australia differs from their history in indigenous American populations. In North America, invading Europeans certainly introduced smallpox on the east coast, for the first time in the seventeenth century; then introduced the white plague on numerous occasions a century later. But in Australia, the two lethal diseases occurred in the same decade, when smallpox reached Sydney in 1789 after visitors introduced it on the north coast in the 1780s, and British invaders with tuberculosis arrived at Sydney in 1788. In 1828, forty years after the first settlers had arrived, the European population had increased to about 36 000,³³ and in the Sydney district, the indigenous population was completely outnumbered. Their isolation from Old World diseases had ended when two of the world's worst diseases invaded Australia, perhaps for the first time. These plagues devastated scattered hunter-gatherer populations around Sydney Harbour almost simultaneously. Their small numbers astonished newcomers.

6

The Frontiers of Eastern Australia 1824–1830

The unexpected appearance in the colony of New South Wales of the worst scourge of eighteenth-century Europe in April 1789 was well known at the time. Evidence of other early outbreaks accumulated when explorers and settlers saw elderly pockmarked survivors not only in recently settled districts but also in more distant parts of eastern Australia. Older immigrants, who knew the disease well, also noticed younger Aboriginal people who had survived it, as well as the much older victims of 1789. The survivors, old and young, were recollected in colonial reminiscences by settlers who recognized traces of the sickness.

It was apparent that the disease had recurred not long before exploration in frontier districts. On the farthest frontier, on the north coast of country that later became the Northern Territory, officials and visitors at the new settlement at Raffles Bay between 1827 and 1829 saw many pockmarked Aboriginals, and others at Port Essington after 1838 saw the same evidence. There are fewer records in Queensland than in the older colonies; however, active smallpox was seen by a convict who had escaped from Moreton Bay in 1826, and newcomers recorded clear evidence of its past presence among Aboriginals in the north-east. In the colony of New South Wales, Charles Sturt described smallpox in Aboriginals on his first expedition to the Darling River in 1829 without recognizing it. On his second expedition, in 1830, he observed signs of the same



Key: ● outbreak H hypothetical outbreak

Eastern Australia: smallpox outbreaks 1824–30

Medical officers and military personnel on the Cobourg Peninsula from 1827 to 1828 and at Port Essington in 1838 left records of pockmarked Aboriginal survivors. British observers left records of them after settling in Queensland. Sturt may have seen very recent outbreaks of smallpox in New South Wales and South Australia in 1829 and 1830. German and British newcomers left copious records of survivors after settlement in South Australia. (See also page 137.)

disease among indigenous Australians on the lower Murray. After the colony of South Australia was founded six years later, many German and British immigrants, who were more familiar with smallpox than Sturt was, realized from the ages of pockmarked survivors that smallpox had occurred in the decade before their own arrival. There is more recorded evidence of smallpox before settlement in eastern Australia than might be expected.

What seems to have happened between about 1824 and 1830 suggests there had been a steady southward and south-eastern movement of smallpox through Aboriginal clans from the north coast to both the south coast and the east coast during those years, and there is abundant evidence that it was the same epidemic that moved into the colony of New South Wales between 1828 and 1832. In this chapter, the historical evidence will be presented by regions, from its presumed source of entry in the Northern Territory, to Queensland on both sides of the coastal range, to north-western New South Wales, finally to South Australia and the south coast. Extensive records of smallpox in the colony of New South Wales is presented in a later chapter.

Aboriginal smallpox in the Northern Territory 1824–30

There was definite evidence of smallpox in 1829 in the Top End of the Northern Territory. In 1835 Thomas Braidwood Wilson, a surgeon in the Royal Navy and Member of the Royal Geographical Society, described it in publications. After eight voyages to Australia as a surgeon on convict transports, he was shipwrecked in Torres Strait in 1829, and with some members of the crew rowed 1600 kilometres to Timor. He reached Raffles Bay at the end of June 1829, two years after the settlement was founded, when Captain Barker was in command, and stayed until 28 August 1829. He became familiar with the history of the settlement, including reports by Assistant Surgeon R. M. Davis about the settlement's health, and by an experienced naval officer, Captain Laws. According to Davis: 'Nothing in the form of epidemic, or contagious disease, has been observed, and the greater proportion of the diseases which have occurred are to be attributed to the want of a due quantity of vegetable food'. The settlers' health was good.¹ The visits of

Macassans were also of interest to Wilson, who said they sailed to Port Essington, eastward by Raffles Bay, through Bowen Straits, ‘and proceed, according to their own report, as far as Cape York’. They spoke well of Aboriginals on the coast of the Gulf of Carpentaria, 4 of whom were going to Macassar with them. But Captain Laws said others were hostile to the visitors.²

Davis had seen Aboriginals with bronchitis in the wet season, and both Davis and Wilson had seen people with pockmarks. Davis said: ‘Several of these people have deep circular impressions—on their faces in particular—as if caused by small-pox. From the inability of making myself understood, the nature of the disease which produced these marks is not yet ascertained.’³ But Wilson said:

The natives described, in language, or rather, by signs sufficiently significant, the history of the malady, which they call *oie-boie*, and which appears to be very prevalent among them. It evidently bears a resemblance, both in its symptoms and consequences to small-pox—being an eruptive disease, attended with fever, and leaving depressions. It frequently destroys the eyes, and I observed more than one native who had thus suffered. Mimaloo’s left eye was destroyed by this disease, hence his English name, One-eye, to which he appeared particularly partial. We could not learn whether they used any remedy, except abstinence.

Before he left the settlement, Wilson added that:

Wellington having promised to bring his wife and children to see us, before we left his territory: he said she would have visited the settlement long ere this, had she not been very ill with the *oyie boyie* (smallpox). This excuse of ill health was (as frequently occurs in civilized society) mere pretence.⁴

Other British observers in the new northern outposts at the time were aware that smallpox had occurred among Aboriginals on the Cobourg Peninsula. Captain Barker of the 39th Regiment, the Commandant at Fort Wellington, was the officer who recorded the visits of Indonesian fishing-fleets from Macassar mentioned earlier (chapter 4). He had a list of praus that called at Raffles Bay in the 1828–29 season, when there were 16 deaths among crewmen on nine praus seen at Raffles Bay before they sailed for Macassar on

11 May 1829, and 9 more on five praus that sailed on 17 May 1829. Barker did not refer to Aboriginal smallpox, but like medical colleagues, he would have observed pockmarked Aboriginals.

Commentary

Information from Foelsche's Aboriginal informant, Jack Davis, at Port Essington suggests smallpox had occurred in the late eighteenth century in the Northern Territory, where there were older pockmarked people. Then, decades after the first outbreaks, and soon after they arrived in 1827, medical officers and others at Raffles Bay saw facial pockmarks on the local people. Their written observations clearly establish that an outbreak of smallpox had occurred there only a few years earlier. The way that its victims talked about *oie-boie*, as they called it, left no doubt about the shock of the disease. Noticeable facial pockmarks, Mimaloo's left eye, and the damaged eyes of others, indicated a recent history of smallpox. It had evidently been introduced on the coast before the British had arrived, perhaps about 1824.

After only two years at the settlement, the newcomers had not identified the source of infection with any certainty. However, by 1829 they had discovered that Aboriginal people had close contacts in the wet season with visiting fishermen in the bays and islands around the Arnhem Land coast, in the Gulf of Carpentaria, and as far as Cape York, if Wilson's impressions are any guide.⁵ Smallpox had occurred in South Sulawesi in the 1820s, so infectious visitors could have transmitted it to Aboriginal people who lived with them and worked with them, trepanging at a number of sites between the Cobourg Peninsula and Cape York about 1824.

It is also possible that infectious visitors introduced smallpox again in the 1828–29 trepang season, when the many deaths of Macassans during their six months' voyage to Australia were reported by Barker. It would have affected a smaller number, who had not caught it in earlier years. Wellington's wife, who did not visit the settlement when she was sick with *oie-boie* before the British left in August, might have been one of them. But the disease evidently did not spread at that time, probably because some people in northern populations were immune after previous severe outbreaks. Young

people who recovered in the 1820s, including some with severe pockmarks, were seen by Captain Bremer and others at Port Essington after 1838.⁶

There were no literate observers anywhere else in northern Australia in the 1820s to record incidences of smallpox. It would have occurred in other coastal districts in the Top End and around the Gulf of Carpentaria, and it probably followed the same paths of infection that it had followed in the 1780s, and would follow again in the 1860s. Colonial officials in the nineteenth century never saw Aboriginal populations on the north coast as large as there would have been before smallpox occurred in the late eighteenth century and the 1820s.

Aboriginal smallpox in Queensland 1824–30

Although there were relatively few well-informed observers in Queensland when smallpox occurred in Aboriginal communities in the 1820s, settlers who occupied land in the 1860s saw evidence of its past presence, and heard of it from Aboriginal informants. As the newcomers told Curr in 1879, they were surprised at the number of pockmarked Aboriginals they had seen. South of the Gulf, Edward Palmer said smallpox had affected Cloncurry Aboriginals during the previous half-century. A few of the older pockmarked people Palmer had seen in north Queensland had probably caught smallpox as children before 1830.

Hundreds of kilometres south of the Gulf, on the Barcoo River and the Ravensbourne Creek in west Queensland, a literate Torraburri tribesman, a trooper of the Mounted Native Police, who was born in the locality (probably a Bidyara man), told Curr that ‘smallpox, called *weeteen*, existed in his tribe before he was born, and that four or five persons, all about fifty years of age, bear the marks of it at the present time’. Several hundred kilometres south of the Barcoo, a settler on the Paroo River in 1863 said: ‘there are undoubted signs of smallpox having visited this tribe about thirty years ago . . . it is said to have half exterminated it’. There were about 500 people in the tribe when he arrived.⁷

Several kilometres farther east in the territory of Kanoloo (Garingbal) people at the head of the Comet River, there was: ‘a

cave full of the bones of blacks, who are said by *Kamungela* [an Aboriginal informant] to have died about forty years back of some disease of the nose. Some of the tribe however, have a few marks which it is thought might be the result of smallpox.’ It was said that the tribe numbered about 500 people in 1860 when Europeans arrived.⁸

In the 1930s Queensland author Donald Gunn could remember that:

when I was a boy, many of the blacks over forty years of age were badly pockmarked. This was the case right from Wyaga near Goondiwindi to Pikedale near Stanthorpe, and old hands told me the same pockmarks could be seen in other districts. It seems to me that this disease was introduced into Australia before the whites took possession, and probably played great havoc among blacks. It must have killed them by thousands. I should not wonder that when the whites took possession of Australia they found the aborigines a much reduced race as far as numbers were concerned. Apparently the disease burnt itself out about the time the whites took possession . . . It may be that the small-pox was introduced by . . . people . . . landing on our far northern coastline.⁹

In 1855 when new settlers occupied the country of the Byellee tribe near Rockhampton, ‘a few of them were marked with the small-pox . . . the tribe described it as having visited them about the beginning of the [nineteenth] century, and carried off large numbers. It is called *wanboy*. Their land went from Keppel Bay to Calliope River, and included Curtis Island. In 1855, there were about three hundred of them.’¹⁰

An anonymous informant visited the territory of the Toolooa people on the watershed of the Boyne River, some fifty kilometres from the coast before settlers occupied it. He told the Commissioner of Police at Brisbane that:

Many of the members of the tribe who are over forty years of age bear the marks of small-pox. On this subject the tradition of the Toolooa people is, that about the year 1835 they were visited by the Burnett tribes, who brought the disease and gave it to them. Such great numbers died of it that the survivors were unable to bury them. The Toolooa name for smallpox is *deelum*.

Squatting runs were taken up in 1854, when there were 700 people in the tribe.¹¹ Other tribes affected about that time included the Meerooni tribe, who called smallpox *tingal*. Their territory stretched from Bustard Bay to Rodd's Bay and back to Many Peaks Range. Offshore, Great Sandy or Fraser's Island was occupied by Europeans in 1849, and Curr said smallpox had never reached it. But the Commissioner of Police said there were nineteen tribes, or 2999 people, who all had distinct names for smallpox.¹²

Tom Petrie, explorer and grazier, was known as a friend of Aboriginals, with whom he had close relations after accompanying his parents to Moreton Bay in 1837. In 1859 he married and settled in the Pine Creek district, twenty-six kilometres by road from Brisbane, with assistance from his Aboriginal friends. When his daughter Constance Campbell Petrie published her father's reminiscences of early Queensland in 1904, she related that when he first came to North Pine nearly forty-five years earlier:

pock marks were very strong on some of the old men; they explained to him how the sickness had come amongst them long before the time of the white people, killing off numbers of their comrades. Pock marks they called 'nuram-nuram', the same name as that given to any wart. (From this Neurum Neurum Creek, near Caboolture gets its name.) The scourge itself was 'bugaram' . . . small-pox was something to be spoken of in a whisper and with bated breath. After the advent of the whites, consumption took hold of the race.¹³

In 1819 21-year-old John Baker was transported to Sydney. After a second life-sentence in 1825 he was imprisoned at Moreton Bay. In January 1826 he went bush, was picked up on the river by Aboriginals who fed him, and thought he was 'Boraltchou', the dead son of one of the tribe. He was with them on travels to the Darling Downs, and stayed with them until 1840, when he gave himself up. J. D. Lang, a Presbyterian minister, later recorded his account of smallpox among them:

A variolous disease, somewhat similar to the small-pox, which had occasionally prevailed among the natives in various parts of the older colony, from the very commencement, happened to prevail among the tribe in which Baker was domiciliated, and proved fatal in many

instances. The natives ascribe this disease to the influence of Budyah, an evil spirit who delights in mischief. Baker, however, had been vaccinated, and did not take the disease; for although the medical men of the colony are of the opinion that it is a different disease from the small-pox, that wonderful specific appears to be equally effectual in preventing its access. The natives, however, could not be aware of this, and accordingly observed that Budyah had no power against Boraltchou, and could do him no injury. In this disease, also, the natives practise *hydropathy*, or the *water-cure*, placing the patient in water, just as Preissnitz—the German hydropathist—would recommend.¹⁴

Smallpox was said to have ravaged populations on offshore Stradbroke and Moreton islands, and according to the explorer Landsborough, tribes with territory between the Albert and Tweed rivers south of Brisbane suffered heavy losses from smallpox before and after Europeans arrived in the colony.¹⁵

Commentary

Smallpox occurred twice in north-eastern Australia before 1830, and it certainly occurred about 1824, soon after it was reported in Arnhem Land. Palmer's comment implied that it was known on the Cloncurry by then, probably after an introduction in the Gulf, where Indonesian fishermen in Macassan fleets had collected and processed trepang for decades. From this presumed point of entry, it is likely that smallpox spread up the Cloncurry River, which became a major focus of infection. Fewer cases would have occurred in sparse and scattered populations in the semi-arid interior of Queensland west of the coastal range, where transmission of infection would have been prolonged, and, if it occurred at all, smallpox would have spread slowly.

However, after contacts on waterways that linked these small populations, there was an outbreak around the confluence of the Barcoo River and the Ravensbourne Creek in Torraburri land about the mid-1820s, when a few old pockmarked tribesmen known to a police trooper had suffered when they were children. There is other scanty evidence of the presence of smallpox several hundred

kilometres farther south, in clans on the Paroo about 1827, a year or so before it reached clans on the Darling, where Sturt would see its results. From centres of infection in west Queensland, smallpox spread on the slopes of the Great Dividing Range, and with contacts in denser populations, it broke out around Goondiwindi, Pikedale and elsewhere in south Queensland, as Gunn recalled. His account suggests incidence and mortalities were higher there than in west Queensland.

Smallpox had apparently occurred east of the Great Dividing Range in the late eighteenth century in coastal districts where there are fragmentary records of its past presence. Byellee people near the site of Rockhampton probably caught it then, not in the early nineteenth century, as Curr believed, because there are no records of it in Australia at that time. About 1860 Tom Petrie knew old Aboriginal men who said the sickness had come long before Europeans arrived. Those who had it as children in the 1780s would have been over seventy in Petrie's day. Landsborough's account of an epidemic before settlement may also refer to the late eighteenth century. From inland centres of infection, smallpox again spread east in the late 1820s, reached the upper reaches of rivers in the coastal range, and spread down to the coastal strip, where newcomers would have seen it. It probably occurred a hundred kilometres from the sea on the Burnett River about 1828, then spread to the Toolooa clan on the Boyne River, who suffered severely after the Burnett clans visited them. Curr's anonymous informant said this happened in 1835, but it would not have taken so long to spread from the north coast. It would have reached south Queensland a year or two before reaching the east coast of New South Wales.

Smallpox spread readily in riverside clans and coastal populations connected by canoe traffic, for they met frequently to exchange goods and fulfil social obligations. The severe illness of many would have caused some to move out of infected districts, and so to spread infection. Outbreaks on the coastal strip were mentioned in records by Lang, the Commissioner of Police, Landsborough and others, and the impact of smallpox in these relatively dense populations in east Queensland was apparently more severe than it was in west Queensland. Smallpox was the first cause of depopulation described there in Curr's day, and later by Radcliffe-Brown. After it occurred for the second time in forty years, the way was open for

settlement. There was no smallpox among Europeans in Queensland, because few had contact with infectious Aboriginals, and any who did have contact would have been vaccinated, as Baker was. There are no records of Aboriginal vaccinations, or distributions of supplies to the sick, which happened in outbreaks in the older colonies.

Aboriginal smallpox in New South Wales 1828–30

When Captain Charles Sturt of the 39th Regiment led an expedition to the Darling in 1828–29, he had been in Australia only eighteen months. Other members of the party included native-born explorer Hamilton Hume, 3 soldiers and 8 convicts. After leaving the government station at Wellington Valley in November 1828, they followed the Macquarie and Bogan rivers to the Darling River, then traced the Castlereagh River to the Darling.¹⁶

Near the Bogan–Darling junction on 2 February 1829, Sturt saw well-trodden paths, and followed the Darling to the south-west, passing seventy huts, each big enough for 12 or 15 men. The travellers also saw large, well-made nets for fish and game. Aboriginals who saw them were alarmed, but led by an elderly man who attracted Sturt's interest, they advanced. Sturt said:

from the moment I approached him, I thought there was a shade of anxiety upon his brow, and an expression of sorrow over his features, the cause of which did not originate with us . . . he seemed to claim at once our sympathy and our protection, although we were ignorant of that which oppressed him . . . As his tribe gathered round him, the old chief threw a melancholy glance upon them, and endeavoured . . . to explain the cause of that affliction, which . . . 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. Nothing could exceed the anxiety of his explanations, or the mild and soothing tone in which he addressed his people . . . We now discovered the use to which the conical substance that had been deposited with such unusual care in one of the huts, was applied. There were few of the natives present who were not . . . marked with it, and it was no doubt, indicative of mourning.

The explorers saw few people before returning upstream in February. Later they saw a large number of Aboriginals, who had visited the camp during their absence.¹⁷ When Sturt returned, 70 men, women and children came to see him. They seemed to be strangers, and several carried fire-sticks, which he connected with the disease, and others had 'violent cutaneous eruptions' all over their bodies. Those they had seen on the Darling were few in relation to the size or number of their habitations, and Sturt said it was evident their population had been thinned.¹⁸ Hume later reported that the Aboriginals were suffering severely from this eruptive malady, that numbers had died, and many more were still dying from its virulence.¹⁹

On 12 February the expedition reached their base camp at Mount Harris and left again for the Castlereagh River, arriving on 10 March. Later in March, Aboriginals surrounded Sturt at the Castlereagh–Darling junction. He reported that 'the natives were dying fast, not from any disease, but from the scarcity of food and, should the drought continue . . . they may become extinct'. In April he observed that the lower tribes also were starving.²⁰

The next reports of Aboriginal smallpox on the Darling River came when Thomas Mitchell, Surveyor-General, led an expedition in March 1835. He traced the Darling from where Sturt left it, nearly as far as its junction with the Murray River. From Orange, he travelled north-west to the Bogan, down the Bogan to the Darling, and down the Darling to the Menindee district. He returned after charting a section of the Bogan and 480 kilometres of the Darling.²¹ A Scot, he was more familiar with smallpox than Sturt was, and referred clearly to Aboriginal smallpox and its survivors on his first two expeditions. He did not mention smallpox survivors in south-eastern Australia during his third expedition in 1836. When Mitchell met Aboriginals on the Darling near Bourke on 28 May 1835, he said:

The party consisted of four men and a boy, followed by seven women and children . . . Most of them had had the small-pox, but the marks were not larger than pinheads. I found, that they had either seen or heard of Captain Sturt's party . . . It seemed to me that the disease

which it was understood had raged among them . . . had almost depopulated the Darling . . . these people were but the remains of a tribe.²²

Farther down the Darling on 23 June, Mitchell met 17 men, almost all marked with smallpox. On 3 July 2 or 3 who followed a convict servant known as ‘Dr Souter’ to the camp were deeply marked with smallpox. On 6 July he saw children and adults.

These natives, as well as most others seen by us on the river, have 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 . . . This is what is termed, according to Souter, the ‘confluent’ small-pox.²³

Near Lake Poopalloe, he observed that:

On this hill were three large tombs of the natives . . . Each being about five feet high; and on each of these were piled numerous withered branches and limbs of trees, no inappropriate emblem of mortality. I could scarcely doubt, that these tombs covered the remains of that portion of the tribe, swept off by the fell disease, which had left such marks on those who survived.

A village of strongly made huts was near the tombs:

One group was in ruins, but the more modern had been recently thatched with dry grass . . . there was one unusually capacious hut, capable of containing 12 or 15 persons . . . In this hut there were many bundles of wild flax, evidently in a state of preparation for making cord or line nets . . . Each bundle consisted of a handful of stems, twisted and doubled over, but their decayed state shewed that the place had been long deserted. A great quantity of the flax, in that state, lay about the floor, and on the roof of the hut.²⁴

He concluded that: ‘The population of the Darling seemed to have been much reduced by small-pox, or some cutaneous disease, which must have been very virulent . . . and the violence was indeed apparent in the marks on those who survived.’²⁵

Early in the twentieth century K. Langloh Parker said the Yualyai (Euahlayi) tribe on the Narran River, near the Darling, still remembered the smallpox outbreak that Sturt had seen:

Ghastly traditions the blacks have of the time when *Dunnerh-Dunnerh*, the smallpox, decimated their ancestors. Enemies sent it on the winds, which hung it on the trees, over the camps, whence it dropped on to its victims. So terror-stricken were the tribes that, with few exceptions, they did not stay to bury their dead; and because they did not do so, flying even from the dying, a curse was laid on them that some day the plague would return, brought back by the *Wundah* or white devils.²⁶

Commentary

On his first expedition, Sturt, who would have been vaccinated as a child, and would not have seen active smallpox either as a school-boy at Harrow or as an army officer, apparently did not recognize the unfamiliar disease among the people of the new land early in 1829, although he must have seen pockmarked people in Europe. He was accompanied by native-born Hamilton Hume, who had no previous experience of smallpox at all. When Sturt saw ‘violent cutaneous disease’ in Bagandji (Barkindji) people on the Darling in February 1829, it is likely that he was seeing people who had recently recovered from smallpox. His description does not suggest any other disease known to Europeans at the time. There was a controversy in 1877 in the Melbourne *Argus* about whether the disease he saw was smallpox. However, it did kill and scar its victims, and as Sturt guessed, marks of white clay, which he saw on the Darling, were used during mourning. Furthermore, his account reflects the behaviour of smallpox in a number of other ways.

As we know, smallpox had not occurred in the south-east for forty years, and the melancholy old man on the Darling, who did not get smallpox himself, may have been immune after an attack in the 1780s. Sturt found out more about the disease from him than from the people he encountered on his second expedition. The young men with him had recovered, as children and young people with it usually did. It is likely that the strangers Sturt noticed had fled from

a neighbouring district, which often happened when people were new to smallpox. If the tribes Sturt saw were starving, the cause was not only the drought, which he blamed, but a shortage of food caused by the simultaneous sickness of the food-gatherers. Inadequate supplies of food and water during severe illness exacerbated the effects of smallpox in indigenous populations in Australia as well as among Native Americans.

Mitchell, who may have seen smallpox in Scotland before emigrating in 1827, first identified the Aboriginals' disease when he saw some with active smallpox outside Sydney in 1831. He saw many people who were noticeably pockmarked on his expedition to the Darling in 1835.²⁷ His description of smallpox differed from Sturt's, in that he saw the victims' pockmarks six years after Sturt saw the lesions of recent infection, and he noticed slight differences in the look of the scars. As Mitchell said himself, he might have seen pockmarks caused by confluent smallpox, and he would also have seen pockmarks exacerbated by bacterial infection. He may also have seen a few older victims, whose pockmarks had faded decades after attacks in the 1780s, when they were children. He found that infection had spread several hundred kilometres down the river, and related it to severe mortalities, corroborating the impressions of Sturt and Hume. Sturt and Mitchell both described large tombs and depopulation, which they associated with the disease. Early in the twentieth century K. L. Parker's Aboriginal informants described the epidemic, confirmed its devastating impact, and associated it with the arrival of the Europeans.

Aboriginal smallpox in New South Wales and South Australia 1830

Sturt saw more evidence of Aboriginal smallpox on his second expedition. He left Sydney on 3 November 1829 to trace the Lachlan–Murrumbidgee river system, with George Macleay, 4 members of his first expedition, several soldiers and convicts.

By the end of November they had reached the Murrumbidgee near Jugiong, where an old Aboriginal described a large river, with good waters, that flowed to the south-west, and its banks were not peopled.²⁸ In mid-December, Sturt said:

There cannot . . . be a numerous population on the banks of the Morumbidgee, from the fact of our having seen not more than fifty in an extent of more than 180 miles. They are apparently scattered along it in families . . . our information . . . was very gradually arrived at . . . as we fell in with the successive families.

Later in December he saw only 90–100 people on the river, and said disease and accidents caused premature deaths among them. In a crowd of 60 visiting the camp, one old man had a club foot and another was blind, but there were young men, and many children with the women.²⁹

On 6 January 1830 the explorers sailed rapidly downstream in a whaleboat, saw over 100 people, went past reedy banks and sighted a river junction. In mid-January they entered a broad new river. They saw no Aborigines, but when they camped on the left bank on 17 January, armed Aborigines approached on the right bank. Some crossed to Sturt's camp, stayed overnight, and went down the bank, parallel with the boat, to meet over 80 men, women and children the next morning.³⁰ These people lived in long, large huts like those seen on the Darling. On 19 January a large group armed with spears was on the right bank, and others approached on the left bank. Numbers, weapons and noise were intimidating, but after a long consultation, some visited the camp. Three old men stayed all night, and 150 watched the boat leave. Sturt said several people he saw were disabled by leprosy or a similar disorder, and 2 or 3 had entirely lost their sight. One of the guides who stayed with Sturt's party was exceptionally strong and tall. No others were seen on 21 or 22 January.³¹

On 23 January the guides left early. Fourteen kilometres down the river a vast number of blacks, painted and armed, threatened the explorers, who saw their spears quivering. 'Some who had marked their ribs, and thighs, and faces with a white pigment, looked like skeletons, others were daubed with red and yellow ochre.' Sturt said the women 'appeared to have had a bucket of whitewash capsized over their heads'. When they ran onto a sandbank and advanced to the water, Sturt was apprehensive: 'with such fearful numbers against us', he doubted the outcome. However, others arrived on the left bank, and the tall strong man intervened to prevent a battle.

Sturt's party emerged confused; then saw a new river coming from the north. They landed on its right bank, and 600 recently hostile people swam over to Sturt.³²

Once past the new river, which was the Darling, Sturt and his party camped on the left bank, saw more huts, and were joined by guides. In oppressive heat on 24 January Sturt recorded: 'Our intercourse with the natives had now been constant. We had found the interior more populous than we had any reason to expect; yet as we advanced into it, the population appeared to increase.' He now saw 'the most loathsome of diseases', and said some victims were so young that they must have been born with the disease. He believed the disease could not have come from the colony, because only the midland tribes were infected. 'Syphilis raged with fearful violence; many had lost their noses, and all the glandular parts were considerably affected.'³³ On 26 January the boat passed country full of lagoons and thickly populated. Huts and footpaths were used by many. 'What the actual number of inhabitants was it is impossible to say, but we seldom communicated with fewer than 200 daily.' They passed a small river, which Sturt named the Rufus, which joins Lake Victoria to the Murray forty or fifty kilometres downstream from the Darling junction. They landed at noon and met 250 people. It was 104°F.³⁴

Past the junction of the Murray and the Lindesay rivers in South Australia on 27 January 1830, curious people crowded around Sturt, and he again recorded that 'disgusting diseases raged among them'. Several days later, when 270 people met the explorers, including some lame and some blind, he doubted that several old men lying on the river bank would be alive twenty-four hours later. About two hundred kilometres from the sea on 31 January, Aboriginal guides introduced them to a large assembly of people.³⁵ The journey continued, and on 3 February 1830 Sturt remarked on '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.' The explorers then passed a tribe weak in numbers on the left bank, followed by one of over 200 people on the right bank, whose women appeared to be prolific. They were near the sea.³⁶

Sturt met large groups on 6 and 7 February, then landed ‘close to the haunt of a small tribe of natives, who came to us with the most perfect confidence, and assisted the men in their occupations. They were cleaner and more healthy than any tribe we had seen, and were extremely cheerful, although reserved in some respects.’ The expedition reached the end of the Murray and the south coast on 9 February 1830.³⁷ They began the return journey in February, and reached Sydney on 25 May 1830.³⁸ Sturt returned to England and in 1833 published *Two Expeditions into Southern Australia During the Years 1828, 1829, 1830 and 1831*.

Commentary

In 1830, when Sturt described diseases he saw in Aboriginals, he was in a whaleboat on the Murrumbidgee, Lachlan and Murray rivers for just over a month, and was not in a position to observe closely the many people he passed on the river banks. An astute and reliable observer, some of his descriptions of people he encountered on his journey down the rivers indicate that some had survived smallpox at a much earlier time. In December 1829 he saw an old Murrumbidgee Aboriginal who was blind, which may have been caused by smallpox in the 1780s. Another had a bone deformity, which very occasionally followed an attack of smallpox in childhood. Before long, Sturt saw several more blind people, who may also have had smallpox, though blindness was often caused by trachoma.

Sturt was ill-equipped to judge how Aboriginals were affected by European diseases that were relatively unfamiliar to him, or by unknown and disabling indigenous disease, such as endemic syphilis, which was not recognized by European observers for another century. When he was on the Murray in 1830, Sturt said Aboriginals suffered from ‘loathsome diseases’, and named leprosy and syphilis. He had probably seen leprosy in Europe, but he did not see it in Australia, because it was not introduced for another forty years, when it was brought to the Northern Territory and the Kimberley with Chinese and Pacific Island labour.³⁹ In the case of syphilis, Sturt said he saw it in Aboriginals while he was on his second expedition.

He clearly meant venereal syphilis, which was prevalent in Europe, but he was unable to account for its presence in the colony. He was not to know that there was an ancient, closely related, non-venereal indigenous disease in Australia. Understandably, his account of symptoms he saw in Aboriginals echoed what he had heard of the Old World disease from other Europeans. As we know, the belief that Aboriginals suffered from syphilis was common in nineteenth-century Australia.

In the twentieth century, contemporary knowledge of New World diseases was confused by populations with indigenous non-venereal treponemal diseases such as yaws and endemic syphilis, and also by venereal syphilis caused by the closely related spirochete introduced by Europeans. Significant differences in the disease patterns emerged. In the primary stage of yaws, the peak period of infection is from one year to fourteen years, when the distribution of infectious lesions on the face, forearms and legs is apparent. The pattern of infection, and the distribution of the less recognizable lesions of endemic syphilis is similar. In the secondary stage, which is fairly mild in yaws, the affected glands are prominent. A tertiary stage occurs later in life, when gangosa, the destruction of the nose and roof of the mouth, may occur as a relatively rare complication.

Venereal syphilis differs from treponemal diseases in that the primary stage usually occurs in people between twenty and forty years of age. In the secondary stage of syphilis, the affected glands are less prominent than they are in yaws, the lesions are denser on the trunk, shoulders and back, and occur less frequently and for a shorter period of time than in the indigenous diseases. In the tertiary stage, visceral and nerve lesions cause severe damage in syphilis, but are rarely seen in the other treponematoses.

The disease Sturt saw was a disease of all age-groups, starting with young children. It was certainly apparent to Sturt, who observed infectious lesions in young people. He also remarked on glands that were affected, a feature of the secondary stage. He had already noticed disabled people who were lame, and who were in the tertiary stage. Then, while in the arid hot north country on the Murray river banks, he added that ‘many’ had lost their noses.

He probably saw elderly people affected in the tertiary stage, but gangosa is regarded as a rare, if unforgettable complication in other indigenous peoples affected by treponematosis. Sturt knew nothing of the strange indigenous infection, and little more about the closely related Old World disease of venereal syphilis. There are no other written records to suggest that indigenous Australians were affected by the characteristic symptoms of venereal syphilis at that time. In Hackett's opinion, the disease Sturt referred to as syphilis was not sexually transmitted venereal syphilis, it was non-venereal endemic syphilis (treponarid), which occurred in the centre and south of Australia, and in hot arid climates elsewhere in the world.⁴⁰

After Sturt returned to the colony in 1835, he still thought that Aboriginals had syphilis, and obtained a supply of blue pills from a Colonial Surgeon to treat them.⁴¹ It is possible that Sturt mistook an indigenous treponemal infection for syphilis, as he had in 1830. Treatment for syphilis may have benefited people with a related disease. However, it probably died out in the small population that survived after the smallpox epidemic.

In 1833 Sturt did not identify active disease in his narrative, and said less about 'violent cutaneous eruptions'. He may not have seen active smallpox on the Murray, but his accounts of incidents on the way suggest indigenous communities were disturbed by the arrival of newcomers, perhaps also by news of the ravages of smallpox before it appeared among them, and by deaths that would have followed it in neighbouring Darling clans. It was not surprising that explorers who appeared at the time of 'the sickness that killed' were met by armed tribesmen who were hostile. They came close to combat with Sturt's party. Soon afterwards, marks of white clay, which Sturt associated with mourning,⁴² were conspicuous in the crowd he saw at the Murray–Darling junction. By then, the crowd may have included bereaved families. Smallpox was on the Darling a year or more before 1830, when it may have reached the river, as indigenous accounts suggest. It was always patchy, and Sturt's one reference to 'violent cutaneous eruptions' in 1830 may have described the lesions of recent smallpox somewhere on the Murray. It was later widely accepted that the disease had broken out during his second expedition, and the retrospective diagnosis of smallpox was never questioned. By 1838 Sturt himself knew one of the diseases he had seen in people on the Murray was smallpox.⁴³

Aboriginal smallpox in South Australia: the later evidence

When the colony of South Australia was founded in 1836, Aboriginal disease became an important issue, as it was in New South Wales. British and German newcomers, aware of relatively recent smallpox epidemics in their home countries, had more to say about smallpox than other diseases in Aboriginals, either introduced or indigenous ones. They met Aboriginal people who had survived the epidemic of 1830, and asked them about it. Older settlers, who knew the disease well, left reliable records of it. Others speculated freely. One of the young settlers was E. J. Eyre, who immigrated in 1833 and travelled widely in south-eastern Australia in the 1830s, where he would have seen pockmarked survivors of epidemics in New South Wales. When he became Resident Magistrate and Protector on the Murray in South Australia in 1841, he saw many survivors. In his *Journals* in 1845, he wrote:

A disease very similar to the small-pox, and leaving similar marks upon the face, appears formerly to have been very prevalent, but I have never met with an existing case . . . It is said to have come from the eastwood [*sic*] originally, and very probably may have been derived in the first instance from Europeans, and the infection passed along from one tribe to another: it has not been experienced now for many years.⁴⁴

When James Hawker recalled his experiences of settling at Bungaree in 1841, he described ‘Old Malo, my blackfellow’.

When I first met Malo I was surprised to see that his face was pitted in an extraordinary manner, evidently from smallpox. As nearly as one could judge a native’s age, he might be about fifty years old. Questioning him about what had caused the marks on his face, he said that when he was a small boy there was a sickness which killed numbers of the natives, that he had it, and when he recovered these marks were on his face. Dr. De Lisle, of the 90th Regiment detachment, saw him when on a visit to Moorundie, and said that he had undoubtedly had smallpox.⁴⁵

In 1844 and 1845, artist and naturalist George French Angas visited South Australia. In 1847 he published an account of what he had seen.

In the year 1789, the aboriginal tribes of New South Wales were visited with the smallpox, which made dreadful havoc amongst them, and swept off incredible numbers. The natives imagined that it was the infliction of an evil spirit. It was this epidemic of which the natives of South Australia speak: they say it came down the Murray from the country far to the eastward, and almost depopulated the banks of that river for more than a thousand miles. I have myself seen two aged men from high up the Murray, beyond the great north-west bend . . . deeply marked with . . . smallpox.⁴⁶

Later, it was common knowledge that smallpox had occurred on the river about the time of Sturt's visit. In 1877 Thomas Moulden commented.

That small-pox decimated the aborigines dwelling below the great north-west bend of the Murray, I can positively say, and I believe that the period of about 40 years is correct. I have seen many natives whose faces were as deeply pitted as those so frequently met with in England 60 years ago.⁴⁷

Five kilometres below Murray Bridge at Swanport in 1880, Billy Poole and several other old men of about seventy years of age often talked about the great sickness that came when they were young, and quickly killed many people on the river. Billy Poole remembered that 'Long time ago one big sick . . . tumble down all about 'long river; die very quick . . . black fellow big one frightened; all run away'.⁴⁸

In 1841 G. Clapham settled on the Murray where it enters Lake Alexandrina, and reported that among tribes on the river and the lake, he saw many adults deeply pitted with smallpox. The victims described how it attacked and killed when it came down the river to the lake and to the sea, carrying off great numbers. The clans had never recovered from those losses of lives, and had since remained relatively few.⁴⁹

Congregational missionary George Taplin emigrated from England in 1849 when he was eighteen. In 1859 he was appointed to teach Aboriginals on the lower Murray and the lakes, where he administered the Point Macleay mission, and assumed the role of physician. He described Aboriginal smallpox in his study of the Narrinyeri, which was published in 1874.

Then, in 1879, the ethnographer J. D. Woods, who said there was some doubt about whether the Aboriginals' disease was 'the true *variola*', republished Taplin's account of it:

They have a tradition that some sixty years ago a terrible disease came down the River Murray, and carried off the natives by hundreds. This must have been small-pox, as many of the old people now have their faces pitted who suffered from the disease in childhood. The destruction of life was so great as to seriously diminish the tribes. The natives always represent that before this scourge arrived they were much more numerous. They say that so many died that they could not perform the usual funeral rites for the dead, but were compelled to bury them at once . . . I think there must have been more than one visitation of this kind, judging from the age of those who are pock-marked. The Narrinyeri attribute all diseases to witchcraft, consequently they employ certain counter-harms . . . Along the shore of Lake Alexandrina are some large mounds of earth. One of these, at Pultowar, was opened last year, and found to contain scores of human skeletons arranged in rows. These were probably the victims of small-pox.⁵⁰

One of the best-known Aboriginals in the Lake Districts was Louisa Karpeny, or Kontinyeri, of the Narrinyeri tribe, who had lived through the great sickness. Early in the twentieth century she was known on stations where she had worked at shearing time, and she camped on the south side of Lake Alexandrina and elsewhere in the district. She met Sir Edward Stirling by arrangement in May 1911 and told him her history. As a child, with others in her family, she was hidden in the river reeds when soldiers in red coats and a horseman appeared, probably in 1836, when the colony was proclaimed. She remembered the *Maria* murders in 1840, when the *Maria* was shipwrecked and local Aboriginals killed 10 crewmen and 15 passengers who had survived the wreck. She also remembered reprisals in the following year, when over 30 Rufus River tribesmen were killed by an avenging expedition. She was then ten or twelve years of age. Smallpox occurred once in her lifetime, when she was a small child, before the soldiers came. A very strong west wind was taken as a sure sign that the sickness was coming. She escaped it, but she knew about scars, deaths and the failure of traditional remedies. The usual mortuary practices were observed.

One of the people who talked to Aboriginal activist Kevin Gilbert in the 1970s was Val Power from Point Macleay at Lake Alexandrina. 'We call ourselves the river people', she said. 'As a clan there would have been two hundred of us, of close-knit relations on my father's side.' Her name was Valmai Blanche Karpeny, and she carried a passport in the Protection era. She told her family history:

My great-grandmother was Queen Louise Karpeny. She was there when Sturt sailed up the Murray. There's a monument to Sturt at Point Macleay . . . When they come down the river they brought smallpox. That just about wiped them out. There was eight thousand in our tribe. I don't know if you ever heard of the brigantine, the *Maria*? My great grandmother was around then.⁵¹

Louisa Karpeny's aunt, Clulwuyrie, who was some years older, caught smallpox. She, too, was well-known. When she died after a long life, the *Adelaide Register* reported her death on 5 July 1911.

A native woman, whose age was probably more than 100 years, died at Paltaloch on Friday. Residents of 50 years or more believe that she passed the century three or four years ago. She was the last aborigine who was pockmarked and related that she was a grown woman when the epidemic carried off so many of her people. In some cases whole tribes were exterminated. She attributed her cure to having bathed in sea water. The deceased was known to white people by the name of Jenny Pougie, but her native name was Clulwuyrie and she belonged to the tribe known as Wethowthenyeri, which means 'power to regulate the heat'. In the hot weather she had been seen chanting her language to change the temperature during a hot spell. The deceased spoke splendid English, and had many friends in and around the lakes. From her account of the small-pox, the epidemic came shortly after the voyage down the Murray by Captain Sturt and party on Feb. 9 1830. She often spoke of the peculiar noise of wind just before the arrival of the small-pox, which she said came from the east, but Mr A. Redman (superintendent of the Point Macleay Mission) is strongly of the opinion in view of the different directions from which the old natives say it appeared, that what frightened the aborigines most was an earthshock.⁵²

In 1911 G. G. Hacket, an old resident of Narrung, Lake Albert, had seen pockmarked Aborigines in that district when he was a lad in 1864. He thought they would have been aged between fifty and sixty, and they did not recollect their own illnesses, except in a legendary sense. The disease came down the Murray, and was brought by an evil spirit. They said it affected old and young, the dead were buried where they died, and in many cases the sick were abandoned and left in their wurleys.⁵³

Molineux, a secretary of the Royal Society of South Australia, arrived in Adelaide early in 1839, when ‘many of the natives were much pitted with marks which they ascribed to a visitation just previous to the advent of the white men on these shores’. He thought it was curious that the disease ‘which appeared to be so fatal to Aborigines had never been communicated to the settlers’. But officials in Adelaide saw pockmarked Aborigines, and, on 10 July 1839, at the Governor’s command, the Inspector of Hospitals advised the public that ‘Vaccination, as a protection against Smallpox, will be performed for the Native Inhabitants, every Wednesday, from eleven to twelve am, at the Native Huts on the Torrens’.⁵⁴

In his reminiscences of the colony in 1838–39 Captain Dirk Meinertz Hahn said: ‘Smallpox must often rage among them as most of them carry thick scars from it’. In a vocabulary of Aboriginal language in South Australia, published in Adelaide in 1840, the Lutheran missionaries C. G. Teichelmann and W. Schurmann included *Nguya*, a word meaning:

a pustule; the disease of small-pox, from which the aborigines suffered before the Colony was founded. They universally assert that it came from the east, or the Murray tribes so that it is not at all improbable that the disease was at first brought among the natives by European settlers on the eastern coast. They have not suffered from it for some years; but about a decennium ago it was, according to their statement, universal, when it diminished their numbers considerably, and on many left the marks of its ravages, to be seen at this day. They have no remedy against it, except the *ngyuyapalti*—small-pox song, which they learnt from the eastern tribes, by the singing of which the disease is believed to be prevented or stopped in its progress.

In 1846 Protector of Aborigines Matthew Moorehouse compiled a vocabulary of the Murray River language, including *nguyongo*, which meant 'itch itch'. This disease was thought by Europeans to be smallpox, as it leaves pits in the skin if suffered to take its own course. The natives state that it was formerly very fatal, and almost whole tribes have been swept away by it.

In the 1840s when John Adams, an early settler, described his experiences in the colony, he recalled an Aboriginal lad of fifteen, who was 'about the only boy about that age in the Adelaide tribe [Kaurna speakers], all that generation appears to have been taken off by smallpox, all the survivors were very much marked'. South of Adelaide on the lower Finniss in the 1850s, a resident saw many pockmarked Aborigines. A scarred survivor in his thirties told him that when he was little, a big wind came from the east. Pointing to his face, he said 'this one come'. Many were affected and many died.⁵⁵

Ethnologist and telegraph operator F. J. Gillen was transferred to Moonta on the Yorke Peninsula as postmaster in 1899, and he found there was a tradition among Aborigines (the Narrunga or Narrang-ga tribe) that resembled the Aranda tradition of smallpox. An old man told him about an old camping-ground where many victims were buried, but Gillen could never find it. An informant of Curr's at Mount Remarkable, at the head of Spencer Gulf, said Europeans had occupied the territory of speakers of the Nukuna language in 1849, when there were 150 of them. Several were marked with smallpox, called *mingi*, and some died of it twenty years earlier. John Hunter Kerr saw pockmarked Aborigines over fifty years old at Port Lincoln on the Eyre Peninsula in 1840.⁵⁶

Commentary

British and German settlers inferred from the ages of pockmarked people that smallpox had occurred more than once before European settlement. Hawker was informed by the pockmarked Malo, who was then about fifty years of age, that it occurred in the eighteenth century, when he was very young, and again in the nineteenth century. Records left by Angas, Taplin, Hahn, Molineux, Teichelmann and Schurmann support Malo's recollections.

Smallpox had spread from the Darling, where it was active early in 1829, to populations on the lower Murray a year or so later. After contacts between previously unexposed people and infectious people, smallpox broke out and spread on waterways and lakes in southern Australia in 1830. Infection was rampant in denser populations, and many people with pockmarks were later seen by settlers in coastal districts on the Eyre Peninsula, at Mount Remarkable and around Spencer Gulf, on the Yorke Peninsula and around the Gulf of St Vincent, in the Adelaide district, on the Murray below the great north-west bend at Moorundie and Swanport, around Lake Alexandria and Lake Albert, and on the south coast. Sickness and deaths in infected neighbourhoods often caused people who were still well to flee, and the smallpox infection thus spread still further when they too became ill.

A wide range of evidence from Aboriginals and Europeans shows that smallpox was already active in southern Australia early in 1830, and that in the following months a series of outbreaks occurred in an area of about 4000 square kilometres. It was probably active in places near the lower Murray before Sturt was on the river in January and February 1830, but his account indicates that smallpox had not yet reached several large clans, or the small, healthy and cheerful clan encountered near the south coast and the termination of the Murray. Oral evidence from Val Power, her great-grandmother Louisa Karpeny and great-aunt Clulwuwyrrie is consistent with outbreaks on the Murray just before and soon after Sturt appeared. Accounts by Europeans of pockmarks after 1836 suggest smallpox occurred less than a decade earlier.

As we would expect, pockmarked survivors consistently said they had suffered as children or young adults. The Finniss River survivor, a man in his thirties in the 1850s, had caught smallpox as a young boy. Billy Poole and the old men at Swanport, who were about seventy in 1880, had suffered as boys or young men in 1830. Pockmarked survivors at Lake Albert in 1864 had survived as children over thirty years earlier. In the Lakes District on the lower Murray, Clulwuwyrrie caught it as a young woman in about 1830, as her memory of Sturt, her recovery from smallpox, and settlers' estimate of her great age in 1911 all suggest.

The incidence of smallpox was high in well-endowed riverland and coastal districts in eastern South Australia, in populations that were dense in the Australian context, where there were many previously unexposed people. After frequent contacts, most had been exposed, and, as early settlers said, most had caught the disease. According to Teichelmann and Schurmann, Aboriginals themselves said the sickness was universal. In the circumstances, mortalities were inevitably high. Louisa Karpeny and Clulwuwyrrie owed their recovery to their youth. However, many others over twenty years of age would not have survived.

Smallpox usually killed many under five years, and Adams said it had killed young children among Kaurna people at Adelaide, except the one young survivor he knew in the 1840s. There would have been more fatalities among women than men, and among the very old only a few immune would have survived. Settlers who later talked with Aboriginal people who spoke English were unanimous about the great number of victims, and this opinion was still common at the end of the nineteenth century. The disposal of the dead was difficult, and the burials of victims were associated with mass burial sites. With hindsight, it seems likely that mortalities in relatively densely populated, resource-rich South Australia exceeded 50 per cent in each epidemic of smallpox.

The effects of smallpox were exacerbated by the loss of family members, by shock, distress and social and economic disruption. Young survivors, such as Billy Poole at Swanport, remembered a time of great fear. Louisa Karpeny and her aunt, and the Finnis River victim, associated the arrival of smallpox with very strong winds and, if Redman was right, with an earthshock. Angas and Hacket found the belief that smallpox was caused by evil spirits was common. Taplin thought Aboriginals believed it was caused by witchcraft, and used counter-charms against it. From the eastern tribes, the South Australian clans learnt that the singing of the smallpox song would prevent it.

Newcomers were soon aware of recent depopulation among Aboriginals. Angas asserted in 1847 that smallpox had depopulated the banks of the Murray for more than 1600 kilometres. In 1841 Aboriginals on the Murray at Lake Alexandrina told Clapham they had never recovered from the loss of lives when smallpox attacked

them, and had been relatively few ever since. Several decades later, Taplin also described extensive depopulation in tribes that were once far more numerous. In 1911 settlers recalled that Clulwuyrie, the last of the pockmarked survivors of 1831, had said that in some cases whole tribes were exterminated. Val Power estimated that there were 8000 in her tribe of river people at Lake Alexandrina, before smallpox ‘nearly wiped them out’. The history of the Karpeny family, which is remembered to this day, is more accessible than are the histories of some other South Australian families.

Conclusion

These records illustrate the spread of smallpox between 1824 and 1830 from northern Australia, through the Murray–Darling Basin in the interior, to southern Australia and the south coast before European settlement. The presence of smallpox north of the Tropic of Capricorn was first underestimated by E. M. Curr, who said smallpox was introduced on the east coast. In 1983 Butlin, who disregarded Aboriginal connections across the continent and their relevance to the spread of smallpox, said the suggested movement of smallpox from north to south before settlement was ‘highly suspect’.⁵⁷ However, we now know that Aboriginal smallpox was seen by reliable observers on the frontiers, and moved south from the north coast into eastern Australia between 1824 and 1830. In the next chapter, we shall find that Aboriginal smallpox was not introduced on the east coast of New South Wales, but occurred there at the end of 1831, after spreading out from the interior three years after Sturt first saw it in the Murray–Darling Basin. We will then consider records of Aboriginal smallpox in the 1860s, which also followed northern introductions.

The Aftermath

Smallpox outbreaks in eastern Australia happened well before European settlement in the Northern Territory, Queensland, north-eastern New South Wales and South Australia. Within a decade, and before Aboriginal societies had recovered from severe mortalities and the shock of virgin soil outbreaks, the pastoral invasion spread

rapidly outside south-eastern Australia, through the best grasslands of Queensland and South Australia. The colonists brought European diseases, and the one most likely to be transmitted to people who had recently survived or escaped smallpox was tuberculosis, which, like smallpox, was passed on by aerosol at a short distance.⁵⁸ There is no clear evidence that it occurred in early British settlements at Raffles Bay or Port Essington between 1824 and 1849, or that Aboriginal people in the Northern Territory were affected before the 1870s.⁵⁹ However, closer contacts between newcomers and Aboriginals in the settled districts caused its frequent transmission, and tuberculosis survived in small populations.

By about 1840, tuberculosis was present in the small European population in southern Queensland, where it infected families by contact and proximity, and it was a major cause of death there in the second half of the nineteenth century. The infection spread to Aboriginal people who had contact with Europeans, it was present in some Aboriginal communities by mid-century, and later in the century it was entrenched, when according to Tom Petrie, consumption took over the whole Aboriginal race. Whereas previously ‘natives lived to a good old age, one would hardly see any old people—their remarkable freedom from sickness seemed to disappear’. He added that they were great believers in the curative powers of the dugong. Petrie had seen sick Aboriginals, ‘unable to walk, apparently in consumption, carried carefully to the mouth of the Brisbane River, and there put into canoes’, and taken to fishing-grounds where dugong were being caught. For some time they would live on dugong, which were then plentiful, and were rubbed with dugong oil. They were then said to be stronger. The oil was used medicinally in Queensland.⁶⁰

In the 1840s in South Australia, Eyre was convinced that diseases introduced by Europeans were the chief cause of the Aboriginal demise apparent in his day. He noticed that some Aboriginal people spent their time ‘in the dwellings of the sick’, and he said ‘very many were in a dying condition’.⁶¹ At that time, tuberculosis was a major cause of sickness among newcomers, and for decades it caused deaths in depleted populations of smallpox survivors who had close contact with settlers; for instance, at Mount Remarkable and at the Point McLeay and Point Pearce settlements.⁶² Apart from

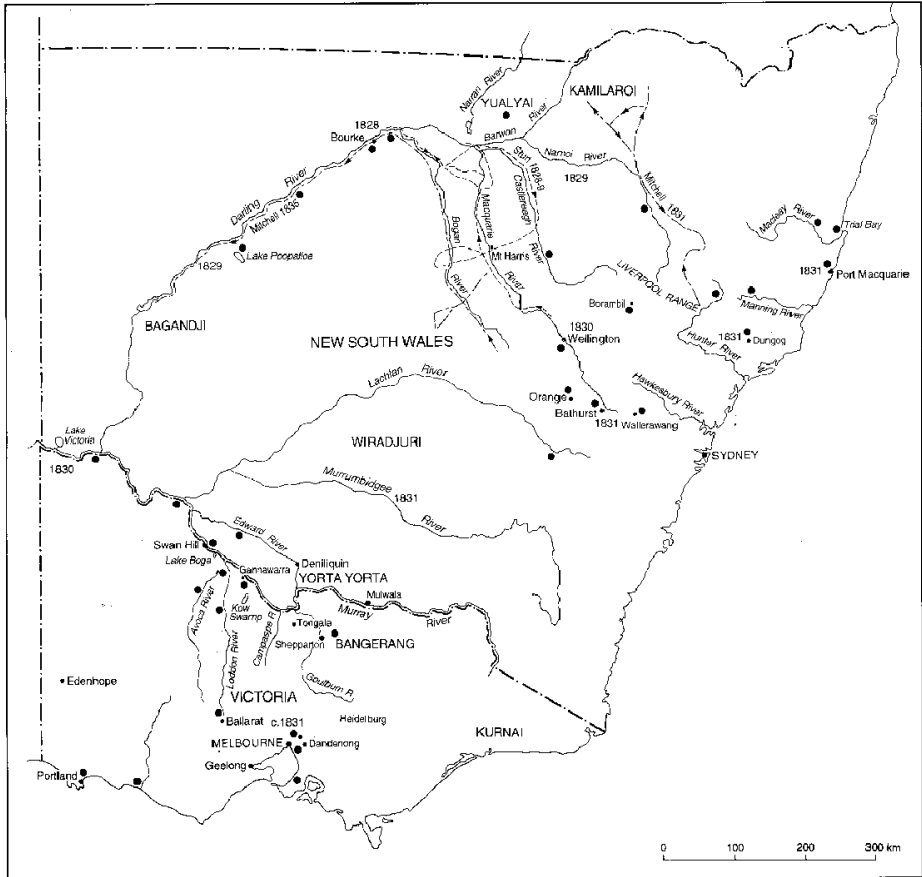
the reprisals that followed the *Maria* murders in 1840, it was the onslaught of smallpox, followed by tuberculosis in newly settled eastern South Australia, that greatly restricted Aboriginal resistance to settlement by Europeans. Newly invaded Aboriginal populations offered surprisingly little sustained opposition to pastoralists, except in central Queensland, where searing black resistance occurred at Eurombah, Hornet Bank and Cullinlaringo⁶³ some thirty years after Aboriginals experienced smallpox, and before sparse European populations north of Brisbane had transmitted tuberculosis to them.

The Colony of New South Wales 1828–32

New South Wales north of the Murray

Old settlers remembered smallpox at Port Jackson in 1789, and Aboriginal survivors with pockmarks were still commented on by European visitors in the 1820s. Early visitors found the colonists healthy, and were surprised by the absence of smallpox and measles. Nevertheless, soon after arriving at Sydney in 1827, John Mair, who was an Assistant Surgeon in the 39th Regiment, found it hard to believe that the colony of New South Wales was safe from smallpox. He had noticed pockmarked Aboriginals, and was apprehensive about what would happen to unvaccinated children if smallpox visited the distant colony again. By 1830 he had obtained a fresh supply of vaccine from England. He advertised that vaccination was available daily in Macquarie Street, and invited applications from country districts for vaccine. He had few responses, because at that time most colonists had no idea that smallpox had broken out in far north-western New South Wales, and they were complacent.¹

From what we already know about smallpox on the distant frontiers, it is clear that by 1830 Australia's remarkable freedom from 'the most dreadful scourge' was at an end. Governor Darling may have been aware of this in 1829, when officials from the Raffles Bay settlement returned, and Sturt came back from his first



Key: ● outbreak - - - - Mitchell's route Sturt's route

South-eastern Australia: smallpox outbreaks 1828–32

From far north-western New South Wales, where Sturt saw Aboriginal people recovering from smallpox in 1829, and Mitchell saw pockmarked survivors in 1835, smallpox spread through the Murray–Darling Basin between 1828 and 1832. Settlers saw active smallpox and pockmarked survivors at Wellington, Orange, Bathurst, Wallerawang, Trial Bay, Dungog, Port Macquarie and the Liverpool Range in 1830 and 1831. It also spread to the south coast in South Australia and Victoria, where pockmarked survivors were seen throughout the nineteenth century.

expedition. Soon after his expedition in May 1830, there were other reports that smallpox had spread in the colony of New South Wales, which at that time included what later became the state of Victoria. In the next few years, escaped convicts and early settlers occasionally met Aboriginals who were terribly sick. The overseer of an estate observed some cases on the Castlereagh River; escaped convict Clark had seen them on the Namoi River; Parson Tom's family noted others when they settled in the Orange district in 1830; and new settlers in New South Wales later recorded signs of recent smallpox in Aboriginal people.

In the second half of 1831 there were vague reports about a disease raging among Aboriginals in the Bathurst district; and in August the *Sydney Gazette* published a letter asking if smallpox was prevalent there. We know that when the surveyor Titman caught it from sick Aboriginals outside Bathurst, medical graduate George Busby, like many other practitioners, mistook it for chickenpox, and that James Bowman, Inspector of Colonial Hospitals, who did not see the patient, agreed with him.² However, thirty-two kilometres from the Post of Bathurst, in August, 4 members of an Irish family at Emu Plains caught smallpox after contact with sick members of an Aboriginal family, and their youngest child died. Army Surgeon Alexander Imlay, who was in the district, met a settler who had encountered some sick Aboriginals, and thought they had smallpox, which he had seen in Scotland. Imlay realized that the disease that had killed the child was the same disease the settler had seen in Aboriginals, and in September he alerted Mair. The Aboriginals' sickness was then identified by more people who had seen smallpox in unvaccinated people in Scotland and England.³

Governor Darling chose John Mair to conduct an enquiry into the outbreak, and he went to Bathurst on 9 October 1831. There were no new cases, but he investigated all the European cases he could. Mair established their histories of contact with sick Aboriginals, traced the epidemic from the interior in mid-1830 to the east coast at the end of 1831, and speculated about its distant origin. Victims and observers described smallpox accurately, and Mair submitted his report to newly appointed Governor Bourke and the Executive Council on 3 January 1832. Bowman, who was present at the meeting, said it was not smallpox. The disagreement was

acknowledged, vaccination was to be performed by colonial surgeons for those who wanted it, and Aboriginals were to be induced to submit to it.⁴

By then, active smallpox was present in Aboriginal communities in the colony of New South Wales. Virgin soil outbreaks had already occurred in indigenous populations that were uniformly susceptible, with the exception of previously exposed older people who had recovered from it in 1789. Between 1829 and 1832 smallpox had swept through the Murray–Darling Basin. It probably reached the south coast of both South Australia and Victoria in 1830, but it did not reach the east coast of New South Wales until the end of 1831. It ravaged many clans for the second time in forty years. When it was all over, pockmarked people were seen almost wherever the settlers went in the south-east.⁵

In 1834 visiting medical practitioner George Bennett, who had seen Mair's report, published extracts from it in *Wanderings in New South Wales*, but not many later writers knew about Mair's work. The interested layman E. M. Curr had heard of Mair, without having seen his report. Butlin, who cited Curr, did not share his opinion that smallpox was endemic from 1789 to 1845, and, as we know, he speculated about an introduction of smallpox on the east coast in 1828. Without Mair's report, he was not to know that smallpox had spread from the interior to the east coast, not from the coast to the interior. Records of Aboriginal smallpox in the colony of New South Wales, and later records of it in what is now Victoria, are presented and interpreted below.

Aboriginal smallpox in New South Wales 1829–31

Mair relied on credible eye-witnesses. After meeting runaway convict George Clark in Bathurst Jail, he said smallpox

made its appearance among the tribes about the Namoi, if the testimony of George Clark . . . who resided with them for several years . . . can be depended upon; and . . . I cannot conjecture any advantage he could flatter himself with from giving a false account of a destructive and pestilential disease, which if it did exist, he had great facilities of observing, and discernment capable of improving the opportunities with which he was favoured. This shrewd and intelligent fellow, then,

states that the disease proceeded from the North West coast, and spared none of the tribes as far as Liverpool plains, attacking 20 and 30 at a time, none escaping its fury. The King . . . of the tribe with which Clark was naturalized was first seized with the disease and died, as had been predicted by the Kradjee. He had previously been with a tribe situated near the Sea, and . . . may have seen the disease before, although he disclaimed having any but *supernatural* knowledge of it.

Clark said the King died after being immersed in cold water, and only 1 of 4 other victims survived the cold water treatment. Those the Kradjee abandoned were sometimes buried alive.⁶

Another of Mair's informants was Andrew Brown, the overseer of a country estate, who had seen smallpox in Scotland. He had heard of its existence in tribes to the north some time earlier, and he first saw it in 5 Aboriginals near the Castlereagh River, 2 in the incipient and 3 in the more advanced stages. He saw one of them afterwards, with pits like those of smallpox in different parts of his body, and he ascertained that the others had died.⁷

Mair saw the Tom family late in 1831. The unvaccinated younger children had fever and eruptions in July or August 1830, when they lived at Sidmouth Valley near Orange. They had pock-marks, but the older ones had been vaccinated, and did not get the disease. James Tom later wrote to the *Argus*:

In the year 1830 I saw some of the Kings Plains tribe of Aborigines 27 miles west of Bathurst . . . lying in a very pitiable state, suffering then from smallpox . . . recent arrivals from Europe, my parents among the rest, pronounced it to be the veritable smallpox. About 10 years after this I pushed about 150 miles further into the interior, to form a cattle station, the furthest out then formed on the Lachlan, called Boranbill, and there I saw the pock-pits in a great many of the Lachlan tribe, and one stone blind and some partially blind. The blind one told me . . . the blindness was caused by the same thing that caused the pock-pits in the face, which were very abundant.⁸

Andrew Brown told Mair the disease was also noticed at Wellington Valley in October 1830, and continued to affect the

Aboriginals there until December. They blamed Captain Sturt for its introduction, were most alarmed about it, and anticipated some grievous calamity, such as a great fire and flood, which was predicted by one of their sages, and which would come from Mount Harris and destroy them.⁹ Vocabularies of the Aboriginal language spoken at Wellington Valley included words for smallpox (*danna-danna*) and pockmarks (*gulgog-gulgog*). In 1839 missionaries told members of the United States Exploring Expedition that, according to their Aboriginal informants, *Darrawirgal* is a brother of *Bai-amai* and lives in the far west. He sent smallpox, which made such ravages among them, because he was vexed for want of a tomahawk; but they now suppose that he has obtained one, and that the disease will come no more.¹⁰

Arthur Rankin, a settler in the Bathurst district, first observed the disease in the tribes near the Lachlan River about the beginning of April 1831, and thought it was communicated by the Wellington Valley Aboriginals late in 1830.¹¹ Another member of Rankin's family had vaccinated several Aboriginals three years earlier; and when their father died of smallpox in 1831 they did not get it. When vaccinations failed, Rankin and his friend Grant variolated some Aboriginals, and another Grant inoculated himself. Of the inoculated Aborigines, 10 had a slight disease, and were restored to health. Others then wanted it done.¹²

Andrew Brown did not see any more cases of smallpox until the beginning of August 1831, when it appeared in 3 Aboriginals, who had been in touch with some who had arrived from the Lachlan, and who had recovered only a short time before they travelled. These men said the disease had been raging in their country, and several had died of it. Some of the Wallerawang Aboriginals, convinced of the contagious nature of the disease, fled to Emu Plains to escape infection; but 3 of them, who afterwards returned, were seized with it.¹³

Imlay was in the Bathurst district in August 1831, and Brown told him mortality among Aborigines from smallpox was very considerable, and several had fallen victims to the violence of the fever before the eruptions appeared. They were sadly alarmed, and fled from the parts of the country where it prevailed. At that time, Imlay himself had not seen any people with smallpox, except 2 boys who

had just recovered from an attack, and who were so distinctly marked that he had no doubt of its genuine character. They said several had died in this neighbourhood, and at Wellington and on the Lachlan great numbers died, and many were still sick. Later, Imlay said the victims he had seen 'were so distinctly marked, and gave so characteristic an account of the Origin and progress of the Disease, believed to be Small Pox, that no doubt remains upon my mind of its identity'. The sick were separated where possible from the rest of the tribe, who accepted vaccination when its advantages were explained, and he vaccinated as many as possible. He told Bowman: 'Measures ought to be adopted without delay to extend Dr Jenner's invaluable discovery, and arrest the progress of so destructive and loathsome a Disease among the Aborigines'.¹⁴

Busby notified Bowman about Edward Titman, the member of a surveying party whom he had admitted to Bathurst Hospital in August 1831 and treated as a case of *varicella* (chickenpox). Busby said Titman had smallpox when he was young, and ought to have been exempt from it. However, he was exposed to the Aborigines' disease while living in the same house as one who was sick, and who 'when convalescent but still in a State of debility, retired from the house . . . and was found dead two days afterwards, from . . . the Severe Cold of the nights . . . There were also other Blacks with the Disease upon them at the same time.' He said Titman's disease was the same as the Aborigines' disease, 'as far as I could judge from the descriptions of unprofessional observers'.

Busby proceeded with a long description of the symptoms and the victims. Vesicles on the hands and feet contained a bloody serum, and were sometimes so numerous on the victim's upper lip and nose as to become confluent, and small pits were observed. In the milder attacks, 'they are indolent during the eruptive fever, but they continue to move about after the eruption has appeared. Many of those who are more severely affected, and especially if females, are carried from place to place by those who are in health.' However, some who were unable to walk, and were carried by friends as long as they were able, were later left behind and perished from want of food. According to Busby, when the victims were given salts and food such as tea or milk, and protected from the weather, they had without

exception recovered. Nevertheless, he also knew of cases where after a little care of this kind, the patient was recovering, and ‘from an anxiety to resume his rambling habits has joined his tribe, and fallen a victim to this premature exposure’. In the same report, he said he had seen only 2 sick Aborigines, and had not formed an opinion of their disease. He attributed the mortality to their unfavourable circumstances.

Busby thought it would be difficult if not impossible to trace the origin of the disease.

It was spoken of by the blacks at Wellington Valley a considerable time before it actually happened there, and they regarded its approach with dread. It appears to have passed through all the Tribes, and from the account of one Black, it has taken the direction of the Murrumbidgee where it is now committing its ravages. At Wellington Valley there has been no case . . . for the last eight or nine months. On the Lachlan there has been none for the last two months, nor have I been able to hear of it from any other part of the Country for several weeks. And I think I may safely affirm that at this moment [October 1831] there is not a case in the district.¹⁵

When he was at Bathurst, Mair saw the 40-year-old Titman six weeks after his discharge from hospital, and said the disease had passed through the regular stages of smallpox, not terminating until the fourteenth day. Fever was severe, with debility, great restlessness and irritability, requiring opiates. Mair said it was secondary smallpox, modified by a previous attack. He saw many pockmarks, some old and some recent. Mair also saw the Cuddy family, of Bathurst at Emu Swamp. An Aboriginal man and a boy in the neighbourhood had recently died of smallpox. During their illness, Mrs Cuddy had sent them milk and food, which were collected by the sick man’s daughter. After her father’s death, the girl continued to visit the Cuddy family, and 4 of them caught smallpox. Their youngest child, a 2-year-old, sickened on 24 August, had a rash the next day, and died on 6 September after a severe illness, which her parents and a neighbour described. Mair said it was confluent smallpox. The mother and 2 unvaccinated children had mild attacks a week later, and were pockmarked.¹⁶ The European cases he saw in the Bathurst

district left no doubt in Mair's opinion that smallpox was prevalent there, and today Busby's opinion that the disease was chickenpox has only historical interest.

Reports of Aboriginal smallpox from Mair's informants in newly settled districts in the interior of the colony in 1830 and 1831 were soon followed by reports from other observers on the east coast. Mitchell left Sydney on 24 November 1831 on his first expedition into the interior. He followed the Hawkesbury and Hunter rivers, ascended the Liverpool Range and camped at a watercourse beside some Aboriginals. He knew smallpox well, and said they were extremely ill, being affected with a virulent kind of smallpox.

We found the helpless creatures, stretched on their backs, beside the water, under the shade of the wattle . . . trees, to avoid the intense heat of the sun. We gave them . . . some medicine, and the wretched sufferers seemed to place the utmost confidence in its efficacy . . . this distressed tribe were also 'strangers in the land', to which they had resorted. Their meekness, as aliens, and . . . ignorance of the country . . . were very unusual in natives, and excited our sympathy.¹⁷

The same week in November 1831 Captain Smyth of Port Macquarie said 30 Aboriginals from Trial Bay and Point Plomer had arrived there infected with smallpox, and in many instances they were covered with pustules. They said they caught it from a runaway from Moreton Bay named Scarm, whom they captured early in October. Smyth saw him after he recovered, and he had pockmarks. The Aboriginals were to stay across the river, and Smyth asked them about the disease: 'it appears they are not unacquainted with it. Many among them not now infected declare they have had it a long time since.'¹⁸

A month later, on 24 December 1831, Mair and Imlay reported that the runaway from Moreton Bay had supplied information that must carry conviction to the minds of those who still doubted the existence of smallpox among the Aboriginals. The runaway, Richard Scarm, then in the *Phoenix Hulk*, had absconded from Moreton Bay some months earlier and met a tribe at Blackrock River, upwards of 50 of whom were suffering from smallpox, which he had seen in England, and described most accurately. He drank out of the vessel used by those affected, and was later afflicted with the symptoms.

Members of the Trial Bay tribe who apprehended him, then took him to Port Macquarie. Captain Smyth said those who brought him into custody had caught smallpox from him, and it then spread through the tribe. According to Mair: 'his skin bears unequivocal marks of the genuine character and recent occurrence of the disease. He says he never before had the Small Pox or Cow Pock, and we can discover no mark of vaccination.'¹⁹

Summing up his investigation, Mair outlined the more important features of the disease. He described elderly pockmarked people, some of whom were blind, who had smallpox as children on an earlier occasion, and were now immune. He said the facts he had uncovered afforded strong evidence of the contagious nature of the disease: 'we have proof of the disease being communicated from one person to another by contact, or through the medium of the air (most probably only at short distance) among the Blacks, evidence as cogent as we can expect to have in such a matter'.²⁰ Corroborating his opinion, he mentioned the entire exemption of one family to the eastward of Mr Walker's Cattle Station, who were at enmity with members of the adjoining tribe who were then suffering from it, but it broke out among Wellington Valley tribes who were constantly in contact with diseased people. This information came from his informant Andrew Brown, who had recently returned from visiting that part of the country.²¹

Mair described symptoms common to all the accounts he had of the disease. Patients lost their appetites, had headaches, chest pains and fever for several days, followed by eruptions on the face that gradually spread more or less thickly over the head, breast and extremities; tongue and lips were involved, and the soles of the feet were often seen to be 'numerously studded'. Eruptions were usually fully developed in twenty-four hours, fever lessened, patients had painful throats and could swallow only liquids. Vesicles developed into pustules within about a week. Those who watched its progress, who had seen smallpox in England, said it was the same. The eruptions were occasionally confluent, and frequently left prominent marks in the skin. Mair had examined these marks in a variety of cases, and they were indistinguishable from the pits of smallpox. The disease lasted from two to three weeks in cases of restoration to health, but even after the eruptions had entirely subsided, and the

disease was over, convalescents were unable to walk for a long time owing to the tenderness of their feet, from which the cuticle had entirely separated. Other effects were troublesome: some lost their eyesight, others had abscesses, hideous ulcers, great debility and emaciation.²²

Death generally occurred on the third day. Secondary fever was rare, because there were early fatalities, and some died before eruptions appeared. Clark described severe disease, followed rapidly by death, in tribes north-west of the Liverpool plains. Eye-witnesses said it proved chiefly fatal to adults and old people, seldom to children. Those who had suffered at a former period, indicated by marks on their skin, escaped it altogether, while few others were exempt. Brown stressed its universality, and mentioned cases of blindness at Mr Walker's cattle station at Wallerawang, where 'the whole tribe' appeared marked, and where there were 3 very old men who had it when they were very young. They had pockmarks, and were not afflicted on the last visitation.²³ Mair said Aboriginals were 'numerously and almost without exception attacked by the disease', but he did not expect it to prevail among Europeans. Few settlers in affected districts had been intimately associated with Aboriginals, and most were vaccinated or had previously had smallpox. All of Rankin's servants at the Lachlan were protected in one of these ways. At Wellington Valley all the soldiers and most prisoners were protected.

Using his informants' estimates, Mair said mortality varied from 1 in 3 to 1 in 5 or 6. Rankin said the number of deaths in persons particularly noticed by him was 20 out of 50, but the average was 1 in 6. Brown said 1 in 5 at Wallerawang, and Clark 1 in 6 on the Namoi. Mair met no opposition from Aboriginals when he encouraged vaccination: 'Those who had not suffered . . . viewed their escape as accidental, and while its frightful symptoms and dire effects were still in their memories, they were willing to submit to a simple operation'. If smallpox could be prevented by vaccinations, 'a friendly intercourse might be established between the Colony and the more distant tribes, leading to highly beneficial results'.²⁴ The Rankins had previously vaccinated Aboriginals, and when the Colonial Secretary advised vaccination against smallpox after the

outbreak, some people listened. Cowper, a doctor at newly settled Braidwood, was one who requested vaccine.²⁵

For the first time since 1789, newcomers who knew the disease had seen both active smallpox and pockmarked people in indigenous populations that inhabited hundreds of miles of territory in newly explored and settled districts. Appropriately enough, Charles Sturt was one of those who recognized just how extensive that outbreak had been. He had found out that the ‘violent cutaneous eruptions’ he saw in Aborigines on his early journeys were actually symptoms of smallpox. He revisited the Murray while overlanding cattle to South Australia, and in 1838, and when he crossed the Edward River on 7 June, he reported:

we were joined by various parties that when united form a considerable body of athletic and well proportioned men. I observed many of them as if pitted by the Small Pox so it would appear the disease which was enjoying such fearful effect upon them when I was on the Banks of the Darling in 1828 and of the Hume [Murray] in 1829, had been universal. 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.²⁶

Further evidence of Aboriginal smallpox and syphilis in New South Wales emerged in the 1840s. On a journey into south-eastern Australia that began at Melbourne in April 1844 George Augustus Robinson, who was then the Chief Protector of Aborigines in the Port Phillip District, was accompanied by a young Englishman, George Henry Haydon, a writer and artist living in Melbourne. They set off on an ambitious journey, supposedly undertaken to improve the condition of tribes in Gippsland. Haydon returned to Melbourne before the journey was complete, while Robinson travelled farther and recorded traces of disease in Aborigines.²⁷ Early in the journey, in June 1844 in the Omeo district in east Gippsland, he recorded: ‘A loathsome disease (Syphilis) among the Natives imported by Europeans is making ravages’. Farther on, at Twofold Bay on the east coast, he said 2 men had died of syphilis while he was there, and later in the interior at Gundagai, he said numbers were suffering from syphilis, several of them badly.²⁸

It was on that journey that Robinson met settlers who knew a good deal about smallpox and its Aboriginal victims, some of whom he saw. At Twofold Bay he was assisted by the first settlers in the district, the Imlay brothers, Scots who were medical practitioners. The youngest of the three Imlay brothers was Army Surgeon Alexander Imlay, who had seen Aboriginals with smallpox at Bathurst in 1831, when he was a colleague of John Mair's. George Imlay was Surgeon Superintendent of convicts; and Peter Imlay was a naval surgeon. Robinson would have found out about Aboriginal smallpox from them.²⁹

Robinson subsequently stayed three days at Yass River, where 300 Aborigines had assembled. He found them a fine and intelligent race, but 'the virulent effects of Variola or Small Pox' were apparent. Near Yass, Robinson was entertained by the explorer Hamilton Hume, who had seen cases of Aboriginal smallpox on his expedition to the Darling with Sturt early in 1829.³⁰ Farther on, Robinson met Wiradjuri speakers on the Murrumbidgee, in a Lachlan tribe, and in another on Tumut Mountain. At Gundagai, he said: 'Many of the Natives are strongly marked by small pox . . . three Natives I accidentally met with had each lost an eye, several others partially blind'.³¹

North-east of Bathurst, near the east coast, Dr E. M. Mackinlay of Dungog recorded in 1845 that smallpox had carried off about half of the local Aboriginal population, principally women and children. He told ethnographer John Fraser that epidemics had occurred on two separate occasions. When he arrived at Dungog in 1840, he observed several oldish men deeply marked with traces of smallpox, and on questioning them, he found that when they were young, a fearful epidemic had raged in the district and carried off great numbers. When it first appeared, they were at 'Black Camp Creek', where smallpox was very virulent. They were unable to bury their dead, and day by day kept moving onwards. Before the outbreak the district was populous, but they never recovered their numbers. Mackinlay also learnt that Aboriginals on the east coast rivers—the Manning, the Macleay and others—suffered severely from smallpox 'about 1835'. He said the contagion may have come from the new settlers, but it was possible that waves of destruction may have spread from north to south, originating in the islands of the Malay Archipelago.³²

Commentary

In the case of syphilis, although European stockmen may have transmitted venereal diseases to the newly invaded tribes, it is unlikely that Protector Robinson was familiar with symptoms of syphilis. Like Sturt before him, he may have seen a closely related indigenous treponemal infection, not syphilis, when he said he had seen a 'loathsome' disease. When Robinson found out about smallpox from well-informed observers in the colony, he described its unmistakable presence in indigenous populations. Smallpox was identified by many colonists in the south-east who had seen it in Scotland and England.

It was unquestionably smallpox that so devastated the south-east before and again soon after settlement. Recent debate about the retrospective diagnosis of smallpox in 1830 evidently resulted from modern unfamiliarity with it, and a lack of reliable records. Busby's report to Bowman about 'chickenpox' was for a long time one of the most detailed and readily available contemporary records, and it may have influenced some readers. Mair's report is still not readily available, but as Fenner's comment suggests, it is the most reliable source for the 1830 outbreak. Except for unusual detail about the tenderness of the victims' feet, which may reflect the situation of Australian victims who needed to hunt and gather for food, Mair's remarks about smallpox in Aboriginal Australia are consistent with today's knowledge of the disease.

References to elderly pockmarked survivors seen by settlers in 1830 suggest that smallpox had occurred throughout most of New South Wales in the late 1780s, probably before appearing at Sydney in 1789. We know that when it occurred for the second time in the colony forty years later, it invaded the Murray–Darling Basin in the remote north-west where it was seen on the Darling by Sturt, on the Castlereagh by Brown, and on the Namoi by Clark, before it reached newly settled districts in eastern New South Wales. It then approached settlements at Wellington Valley, Bathurst, and on the Lachlan in 1830 and 1831, as Aboriginal people feared it would. From centres of infection in the interior, smallpox evidently travelled over the Great Divide and reached the east coast by the end of 1831. It had also travelled farther into the interior and reached the Murray in those years.

The incidence of smallpox in indigenous populations was inevitably high. Clark said 20 or 30 people had caught smallpox at the same time, and Scarm had seen 50 sick at one time. Members of families who travelled together, ate and slept together, were unlikely to avoid infection. Flight from heavily infected districts spread it, and almost certainly increased the overall incidence of the disease. Only a few were likely to avoid smallpox. Second attacks were extremely rare, so elderly pockmarked people who were immune were safe, and some who were at enmity with infected neighbours escaped, as others in remote localities may have. Otherwise, only those who were vaccinated by Imlay and Mair, or by concerned settlers such as the Rankins, were safe. A few were variolated by settlers, without spreading infection further, as far as we know, and were probably safe.

The mortalities of smallpox in naive populations were always severe. Except for children aged from five to nineteen and young adults, who often recovered, survival was uncertain. Economic and social disruption at a time of universal sickness affected supplies of food and water, which were essential for recovery. In those circumstances, mortalities were inevitably exacerbated. If Mair's and Busby's comments about the convalescents' painful feet were accurate, the Australian hunter-gatherers were for some time unable to obtain their usual food supplies. It follows that, for many reasons, mortalities in Aboriginal populations in 1830 were high in newly settled and unsettled districts alike, perhaps almost as high as in Sydney in 1789. The great majority were highly susceptible, some had severe confluent smallpox, the immune were few and elderly. It is likely that over half of those who caught smallpox died of it, in particular the very young, many women, including those who were pregnant, and many previously unexposed people over twenty.

By contrast, there were only 8 recorded cases of smallpox among Europeans in New South Wales who had contact with sick Aborigines in 1830 and 1831. Most Europeans had been vaccinated. For example the older Tom children were not affected, and the younger, unvaccinated Tom children recovered. Mrs Coddy and 2 of her children survived, although none had been vaccinated. Her two-year-old child had confluent smallpox, and, as far as we know,

hers was the only European smallpox death. The unvaccinated Scarm recovered.

Depopulation in Aboriginal clans after two smallpox epidemics in forty years was extreme, as contemporary sources indicate. Enumerations of Aboriginal populations, prepared for the Senate Select Committee in 1845 by a wide range of officials a decade after Mair's enquiry, were consistent with the effects of smallpox. Smallpox was mentioned, population estimates were all low, and there were nearly twice as many men as women in a number of enumerations.³³ Notwithstanding other possible reasons for differences in numbers between the sexes, we know that smallpox killed over 10 per cent more women than men, which may have affected generations of childbearers, and in that way may have contributed to the demographic catastrophe that followed the biological one. In all the circumstances, Mackinlay's later estimate of the damage is feasible. There are precedents for long-lasting depopulation after smallpox epidemics in nineteenth-century South-East Asia as well as in North America.³⁴

New South Wales south of the Murray

The earliest records of smallpox among Aboriginals in the unsettled south-east part of the country that later became Victoria mention several of the pockmarked survivors of 1789, who were seen by crewmen on ships visiting Port Phillip in 1803. The *Cumberland* from Port Jackson reached the Heads on 20 January, and on 18 February James Fleming saw 11 Aboriginals: 'Two of them appeared to be marked with the small-pox'. On 10 October 1803 First Lieutenant Tuckey on *HMS Calcutta* mentioned another Aboriginal whose face was deeply pitted as if from smallpox.³⁵

There were no other literate observers to leave records of pockmarked Aboriginal people in the south-east until the late 1830s and 1840s, when new arrivals in that part of the country saw signs of the past presence of Aboriginal smallpox in the Port Phillip District and on the Murray. Some observers unhesitatingly described more of the elderly pockmarked victims of 1789. None of them had ever seen active smallpox, although the escaped convict William Buckley said

he had seen a fatal disease among the Aboriginals with whom he was living in the Port Phillip District.

Aboriginal smallpox in Victoria 1830–31

In northern Victoria a number of observers remarked on many elderly people who were scarred by smallpox. Peter Beveridge, who arrived in 1839, joined his brothers on stations near Swan Hill in 1845, and knew Aboriginals on the Murray, Murrumbidgee and Darling for over twenty years. His ethnographic studies included one about Aboriginal ovens, which some settlers said indicated a one-time larger population. When interest in Aboriginal smallpox revived in 1877, Beveridge wrote to the *Argus*, and his letter was republished in 1883.

All the very old Aboriginals in the Colony show very distinct traces of small-pox . . . it came . . . down the rivers . . . the death-rate assumed such immense proportions, and the panic grew so great, that burying the bodies was no longer attempted; the survivors who were strong enough merely moved their camps daily, leaving the sick to die unattended . . . that manner of death was indeed a terror to them . . . From our conversations with the natives . . . we think it must have come from Sydney, and if about 40 or 50 years since the inhabitants of that city passed through the ordeal of this plague, there cannot be any doubt remaining of its origin. To this day the old men speak . . . shudderingly, and with such an amount of loathing and horror as it is impossible for any other Aboriginal evil to elicit . . .

This small-pox infliction seems to be the only occasion upon which great numbers died together from the same cause. The natives attribute the pestilence to the malign and magical machinations of tribes with whom they were not on terms of amity.³⁶

James Kirby, who was at Swan Hill in 1845, also knew smallpox had occurred before settlement. He said: ‘We noticed that several of the blacks were pock-marked, but those who bore these marks were all old men’. Others remembered elderly pockmarked people on rivers in northern Victoria. Upstream on the Murray, A. M. Campbell was on Gannawarra Station in 1845, and there were several Aboriginals who had marks such as those left by

smallpox. They were chiefly aged persons, who told him that the disease that caused the marks proved fatal to many.³⁷

Then in February 1838 the overlander Joseph Hawdon saw recently pockmarked people in north-western Victoria, at the Murray–Murrumbidgee junction and near Swan Hill.³⁸ They were also seen on the Avoca and Loddon rivers in the 1840s, and were known to officials. Kirby also mentioned a pockmarked man who wanted an English name: ‘The man we named Sir Robert Peel was a tall fine fellow, deeply pock-marked, and a great scoundrel to boot’. According to Kirby, Sir Robert Peel was one of the Murray Aboriginals who killed 8 of Faithfull’s party near Benalla in 1838.³⁹

Protector E. S. Parker, who arrived in 1838 to serve under Robinson, contacted Loddon Aboriginals in 1839. He described their small numbers, when recorded in his memoirs that: ‘in our particular region, ranging from the Divide to the fringe of the Mallee, and from the Macedon Range to the Pyrenees, the total aboriginal population was little more than three hundred’.⁴⁰ One of the reasons for this was an outbreak of smallpox in the early 1830s, before Parker visited the clans. Governor Hunter’s nephew, John Hunter Kerr,⁴¹ who was aware of the history of smallpox in Victoria, photographed a badly scarred Loddon Aboriginal later in the century. Not far away, at Lake Boga, local settler A. C. Stone thought large cooking ovens indicated a one-time large population. He had heard of a swiftly killing disease that affected a great number of the Boga tribe. It came like a fog, low-lying over the land. Early in the twentieth century, numbers of skulls were seen on the banks of nearby Lake Baker, and the awful fear of the poison-fog lasted until recent years.⁴²

When E. M. Curr lived on his father’s runs from 1841 to 1851, he knew clans at Tongala and on the Murray, Campaspe and Goulburn rivers. Two groups, 150 Wongatpan people and 50 Towroonban people, and eight other clans, were collectively known as Bangerang. Their land extended south as far as Yea and Kilmore, and Curr said ‘the whole Bangerang race numbered not less than twelve hundred souls’ in 1841. Ten years later, the 200 local Bangerang people had dwindled to 80, which Curr attributed to disease and their changed life.⁴³ He knew pockmarked people, and by 1883 he was preoccupied with evidence of depopulation caused by smallpox. There were not only disused ovens, but trees growing in

them in 1841 had been there fifty years. He also thought he had seen a child with smallpox in the 1840s. In 1887 he reported:

A considerable portion of the Bangerang were pitted with small-pox, some of them dreadfully so; and in 1843 . . . I saw amongst them a child absolutely suffering from that disease. One or two children, of not more than ten years of age, bore its marks unmistakably. Of the Bangerang tribes fifty or sixty persons are now all that remain.⁴⁴

E. M. Curr's contemporary, the Victorian pastoralist Albert A. C. Le Soeuf, had told him on 14 February 1877 that:

I am perfectly certain that the native tribes suffered very severely about the year 1830 or '31 from a disease, which, if not smallpox, at all events killed large numbers and left behind on the survivors exactly similar marks. When, as a boy of twelve years of age, I first visited Goulburn River, in 1841, there were a number of the Oorallim tribe deeply pitted, and one or two whose faces were seamed and scarred all over. There were also two or three children of my own age who had had the disease, and they told me that it was when they were about two years old. I also learned that before the disease broke out they had been far more numerous; in fact whole tribes were carried off at that time . . . I will mention a few . . . there were pock-pitted people higher up on the Goulburn. In after years, I saw the same thing among the Lower Loddon, Swan Hill, and Bung-bung tribes. If, as some of the newspaper correspondents assert, very old people had the disease when they were young, there must have been two outbreaks, as there is no mistake about the havoc committed about the time stated.⁴⁵

The most recent record of Aboriginal smallpox on the Murray and its tributaries in New South Wales and Victoria was published in 1993 by Wayne Atkinson, a descendant of the afflicted people. He referred to population losses caused by smallpox among Yorta Yorta people, whose territory stretched from Deniliquin and the Edward River to Kow Swamp, Mulwala and Shepparton. He said smallpox reduced them from 2400 to 1200–1400 before white settlement.⁴⁶

In far western Victoria, the Hamilton family settled at Ozenkadnook and Bringalbert in the Edenhope district in 1846. James Hamilton, who was then eleven, later remembered a place where

many Aboriginals were buried. They died ‘of some epidemic’ before Europeans came to that part of the country. Local tribesmen avoided the place, and all he could find out from them, was ‘All about very sick, and tumble down dead like rotten sheep’. Later, local historian E. R. Trangmar said smallpox travelled south from the Darling and Murray rivers as far as Edenhope, where mass graves and pock-marked faces were associated with the onslaught.⁴⁷

Some other newcomers in Victoria in the 1840s were less familiar with smallpox. Robinson, newly arrived from smallpox-free Tasmania, attributed population losses among Aboriginals in Victoria to frontier conflicts, and did not at first recognize signs of smallpox among Aboriginals. At Portland in May 1841 he noticed that 21-year-old Pullerteerrang Harry’s nose was pitted and scarred, without realizing that smallpox was its likely cause. He mentioned an extinct group on the Hopkins River who were not killed by Europeans, but died from disease. He eventually understood the damage smallpox had inflicted during his overland journey in 1844, and informed the Select Committee about Aboriginal smallpox in 1845.⁴⁸

Several years later, James Dawson settled east of Portland at Port Fairy, where he got to know local clansmen, and saw a very severely pockmarked Hopkins River victim. Writing his reminiscences in 1883, he described Aboriginal smallpox, which occurred in 1830, came from the west and was spread by messengers with news of its ravages. His Aboriginal informants, who called smallpox *meen warann*, or chopped root, apparently associated it with what they had eaten, as some Asian victims did. Decades later they still had vivid memories of the great number killed by the disease.⁴⁹

Further evidence of Aboriginal smallpox at Port Phillip in 1789 was published much later in the *Australian Medical Journal* by Dr David Thomas, who said that when he arrived there early in 1839, he saw Aboriginals of the Yarra, Geelong and other tribes, all of them rather advanced in years, who had the pits of smallpox. The only answer they could give to his queries as to where and how they got it was that it had come a long time ago and killed many of the indigenous people. Thomas thought there was no doubt that it had extended over various parts of the country. Governor Hunter’s nephew, John Hunter Kerr, had similar impressions. At Heidelberg on the Yarra in 1839 and 1840 he saw older men who were deeply

pitted with smallpox, and in every case the person so marked appeared to be over fifty years of age. When asked how long ago smallpox attacked them, their answers were always the same: 'Long time: when me picaninny fellow'. Kerr thought it had spread over the continent in 1789.⁵⁰

On the outskirts of the Port Phillip District near Ballarat in the 1840s, several adults were strongly marked with smallpox at the time the area was taken up for pastoral occupation.⁵¹ After living with Aboriginals on the Barwon River near Geelong between 1803 and 1835, William Buckley said he had not seen any European contagious disease prevalent among them, which he found strange. Nevertheless, he did recollect that he had seen '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 affected with ulcers of a very painful kind.'⁵²

Peter Snodgrass arrived in the Port Phillip District in 1838, and was the first settler to take up a station on the Goulburn River west of Yea. When he arrived, there were probably about 500 or 600 Aboriginals, scattered in small tribes in various parts about the rivers and creeks, who occasionally collected in large numbers. He said that from their own statements, they seemed to have been much more numerous a few years before settlers arrived among them, but they suffered severely from smallpox, and many bore evident marks of it. Such individuals were seen for decades, and the numbers diminished until there were perhaps scarcely one-fifth of the number he first saw.⁵³

In the 1840s others on the outskirts of Melbourne knew about Aboriginal smallpox. Protector William Thomas at Narre Warren, Dandenong, and Protector Parker from the Loddon had heard of the mythical rainbow serpent. It was linked with disease and death: it caused smallpox. Of all the beings most dreaded by the Aboriginals, the principal one was the *Mindy*.

When the *Mindy* is in a district the blacks run for their lives, setting the bush on fire as they proceed, and not stopping to bury their dead or attend to any seized. Many drop down dead on the road. When

seized, pains seize them in the back, with violent retching. When they try to get up they fall down; those not seized are quite well . . . Any plague is supposed to be brought on by the *Mindy*.⁵⁴

Robinson's companion of 1844, George Henry Haydon, was a friend and shooting companion of Bembo, an Aboriginal from the Werribee tribe, and he had studied Aboriginal language and customs. When Haydon left the colony in 1845 after five years in Melbourne, he said he had never heard of a case of smallpox while he was there, but the way many Aboriginals were disfigured indicated to him that it had made fearful ravages among them at some former time.⁵⁵

There were few settlers, and no records of smallpox at Westernport or in forested Gippsland in 1789 or 1830, when it was seen by observers in the Port Phillip District. In 1886 Curr said Gippsland escaped smallpox, adding the following year, that 'no traces ever existed in Gippsland, as I have been informed by many persons besides Mr Bulmer, who also remarks that the Blacks of that district have no name for the disease'. Curr's informant, John Bulmer, was twenty when he migrated from England to Melbourne in 1853. In 1855 he became a missionary to tribes on the Murray, then took charge of a new settlement in Gippsland, where he worked with the Kurnai for many years,⁵⁶ and was known to Curr.

Commentary

Early records of outbreaks in southern Victoria in the late eighteenth century are few but reliable. Visitors who recognised smallpox saw pockmarked people at Port Phillip in 1803. In 1839 Thomas and Kerr saw elderly pockmarked people on the Yarra and at Geelong, who vividly remembered outbreaks in their childhood. Still later, King of the Medical Board of Victoria saw very old survivors of the first epidemic near the Murray.

But there were no settlers south of the Murray to see active smallpox in 1830, and for years some vaccinated settlers, such as Rusden, did not recognize pockmarks, and called the disease 'native pock'. Other records of the second epidemic are also confusing. The pockmarked Buckley, who did not name the Aboriginals' disease, was on the Barwon about the time smallpox occurred elsewhere in

Victoria, and may have seen it, although his description of ‘a dreadful swelling of the feet’ is an uncharacteristic description of smallpox. However, Mair and Busby, who described smallpox in New South Wales, said convalescents could not walk for some time because of their tender feet, which is also uncharacteristic of smallpox. Their accounts suggest hunter-gatherers may have had unusual problems after smallpox.

We have already stated that records of the second epidemic were disputed by Diane Barwick, and others have hesitated to acknowledge that a second epidemic hit Victoria in 1830. Nevertheless, a number of the 10 000 or so settlers by 1840 did recognize pockmarks acquired in the previous decade, and left records that clearly refer to the recent presence of smallpox. These included Le Soeuf, Curr, Snodgrass and Parker, and some, such as Haydon and Robinson, mentioned more pockmarked people than the small number of pockmarked old people they knew, who had recovered as children fifty years earlier, and who were then aged over sixty or even seventy. That the second outbreak occurred in the future colony of Victoria in 1830 and 1831, when it occurred on the Murray in New South Wales and in the future colony of South Australia, is certain. When interest in smallpox revived after its resurgence in Europe in 1870–75, settlers recalled Aboriginal smallpox and its victims. Curr, who stated in *The Australian Race* that smallpox was endemic in Australia after 1789 was mistaken in claiming he had seen active smallpox in an Aboriginal child in 1843, because there was no smallpox in Australia at the time.

In the country west of Port Phillip, from the Murray to the south coast, records of Aboriginal smallpox indicate that it was ‘universal’ in 1830 and 1831, as observers said it was in New South Wales and South Australia in those years. Close contacts in relatively dense populations on Victoria’s rivers and lakes, and in coastal districts, meant that smallpox spread quickly over short distances in accessible neighbourhoods. Its incidence in the dwellings of closely related clans in semi-settled and well-endowed districts, from Portland and Port Fairy to the Port Phillip District as far as Westernport, would have been high. With high case-fatality rates as well, mortalities would have been severe. Smallpox was the first and worst cause of depopulation in Victoria, and it was the second cause as

well. In the absence of most other lethal diseases, European or indigenous, the extinct group on the Hopkins River, mentioned by Robinson, would have been its victims. They were not the only lost clan. We know the extinction of whole families was not unusual in naive populations afflicted by smallpox, and that relatively dense populations in South Australia also suffered badly.

Western District historian Jan Critchett, who studied Robinson's records closely, has questioned his description of Harry's nose as evidence of smallpox. Nevertheless the description is characteristic of facial disfiguration caused by smallpox, as pictorial evidence acquired during its eradication establishes. Critchett also questioned smallpox as a significant cause of depopulation in what is now the Western District of Victoria, and says that in official reports, Robinson blamed the great loss of life there on frontier conflict in the early 1840s. However, Robinson's account of his journey in New South Wales in 1844 shows not only that by then he had a clearer understanding of the physical effects of smallpox, but also that he found it to be a most significant cause of depopulation.⁵⁷

Depopulation in Victoria may have been greater than it was in Aboriginal populations in New South Wales. The highest incidence of smallpox, and the most severe mortalities it caused in Victoria in 1830, were in populations on the Murray and tributaries, around river junctions and lakes, and in coastal districts, where abundant resources had once supported larger populations, which were dense by Australian standards. Archaeological evidence may reveal late changes in population density caused by smallpox.

There were fewer enumerations of local populations in Victoria, but in a count in the Wimmera in 1863, there were 136 men, 77 women, 20 boys and 7 girls, a total of 240,⁵⁸ which was similar to those published by the Select Committee in 1845. However, Critchett questioned the demographic impact of smallpox in the Western District, because in 1841 in Robinson's enumerations, there were more women than men. He recorded 780 men, 1050 women, and 1106 children. Barwick also found evidence of 'excessive masculinity' in Victorian Aboriginal populations in 1863 and earlier. Critchett's tally of Aborigines killed by Europeans suggests women may have outnumbered men in 1841, because frontier conflicts in the Western District reduced the number of men.⁵⁹

Fragmentary but convincing evidence of smallpox at Port Phillip in 1789 draws attention to the Bay as a focus of infection, and to the absence of any evidence of smallpox east of Port Phillip at Westernport, and farther east in country that became Gippsland. Curr's account of the absence of pockmarks in Gippsland, and descriptions of pockmarked people elsewhere in Victoria, were accepted as evidence of Gippsland's freedom from smallpox by Isabel McBryde.⁶⁰ But claims by Curr, Bulmer and other informants that pockmarks were never seen in Gippsland are inconclusive, because Curr was rarely in Australia before 1850, and Bulmer did not arrive until 1853. By comparison, later evidence of smallpox in Melbourne clearly shows that the disease had occurred there in 1789. It is reasonable to think it may also have occurred east of Port Phillip in 1789.

If the lethal infection spread a short way to Westernport, then into Gippsland through traders or after ceremonies, severe mortalities due to smallpox may have been followed by deaths due to tribal wars against 'enemies' thought to have caused long-remembered outbreaks. Robinson said Gippslanders 'nearly annihilated' a whole Westernport tribe, and La Trobe said the Kurnai of Gippsland had been at variance with Westernport tribes 'from time immemorial'.⁶¹ Recently, Don Watson attributed disturbances in Gippsland to sorcery and payback killings after deaths caused by contact with Europeans and their diseases.⁶² But smallpox was a more likely cause of payback killings than diseases introduced by Europeans before 1850. If it spread to Westernport and Gippsland as well as the rest of what is now Victoria in 1789, it may have halved the original population before returning in 1830.

Conclusion

From 1829 until the end of 1831 smallpox spread through the Murray–Darling Basin in the interior of eastern Australia, and reached the east coast and the south coast of the colony of New South Wales, including country that later became Victoria. Except for Braidwood Wilson and Captain Smyth, recently back from Raffles Bay, the origin of outbreaks they saw in Aboriginals in 1830 and 1831 eluded settlers such as Beveridge and Curr in Victoria,

who saw no active smallpox, and few pockmarked survivors until the 1840s, when they assumed it had been introduced at Sydney. Sturt's reports, and later the first European death from smallpox in Australia in 1831 had no doubt alerted Darling and an inner circle to the unexpected presence of smallpox in the colony. He took appropriate action on receiving Mair's astute report of his journey in New South Wales, which indicated that smallpox had originated in the interior, not on the east coast. Thirty or forty years later, Mair's work was vindicated, when inland explorers reported recent smallpox in the interior.

The devastation of Aboriginal Australia by smallpox, and the Australia-wide deaths that followed on several occasions, paralleled the destruction of Native Americans by the same disease. In the nineteenth century smallpox swept north from the Gulf along the Mississippi and Missouri rivers. Massive outbreaks occurred at the beginning of the century and in the 1830s. The second epidemic extended over the whole continent west of the Mississippi. Like the second Australian epidemic, it was better recorded than the first, because there were more Europeans to see cases, and it may not have been worse than the first; the same doubt applies to the second Australian epidemic. Depopulation after two American epidemics was extreme; for instance, a Mandan population of 1500 to 2000 was halved on the first occasion; the next time, only 27 Mandan were left in North Dakota. Famous tribes suffered heavy losses: Pawnee, Choctaw, Apache, Comanche and Sioux. Some tribes merged. By 1838 the terrors of severe smallpox among Native Americans on the western frontiers dispelled settlers' fear of attack.⁶³ When smallpox spread over vast distances during long periods, it may have caused more deaths than intermittent frontier violence did in America, as it certainly did in Australia in 1789, the 1820s and early 1830s. Jared Diamond, who wrote of Aboriginal hunter-gatherers being shot and driven off their land, neglects the effects of the main killer, smallpox, and why the disease precluded effective resistance to the invaders.⁶⁴

The aftermath

Indigenous Australians thought Europeans had caused smallpox, and when they knew its full horrors, Murray Aboriginals came close

to combat with Sturt (in 1830), and Mitchell found some Darling River Aboriginals 'implacably hostile'. The belief that Europeans were responsible for the disasters of smallpox was long-lived, and led the indigenous people to retaliate appropriately, as implied by the account given to Protector Parker. Sorcery was meant to retaliate and to give Europeans dreadful sores, make them blind, and kill them. The pastoral invasion by 250 000 settlers followed in New South Wales and Victoria. The scale of that invasion, intermittent frontier violence and changes in available resources, prevented depleted populations of smallpox survivors from returning to their old life in south-eastern Australia. Some, such as Eliza Warren from the Yarra Yarra tribe, Rosie and Ellen from the Carngham tribe, were photographed in the 1860s. Old Kurnai people were vaccinated against smallpox in 1876.⁶⁵

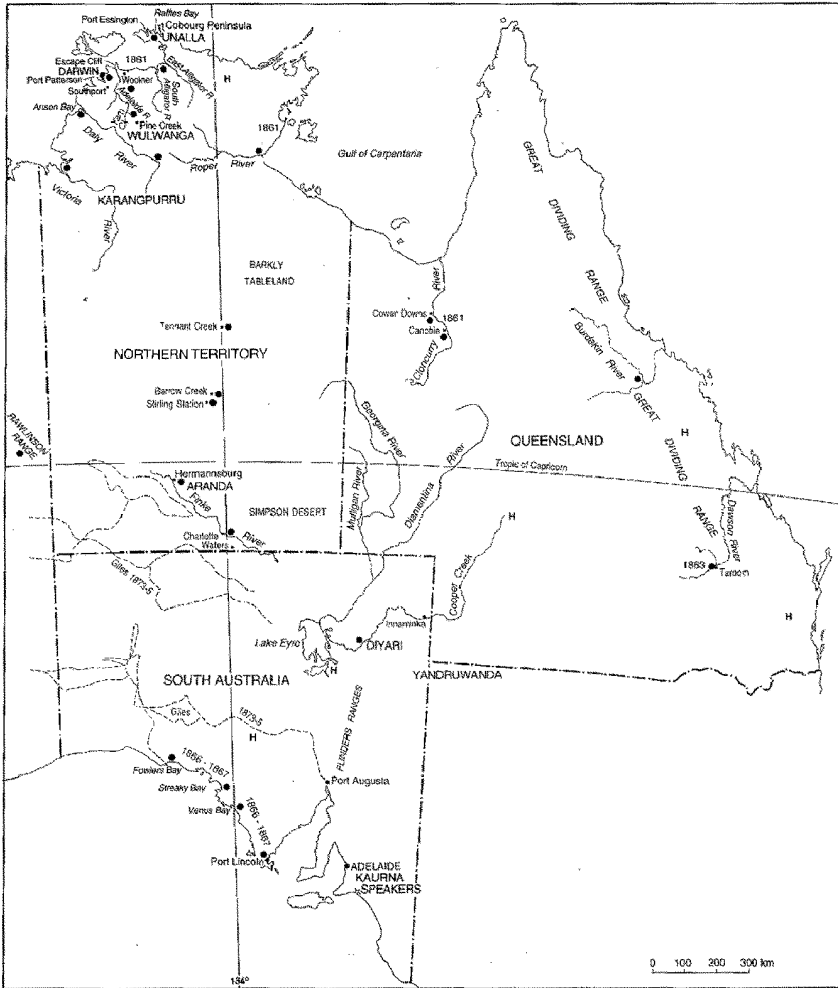
Tuberculosis was the most damaging disease introduced to indigenous people by newcomers. It was present in European populations in the south-east that were too small before the gold rushes to maintain most of the infectious diseases of Europe. It was certainly introduced at Sydney before 1800, and by 1845 it was common in the European population of about 150 000 in the older districts of New South Wales. There was 'consumption' in Aboriginals at Port Macquarie and Picton, and 'pulmonary disease' at Goulburn. In the 1830s tuberculosis was present in the small European population of the future Victoria, and when settlement spread in the 1840s it was frequently transmitted to Aboriginals in the settled districts.⁶⁶ Curr said it was the great cause of death on every station that had been occupied for at least ten years. When the number of cases increased in large European populations, tuberculosis increased also among Aboriginals. In Victoria in 1861 it was said that those with consumption died in weeks, not months.⁶⁷ Settlers, including the Protectors, still noticed traces of smallpox in Aboriginals, and they knew of more recent deaths among them due by tuberculosis. Half a million newcomers, who arrived in the following decades, saw only small clans in well-endowed districts, and the belief that Aboriginals were a 'dying race' lasted well into the twentieth century.

8

Eastern Australia 1860–1867

During an epidemic of Aboriginal smallpox in eastern Australia in the 1860s, active smallpox was initially recorded by only one medical practitioner and several settlers and policemen on the far west coast of South Australia. However, expeditions by explorers John McDouall Stuart in 1862 and Ernest Giles in the 1870s revealed the extent of the damage smallpox had recently caused. Newcomers in Queensland and western South Australia also left records of pock-marked Aboriginal victims who had survived the 1860s outbreak, as did others who were in the Northern Territory during and after the construction of the Overland Telegraph Line in 1872.

Aboriginal smallpox was more widely recognized after the Franco-Prussian War of 1870–71, because there was a resurgence of smallpox in Europe, and more shipboard introductions and lethal epidemics in colonial ports than ever before. After a shipboard introduction and some deaths in an immigrant Sydney family in 1876 and 1877, the Melbourne *Argus* published letters about Aboriginal smallpox. Some old settlers mentioned its recent presence in frontier districts in Queensland, the Northern Territory and western South Australia. European epidemics in the 1880s in Sydney and Melbourne ensured public interest. The Royal Society of South Australia, with Sir Edward Stirling on its Council, published accounts of



Key: ● outbreak H hypothetical outbreak - - - Giles' route

Eastern Australia: smallpox outbreaks 1860–67

Giles was one of a few settlers in the interior in the 1870s who realized smallpox had recurred in the 1860s. He saw pockmarked Aboriginal survivors in almost every part of the country in which he travelled in Western Australia, the Northern Territory and South Australia. Telegraph operators saw them in remote places in Western Australia, Queensland, the Northern Territory and South Australia after the construction of the Overland Telegraph Line. Missionaries saw them on the Finke River, and policemen and settlers saw them in the Northern Territory and South Australia.

Aboriginal smallpox by Foelsche, Lutheran missionary Schulze and other correspondents.

Smallpox was topical when E. M. Curr, who had seen its results on the Murray in Victoria, enquired about it from correspondents around Australia in the 1870s and 1880s. Before any professional accounts of Aboriginal smallpox were published, Curr became an authority on the subject. The squatter and author had been educated in Europe between 1829 and 1839. As a schoolboy, he probably would not have seen active smallpox, although he would have seen pockmarked Europeans. When he returned to Victoria to manage his father's sheep runs in the 1840s, he saw for the first time Aboriginals with pockmarks. His interest in Aboriginal Australians led to a search for ethnographic material that lasted for decades, and the questionnaires he sent to informants included questions about smallpox. He edited their replies and included them and his own account of the history of Aboriginal smallpox in *The Australian Race*, 1886–87.

Curr's three volumes were, and still are, an unusual source of evidence, and his work was influential. Frank Tidswell, Microbiologist to the New South Wales Board of Health in 1898, was one of the first medical scientists to review the history of Aboriginal smallpox, and he followed Curr closely.¹ A. R. Radcliffe-Brown used Curr as a reference in 1930. Curr's influence is recognizable in Butlin's hypothesis, that Aboriginal smallpox came with the First Fleet, and in his emphasis on south-eastern Australia before 1850. Curr's influence can be seen in White and Mulvaney's 1987 estimate of the former Australian population, where they suggested the impact of smallpox was greater in the south than the north.

Curr conceded that smallpox was introduced at least once in north-western Australia after 1845, probably by 'Malays', as his informants in Western Australia said. But he said there was nothing to show that it had ever occurred north of the Tropic of Capricorn and east of 134° longitude, which is hard to reconcile with his informants' evidence of it in eastern Australia in the 1860s. In effect, Curr was the first authority to dismiss the possibility that an introduction in the north had caused an epidemic in eastern Australia in the 1860s. A century later, Butlin ignored common and unpredictable accidents of infection, which persisted until smallpox was

eradicated, when he said the visits of trepangers on the north coast in the wet season were unrelated to Aboriginal infection.²

With up-to-date knowledge of the Macassan connection, the history and epidemiology of smallpox in the archipelago, especially in South Sulawesi, and Aboriginal lifestyle in monsoonal Australia, the question is no longer whether infectious Macassan visitors occasionally transmitted smallpox to indigenous Australians, but how often it happened. Smallpox was undoubtedly increasingly prevalent among Indonesians in the nineteenth century, and in the 1860s, most Aboriginals still had no contact with the infection. Immunity was conferred *only* by a previous attack, and would have been unusual, even on the coast.

Smallpox may have been introduced to small or isolated bands, where it died out in the wet season before many people caught it. Moreover, when the Macassans landed, Aboriginals were widely dispersed in clan territories and were sometimes outnumbered.³ If smallpox was prevalent when the Macassan fleet sailed, and the Australians had not been exposed for decades, it is likely that a number of cases occurred between the Top End and the Gulf. Daily contacts in small groups of related family members, who slept and ate in wet weather shelters,⁴ were inevitably occasions of infectious contacts, and secondary cases could have continued for months.

When the fishermen departed, life in the dry season favoured the spread of infection. Face-to-face meetings between healthy people and others recovering from attacks, but still infectious, were likely when neighbouring families moved about gathering food in the early dry season. Travelling, visiting, talking, trading commodities and performing rites and ceremonies with visitors from other areas and language groups followed, and contacts between Aboriginal people multiplied.⁵ Within weeks of an index case, that is the first case in a previously unexposed district, unexpected sickness and deaths would have caused fear and flight. In the Victoria River District, some erected brush walls to protect themselves, but nothing could stop smallpox spreading after a monsoon.

Communication networks⁶ allowed infectious contacts in larger tribal populations as well as local bands. Although introductions that caused widespread outbreaks in significant numbers of susceptible people were rare, they did occasionally happen. They

happened more often in eastern Australia than Western Australia, and after it appeared in coastal clans in 1861, smallpox spread unchecked through unsettled and newly settled districts in eastern Australia. Curr did not have all the evidence of it in Queensland, the Northern Territory and South Australia in the 1860s that is available now, and Butlin did not include the 1860s in his work.

Colonists in northern Australia after 1860 realized they were seeing people who had suffered smallpox not long before. Survivors spoke of die-offs, some had lost the sight of one or both eyes, and many were pockmarked. Newcomers who knew smallpox could tell roughly when it had last occurred from the ages of survivors, who had been children or young adults when they had acquired pockmarks.⁷ Foelsche thought it had been introduced in the Gulf by Macassans about 1861, long after the soldiers left Port Essington in 1849. He and Palmer realized it had also occurred in the 1820s. In 1861 it broke out after being absent for nearly forty years, and when it reappeared, the only immune people were middle-aged and elderly survivors of earlier attacks. So wherever it occurred, there were virgin soil outbreaks, when the lethal virus was at its worst.

Aboriginal smallpox in Queensland 1861–65

In August 1881 Foelsche's paper about the Aboriginals of northern Australia was read to the Royal Society of South Australia. He said smallpox had been introduced

on the last occasion by a prahus that visited the Gulf, for they leave so soon as the South-East monsoon has fairly set in, and shortly after the prahus had left the disease appeared, coming up the coast from the Gulf with the S. E. winds, as stated by the natives, and travelled through all the tribes to the westward.⁸

Several of Curr's correspondents had settled in north Queensland after 1860, and had seen scarred Aboriginals on the Cloncurry River. About 150 kilometres south of the Gulf, an anonymous settler on Conan Downs station [Cowan Downs, 19° latitude and 140° longitude] knew about smallpox, but his writing was hard to decipher. In 1879 he claimed there were 1000 Miappe people when Europeans arrived in 1860 [they spoke the Mayi-Yapi language]. By

1868 there were only 250, and by 1879 only 80 remained. He said when he met them in 1868, some were lightly pitted with smallpox, and when he later wrote to Curr, he thought a few were still alive. The settler attributed their catastrophic demise to slaughter at the hands of the Native Mounted Police, and to diseases, in particular 'venereal diseases' (in that climate he might have seen yaws) and measles. Curr's other informant was Edward Palmer, a pastoralist from New South Wales who moved to Queensland in 1857. Palmer knew the first settlers in the Carpentaria district in the early 1860s. In 1864 he was a proprietor of Canobie station, 200 kilometres south of the Gulf on the western bank of the Cloncurry, where Miappe people hunted, and he named four 'tribes'. Palmer said: 'Smallpox, with which the tribe were afflicted within the last half century, is called *nyamooroo*'. The prevailing disease was consumption.⁹

Another informant was W. O. Hodgkinson, explorer and journalist. He was Mining Warden of north Queensland goldfields in the 1870s and 1880s. He was at Maytown between 1882 and 1884 when he told Curr about Breeaba [Gugu-Badhun] people on the headwaters of the Burdekin River. He heard about smallpox from a very intelligent middle-aged Breeaba woman, who lived at Maytown on the Palmer River.

It is called chin-chin, and is said to have proved fatal to many at some recent period. The woman Wonduri . . . declares that the tribe decided at the time of this scourge that anyone it attacked should be killed without delay whilst asleep, and that this plan was carried out.

The disease was not heard of nearer than 480 kilometres farther south, so Curr had no confidence in Wonduri's statement.¹⁰

Newly acquired pockmarks were seen in Aboriginal people at Taroom, perhaps Gangulu people of south-central Queensland in 1864. Telegraph officer 'W', who wrote to the *Argus* in 1877, had known Aboriginals in Victoria, Queensland and South Australia since arriving in Australia thirty years earlier. He recorded that:

during all that time it has been matter-of-course belief with me that they were subject to attacks of the disease [smallpox] at periodical intervals. When I was on the Upper Dawson in charge of the telegraph station at Taroom, about thirteen years ago, I saw many blacks whose appearance would lead anyone to think they had had small-pox. They were pitted all over with good-sized marks.¹¹

Commentary

Palmer told Curr smallpox had occurred on the Cloncurry River in the last fifty years, and the Breeba woman Wonduri described desperate measures taken by people on the Burdekin, who apparently already knew its horrors. It seems clear that it occurred at least once in north Queensland before 1860. There is no evidence of active Aboriginal smallpox in Queensland in the 1860s, but several informants later reported that they had seen pockmarked people who may have been infected then. Smallpox had evidently occurred in the south-east of the Gulf, where pockmarked people were seen after Europeans established stations on the Cloncurry. At Cowan Downs, the Mayi-Yapi were 'lightly pitted' in 1868, and faded scars suggest they had suffered some years earlier. At Canobie, Palmer probably saw recent survivors as well as elderly ones. Infection spread from the Gulf to Breeba people on the headwaters of the Burdekin, and others on the east coast. Its impact in tropical north-eastern Australia would have been as severe as it was after the monsoon on the north coast.

Infection continued to spread, and smallpox reached clans in central Queensland, where it broke out on the Upper Dawson not long before 'W' saw newly scarred victims in 1864. The epidemiology of smallpox and its history on other continents show clearly that sporadic cases of smallpox did not occur, and it must have been more widespread in half-settled Queensland than available records indicate. It was probably an important cause of depopulation in the early 1860s in districts in east Queensland that were well endowed with natural resources before European settlement. It probably did not spread far into settled south-eastern Australia at that time.

Aboriginal smallpox in the Northern Territory 1861–63

Neither explorer McDouall Stuart nor surveyor Boyle Finniss left any record of Aboriginal smallpox in the Top End in the 1860s, which is surprising, because older settlers, such as Finniss, must have seen pockmarked survivors in Britain before emigrating, and facial pockmarks on Aboriginals would have been obvious in 1864. Others who followed when Escape Cliffs was abandoned knew the disease; for instance, the Resident at Darwin in 1870, a relative of the White Rajah, had probably seen it previously in Sarawak.¹²

Some newcomers did refer to pockmarked and blind Aborigines. Paul Foelsche reached Darwin in January 1870, and E. O. Robinson arrived on the Cobourg Peninsula in 1874. Neither saw Aborigines with active smallpox, but they reconstructed the history of the most recent Aboriginal epidemic, describing its traces, and what survivors had told them. In 1880 their estimates of the size of the population affected by the last outbreak on the Peninsula were similar. Foelsche's information probably came from Finniss, who had revisited Darwin in 1870.

The Royal Society of South Australia published Foelsche's paper in its 1881–82 volume. He reported that smallpox was the disease most dreaded by the Aborigines, that it caused great havoc in infected tribes. Each tribe had a name for it. The 30 Port Essington tribesmen, known to him as *Yiarick* and also as Unalla, called it *meeha-meeha*. Local Aborigines told Foelsche that the last outbreak at Port Essington had occurred after the soldiers had abandoned the settlement in 1849. Foelsche calculated that the last time smallpox made its appearance on the north coast was 'about twenty years ago'; it came one year shortly after the praus set sail for Macassar about the end of May, at the beginning of the dry season. It came from tribes to the east when the grass was burnt, and it travelled west. The disease killed many, both old and young, but when the rain came back about October or November, it disappeared. Jack Davis, a tribesman about fifty years old who spoke English and 'Malay' fluently, predicted that it would come again when he was an old man. Foelsche's paper was republished with minor revisions in 1912. *Yi'arick* meant smallpox, and he repeated that smallpox was introduced 'about twenty years ago'.¹³

Ten years after Foelsche arrived at Darwin, he received a printed list of questions from Curr about Aboriginal smallpox. Six years later, his replies were published in Curr's first volume of *The Australian Race*. He told Curr that in 1881 the Unalla tribe, whose land extended from Raffles Bay to Port Essington and midway up the Cobourg Peninsula, and who were once numerous, had been reduced to only 30 people: 6 men, 12 women, 9 boys and 2 girls, whose names he listed. Smallpox was *mea-mea*, and 4 pockmarked survivors were still alive. The local tradition was that they had caught the disease from trepang fishers. But when Curr edited

Foelsche's replies to his questionnaire in 1886, he was confused about when smallpox was introduced on the Cobourg Peninsula. In his Raffles Bay entry, he said it was 1866. In an earlier chapter, he said it visited tribes on the Cobourg Peninsula in 1865 or thereabouts. He also said the tradition of the Cobourg tribes was that trepangers had brought it in 1860.¹⁴

Before becoming a customs officer for the Government of South Australia in 1882, Robinson was manager of the Cobourg Cattle Company, a buffalo-shooter and a trepanger. His camp was near the ruins of the old military settlement at Port Essington, and he knew the local Aboriginals well. He spent as much as nine months at a time with them, 'and no white man nearer than Port Darwin'. In a letter to A. W. Howitt in 1880 he said that smallpox was already known to Aboriginals when Europeans came in 1838, and that it was probably a result of Macassan contact. He told the South Australian Government in 1882 that from an estimated number of 200, 'the Port Essington tribe nearly all died and now only number 28 all told'.¹⁵

In 1881 Foelsche reported that smallpox had spread west from the Cobourg Peninsula through the Alligator and other tribes and reached Aboriginal people at Darwin. They called it *goobimwah*, and Foelsche had no doubt that the malady had raged there at the same time as at Raffles Bay. It broke out in the dry season between May and November, and killed many people, both old and young. Mangminone, alias Mr Knight, who lived at Darwin, was about twenty-five when Foelsche told his story. Deeply pitted all over the face, Mangminone had smallpox when he was about five years old, before Europeans arrived with Finniss at Escape Cliffs in 1864. Some survivors became totally blind. Four blind survivors lived near Darwin. Tribesmen recalled the devastation when young and old were stricken, and a great many died, so many that they could not bury them all, 'but left the corpses lying about'. Foelsche's opinion, that it came with trepangers, was recently supported by Macknight. The Macassan language was the most common language spoken on Macassan praus, and the word for smallpox, *puru-puru*, appeared as *purrer-purrer* in a vocabulary from the Woolner district at Darwin in 1869. The Aboriginal name for Escape Cliffs, fifty kilometres from Darwin, is Pater-purrer.¹⁶

When Curr described smallpox at Darwin, he included more information from Foelsche. The tribe was the Larrakiya, whose territory extended from the mouth of the Adelaide River, west to Port Patterson and forty kilometres inland. There were 500 Larrakiya in the early 1880s: 100 men, 120 women, 150 youths of both sexes and 130 children. They had not decreased since Europeans arrived. Smallpox occurred twenty years earlier, and came from the east. Six pockmarked survivors aged between twenty-five and forty were still alive.¹⁷

A Darwin Aboriginal, respected by the whole tribe as a learned man, told Foelsche about the creation and origin of his people. A good man called Nanganburrah lives in the bowels of the earth:

He a long time ago made one blackfellow, called him 'Dawed', and taught him how to make blackfellows. 'Dawed' made plenty of boys and girls, who grew up and multiplied. When Dawed was an old man the blackfellows growled plenty, and would not do what he told them; he then made them very ill, and plenty died, but some got better.

Foelsche told Curr the deaths of those who died before they were old were attributed to the incantations of their enemies.¹⁸

Aboriginals had a specific remedy for smallpox. They used thick milky-looking juice from a leafless vine found along the shores of mangrove flats. It was put on sores and left until scabs formed. According to Foelsche, 'This remedy is said to be a sure cure, although some who used it lost their eyesight; but strange to say some patients object to having it applied, but why they cannot explain'. He and a local Aboriginal showed the vine to Professor Ralph Tate of Adelaide, founder of the Royal Society of South Australia, who visited the Northern Territory in 1882. He identified the vine as *Sarcostemma australe*, which he identified in Foelsche's published paper. He could not estimate its value as a cure, because smallpox had not occurred since Europeans occupied the Territory.¹⁹

Others at Darwin in the late nineteenth century shared Foelsche's opinion about the inroads of smallpox. In 1883 the Resident said that 'the natives along the coast, which is frequented by Malays, are considerably marked by smallpox'. When he was there in the 1880s, R. Coppinger commented that 'Smallpox has made

sad ravages among this tribe of natives, and accounts for the large proportion whom we found to be wholly or partially blind'. In northern Australia early in the twentieth century, medical practitioners Ramsay Smith and Basedow saw a number whose faces bore 'genuine and typical pock marks'.²⁰

Outbreaks were not confined to Aboriginals in coastal districts in the Top End, as the northern part of the Northern Territory is now called. After twenty-two years in Australia, W. B. Wildey toured the Top End in 1873 after the Overland Telegraph Line was constructed. He walked 200 kilometres to Yam Creek, keeping a daily record of the settlements he visited within about eighty kilometres of Darwin: Escape Cliffs, Anson's Bay and Southport. Another eighty kilometres inland, he crossed the Adelaide River, and eighty kilometres farther on, he camped at Yam Creek. The next telegraph station was Pine Creek. Wildey commented on the health of Aboriginals: 'Swamp fever and ague, and small pox, or a very similar pock, are very prevalent; many have lost one eye from disease, and some are covered with leprous-looking sores and boils'. He saw fearful sores on eyes, necks, and other parts of children's bodies, that were 'actually festering, and bitten by numerous flies'. Foelsche had often seen boils.²¹

Wildey described tribes in districts he visited. The Larrakiya at Palmerston, Southport and Escape Cliffs were not numerous, perhaps 300. Wulna people on the Adelaide River were numerically stronger. There were Wogait people at Anson's Bay, Aguagwillahs south-east of Southport, Wulwanga people near Yam Creek, and Gwoolingahs near Pine Creek. He thought there were separate tribes about every hundred kilometres to the south.

When German traveller Erhard Eylmann published an account of Aboriginals in South Australia in 1908, he included smallpox.

I heard from the Wulwanga, about forty years ago a severe epidemic struck the whole Roper River and Daly River regions. The number of deaths was said to be so great that many people had to be left unburied and that almost all the tribes were reduced to half their number. This disease was reported to me as small-pox. However, I do not believe that it was small-pox, for . . . there can be only few Aboriginals with

pockmarks in the whole colony. Two Wulwanga Aborigines of about sixty, one of whom maintained that he had lost an eye, and the other the top joint of the big toe on one foot through the disease, both had smooth, unpockmarked complexions. From the information given me about the cases of skin infection, it is not to be ruled out that it was an epidemic of a kind of pestilence. The disease was said to have travelled from the Yermangel, who live at the mouth of the Adelaide River. This would suggest that it had been brought in by strangers, perhaps Malays.²²

In 1881 Foelsche described pockmarked Aborigines among all the inland tribes, and Robinson thought smallpox had ‘spread like wildfire amongst the natives not only along the coast but a considerable distance inland’.²³

Over a century later, these versions of the spreading scourge were echoed when Deborah Bird Rose asked modern Aboriginal people in the Victoria River district about it.

Allan Young, a Karangpurru man, said that a very long time ago a terrible sickness came upon the Karangpurru people. He identified the place of origin as a sickness site—Dreaming site of origin of illness—that marks a boundary between Karangpurru and their northern neighbours the Wardaman people. From there, he said, sickness devastated Karangpurru people and spread east to the Mudbura, west to the Ngaliwurru, and southwest to the Ngarinman people. These neighbouring peoples knew of the sickness and erected brush walls in an attempt to inhibit the spread of disease.

The disease occurred before the country was occupied, and the devastation caused by smallpox in Karangpurru people in open grassland was followed by the depredations of overlanders, pastoralists and miners. An original Karangpurru population of about 500 was finally reduced to 2 men, whose children identify themselves as Karangpurru. Neighbouring Ngarinman people, who had refuges in rough sandstone country with deep ravines and gorges, did not suffer as severely, but Nyiwanawu riverside people farther west were more badly hit by smallpox than others were, and their capacity for tribal warfare was seriously weakened.²⁴

Commentary

Although no active smallpox was seen by these observers, there is evidence of occurrences among Aboriginals in the Top End of the Northern Territory, certainly in 1861. Inland, pockmarked Aboriginal people were seen before and after the Overland Telegraph Line was completed in 1872. Eyewitnesses reported pockmarked Aboriginals in the south-west of the Gulf, on the Roper River, on the Cobourg Peninsula, at Darwin and on coastal rivers: the Adelaide, Alligator, Daly and Victoria. It occurred inland at Yam Creek and Pine Creek before 1870. Most tribes and bands named in records were affected, from the well-known Larrakiya to others whose identity was uncertain after change and depopulation. Foelsche, Robinson, Eylmann and Allan Young tell the same story of the spreading scourge that travelled around the coast and a long way inland, leaving a terrible trail of dead and disfigured victims.

Foelsche's opinion, that smallpox occurred about 1861, was based on the ages of survivors he met at Darwin before 1881, when there were 6 pockmarked victims aged from about twenty-five to forty, of whom Mangminone was one. Eylmann thought Wulwanga survivors in 1908 were then in their sixties, and had been infected as children more than forty years earlier. Some elderly people photographed by Basedow in 1922, including Daly Pulkara's father's brother, had probably acquired pockmarks as children some sixty years earlier.

There has always been uncertainty about the reported ages of Aboriginal people, and records of pockmarked people do include vague references to very old people who may have survived smallpox earlier in the nineteenth century, and were older than those known to Foelsche and Eylmann; but it is fairly clear the last significant outbreaks on the north coast were in 1861. Ralph Tate said smallpox had not occurred since Europeans arrived in the Northern Territory, and early settlers left no records of active smallpox. In the following years, Wildey and Eylmann probably saw bacterial infections that exacerbated scars, and their reports do not imply that there were more outbreaks after 1861.

The incidence of smallpox in the Top End in 1861 is usually underestimated, because records refer mostly to victims who had

severe permanent pockmarks that were still obvious when Europeans arrived a decade or more later. However, pockmarked Aboriginal people on the coast and rivers were noticed by observers for over fifty years, which suggests that the incidence in a relatively dense population had been high, when susceptible members of extended families who met in the dry season were exposed. Like the Kimberley people, those in Arnhem Land and elsewhere in northern Australia in the 1860s were unvaccinated, and did not have the outside support that had reduced mortalities near Roebourne, Geraldton and Dongara. With most people sick at the same time, they were subject to the crises of severe illness and social and economic disruption.

Survivors rightly described many deaths, although mortalities seem to have varied in different localities. Some mortalities were more severe. The loss of all but 30 of several hundred Unalla, who had accumulated since an earlier outbreak on the Cobourg Peninsula, may have resulted from more infectious contacts with arriving and departing fishermen than other clans had. The neighbouring Larrakiya population had also been depleted by smallpox outbreaks in the nineteenth century, including one in the 1860s; but by the early 1880s it was much larger than the Unalla population. These Arnhem Landers were more likely to die of smallpox than to acquire immunity to it through their relations with Macassans. As we have seen, many visitors from South Sulawesi were themselves susceptible to smallpox in the nineteenth and twentieth centuries, which did not help their Australian hosts.

Smallpox was probably why Nyiwanawu people disappeared before Europeans arrived, in a die-off that rivalled Unalla losses. Smallpox was the first reason why Karangpurru people were so savagely reduced, although British invaders accelerated their losses. Denser populations, such as some on the Victoria River, suffered badly. Conversely, Ngarinman people in rough inaccessible country with ravines and gorges would have had fewer infectious contacts.

Aboriginal reactions reflected the magnitude of the disasters of smallpox in isolated societies around the world. Its origins were identified with sickness sites, hoped-for remedies proved useless, the

deaths of victims were ascribed to the incantations of their enemies, and the number of the dead was exceptional. A mythical ancestor was said to have shown displeasure.

Estimates of tribal populations in the Top End in the decades after smallpox varied widely, from 500 to 300 or much less. Smallpox probably halved most of them in 1861, ten years before the permanent European occupation of Darwin, and twenty years before the Victoria River district was occupied. Eylmann's guess in 1908, that half the Wulwanga died, was consistent with some earlier estimates, and with mortalities in other naive populations. Diamond's suggestion, that Aboriginals in northern and western Australia, in country useless for food production, 'survived more or less intact', ignores the many smallpox deaths of the 1860s.²⁵

Alan Frost has suggested the situation of Aboriginal families in coastal Arnhem Land in the 1860s was likely to have been strikingly similar to that of people in family compounds in Nigeria in the 1950s, where smallpox spread slowly. But smallpox was heavily endemic in Nigeria, and spread slowly in a population of over 50 000 000, because many were immune after previous attacks or after vaccination. Equatorial rainforest also slowed its progress.²⁶ In northern Australia it probably spread slowly in Arnhem Land and elsewhere in the wet season, However, close contact with infectious visitors in the early dry season, followed by scattered Aboriginal cases in families along the coast, from the Cobourg Peninsula to the south-east of the Gulf, would have triggered outbreaks that continued unabated in the dry season. Except for those who had just recovered from attacks, and a few who had caught it forty years earlier, Aboriginal people, unlike Nigerians, were all susceptible, inexperienced, and unvaccinated. Not only would it have spread readily when they travelled, it would have spread more easily than it did in Nigeria, because it was a new disease and associated with sorcery or supernatural beings. When people fled to escape, it spread like wildfire, as Robinson said. The situation in the 1860s was more like that of scattered and isolated populations in the southern third of Africa mentioned earlier, which were free of smallpox until the eighteenth century, when three epidemics occurred in a hundred years.²⁷

Aboriginal smallpox in the Centre 1865–66 Northern Territory, Western Australia, South Australia

Newcomers to the Centre in the 1870s saw pockmarked people in several Aboriginal tribes. The telegraph officer ‘W’ saw marks on Aboriginals at Tennant Creek:

when in charge of Barrow Creek station on the overland telegraph line, I had occasion to visit Tennent’s Creek—a station 175 miles north—and I saw there many blacks whose skin showed all the appearance of small-pox; and as the natives there wear no clothing whatever, the multitude of marks on some of them was astonishing. Here, again, I saw a deaf and dumb black man, whose skin was marked all over as if he had suffered from small-pox, but as I am not a professional man, and for the further reason that converse with the Tennent’s Creek blacks was out of the question, perhaps these marks were caused by something else. In any case, the marks were precisely like those left on the skin after recovery from the disease, and as far as could be ascertained, were caused by small-pox, or something very like it.²⁸

Telegraph officer ‘W’ was not wrong. Astonishingly enough, over a century later, talk of the disease and references to its likely origin have persisted in a tribe at Tennant Creek. In Warumungu, as spoken in that area today, the word for ‘sick, a sore’ is *puurru*, like the Macassarese word meaning pustule, including smallpox pustule.²⁹

Other observers, whose contact with desert nomads lasted longer than the telegraph officer’s, saw pockmarked Aboriginals near the Telegraph Line. One was the Revd Louis Schulze. When he was at the Lutheran mission established on the Finke in 1877,³⁰ he saw diseases among Aranda people. At a meeting of the Royal Society of South Australia in 1891, he said: ‘The scars on the faces of some afford evidence that smallpox affected them some 16 or 18 years ago. They call the disease “*Pania*”. Some are said to have died of it.’³¹

In 1894 Lutheran Pastor Carl Strehlow began his twenty-eight-year mission to the Aranda at Hermannsburg. In an anthropological study, he described smallpox among them:

At some former time there must have been an invasion of small pox—the marks, or pitting on some of the natives is proof-positive.

This illness, which evidently travelled to them from the whites, or foreigners, caused many deaths among them. The charm doctors devices were wholly ineffective as also were the other remedies tried by the natives to cope with the scourge. They bathed the afflicted not only with . . . (pine), and . . . (two bushes) solutions, but resorted to drastic measures, in that they beat the livers of wild dogs soft and rubbed that into the pox-infected parts. Others of them made a salve from excrement and urine . . .³²

In 1908 Eylmann said: ‘I have myself seen only one old couple in Hermannsburg and one lubra on Sterling Station with pock-marked faces’. But he also described tribes that claimed knowledge of fatal diseases that spread from band to band: ‘For example, I heard from Arunta people that before the arrival of the white people, pestilential diseases claimed large numbers of victims from time to time in the interior’.³³

Over 600 kilometres west of the Overland Telegraph Line, explorers Ernest Giles and Harry Tietkins stopped in the Rawlinson Range in 1873. In a letter read at a meeting of the Royal Society of South Australia in 1882, Tietkins described the Range in the heart of the continent. It was uninhabited, waterless desert country covered with dense scrub of mallee and mulga, and not easily crossed even by the natives.

There they found a people quite isolated from the rest of the world, breathing the pure dry air of the interior, who wandered in small communities from place to place, who seldom camped or remained a whole day in one place, deeply marked with smallpox. What measures they took to prevent contagion or alleviate their sufferings, or how many were carried off, would probably be never known.

Of fifteen or twenty men who visited the camp eight were unmistakably [*sic*] marked with smallpox.

Giles, who explored country in the Northern Territory, South Australia and Western Australia, reported that in almost every part of the continent in which he has travelled, whether in settled or unexplored country, he has met Aboriginals pitted with smallpox, though he has mentioned the fact only once (at the Rawlinson Range) in published accounts of his explorations.³⁴

When Curr said no marks of smallpox were seen on Aborigines near Charlotte Waters telegraph station, one of his informants was F. J. Gillen, who began working on the Overland Telegraph Line when twenty years old in 1875. His duties included supervising repeater stations on the central section of the Line from Charlotte Waters to Tennant Creek, and he was appointed station master at Alice Springs in 1892. A Protector of Aborigines, he assisted the Horn Scientific Expedition to Central Australia in 1894. Stirling was also a member of the expedition. In 1911, when Stirling asked Gillen about Aboriginal smallpox in the interior, Gillen said:

thirty years ago when he lived at Alice Springs it was a common thing to see old natives pitted with small-pox all along the telegraph line from Charlotte Waters to Barrow Creek; but he saw no young natives similarly marked. Old blacks of the Arunta tribe, which occupies a large part of the tract of country just mentioned—that is the heart of Australia—had a tradition that a terrible disease traversed their country and destroyed great numbers of their people.³⁵

Mounted Constable Samuel Gason, who was at Barrow Creek in 1874 when Aborigines attacked the station, published an account of the Dyari and adjacent clans in northern South Australia that year. He described their diseases, including *mooramoor*:

Unquestionably smallpox, to which the natives were subject evidently before coming into contact with Europeans, as many old men and women are pockmarked in their face and body. They state that a great number have been carried off by this disease and I have been shown, on the top of a sandhill, seventy-four graves which are said to be those of men, women, and children, carried off by this fell disorder.³⁶

Commentary

Pockmarked Aborigines were observed in several isolated places in the Centre after 1870, probably due to smallpox in the 1860s. However, the word *mooramoor* was a common word, perhaps unknown to Gason, which referred not to the disease itself, but to a mythological ancestor thought to have caused it.

The presence of Aboriginal people who had apparently survived smallpox in arid Australia surprised nineteenth-century observers.

Some evidence of how smallpox may have behaved in scattered nomad populations is provided by observations during the smallpox eradication programme in Somalia. Face-to-face transmission was greatly reduced among nomads in open air; however, even among Mandeelo nomads, 19 cases occurred in a group of 46 people over a period of five months, while they ranged over an area about seventy kilometres by thirty-five kilometres. It was clear that smallpox did not always die out quickly in small sparse populations, where sometimes transmission was prolonged, but spread slowly, with one or two cases at a time.³⁷

Smallpox may have spread in the same way in arid Australia. People in the unsettled parts of the Northern Territory, Queensland, New South Wales and South Australia were not seen by Europeans until the Burke and Wills expedition in 1861, and Howitt's searches for the members of the expedition on the Cooper in 1861 and 1862.³⁸ He described the desert-dwellers on waterways and salt lakes north of the Flinders Ranges, whose trading activities and festivals linked them with neighbouring clans. Some Diyari and Yantruwunta people who met others from the Darling River back country, Lake Lipson and Sturt's Stony Desert, caught smallpox later in the 1860s.³⁹

We know that chains of connection linked people in arid regions who were drawn together by distributions of scarce resources and shared ceremonial lives. Nomad networks stretched south from the Gulf and the Cloncurry in Queensland to Port Augusta in South Australia, and west from the Darling to the Finke. Pituri, the narcotic from the Mulligan–Georgina district, was among resources exchanged in eastern Australia. It was widely distributed to north Queensland, Lake Eyre Basin and north-western New South Wales. Intersecting routes joined local and regional communities.⁴⁰

Communication between tribal populations necessitated travelling long distances, usually by more than one person, and always with permission from owners of territory being crossed. Diyari travellers were adventurous. Travelling north, they covered 400 kilometres from the Cooper River to the borders of the Simpson Desert to obtain pituri. Travelling south in winter, Diyari men and others covered 500 kilometres in two months on journeys to ochre mines in the Flinders Ranges. Farther west, Aranda men, chosen for endurance and dedication, were known as 'great travellers'. Travelling in

the cool of night with a companion, they could cover several hundred kilometres in two days, even in waterless country, when it was time to arrange ceremonies with kinsmen. On very rare occasions, youths accompanied initiated men.⁴¹

Local exchanges of commodities meant shorter journeys and more person-to-person contacts between the parties. So did exchanges at popular gatherings of some hundreds at ceremonies, where meetings, gifts and hospitality were usual. If they travelled during the incubation period after infectious contacts farther north, long distance Diyari, Aranda or other traders became links in chains of infection extending from north-eastern and northern districts to scattered bands of desert-dwellers. Chance infectious contacts with people travelling long distances while bartering scarce goods or attending ceremonies were the most likely cause of infection with smallpox in desert populations.

When the strange disease re-appeared after nearly forty years and there were unexpected deaths, fearful still-well susceptible people, some incubating the virus, would have fled. When several sickened, crises of illness and death continued, and some camps were left without adults to procure food, or elders whose support was needed. Messengers sent to tell kinsmen spread infection to adjoining clans. Once infection was present in a district, there were opportunities for it to spread. Contact infection was always possible among people who lived near each other. In Diyari beehive-shaped shelters, sealed with an insulating layer of sand, contact infection was almost inevitable in cold winter and spring weather, when smallpox was active. Aranda people in the Centre used wet-weather shelters when it rained. If one person happened to be infectious, daily activities afforded opportunities for transmission. Women and girls collected seed for hours, and ground roots at night as well, according to Schulze. In those conditions, smallpox spread slowly, and scattered new cases could have continued for months in desert families, until there were few susceptible people in some bands.

More than a decade after the ill-fated Burke and Wills expedition, the astonished 'W', curious Germans and the startled Tietkins saw sure signs of recent smallpox, which they could not explain, when they encountered desert-dwellers on waterways in arid regions. Outbreaks had occurred at Tennant Creek, Barrow Creek, Finke

River and Charlotte Waters in the Northern Territory, in the Rawlinson Range over the Western Australian border, on the Cooper and almost certainly at Lake Eyre in South Australia.

People who had acquired pockmarks in the 1860s were seen by telegraph officers and policemen after 1870, explorers in 1873, and Lutherans after 1877 when Hermannsburg was founded. In 1890 Schulze guessed an outbreak had occurred between 1872 and 1874. However, in 1908, Eylmann said victims in their sixties had survived as children, presumably in the 1860s.⁴² Aranda people, mentioned by Strehlow in 1922, might also have acquired permanent pockmarks, or badly infected scars, in the mid-1860s rather than earlier. Gason saw 'old' survivors before 1873, but how old were the seventy-four graves on the sandhill? Thirty years after he first saw them, Gillen recalled only old Aranda survivors, and believed that smallpox was a recurring phenomenon.

The incidence of smallpox in desert populations in the 1860s was erratic. It was high on the Finke, a popular meeting place with permanent water where visitors were frequent,⁴³ and pockmarked people were observed over a period of fifty years. By 1908 Eylmann remembered seeing only 3 cases there, although it had been a focus of infection. Nearly half the men that Tietkins saw in 1873 were pockmarked, nearly as many would have died. Some escaped the coincidences of infectious desert meetings. Two hundred warriors who escaped or survived smallpox were on the hill above Barrow Creek when Aboriginals attacked in 1873, and 'dusky forms appeared by scores'.⁴⁴ In 1894 Stirling saw no signs of it in the Centre, and Strehlow later did not say how many survivors he knew.

Many of those in isolated bands in desert country would have perished when very sick people of any age or either sex were too far from food and water, and the loss of people who gathered desert harvests endangered the survival of all. Mortalities made a lasting impression among survivors. Diyari people remembered losing men, women and children. Aranda thought smallpox was caused by demons or sorcery, to be counteracted by medicine men with powers they believed in implicitly.⁴⁵ They still spoke of the deaths in Gillen's day.

Records of smallpox in arid Australia are more than matched by records of it in African and American deserts. In East Africa in

the nineteenth century, it spread widely in Sudan and Ethiopia, where poorly vaccinated people travelled to avoid it. Mortalities varied, from a rate of 50 per cent among children and 80–90 per cent among affected adults. In New Mexico and Arizona after forty-five years without it, smallpox spread through the Hopi Tribe in 1898. Officials used quarantine and vaccination to control it, but traditional Hopis refused treatment, and caught smallpox when they travelled between pueblos for communal dances. Of 220 traditional Hopis who contracted smallpox, 163 people (74 per cent of known cases) died.⁴⁶ However, overall mortality in sparse desert tribes in arid Australia, where some bands escaped infection and the incidence of smallpox was relatively low, was a less significant cause of depopulation than it was in denser populations in Australia and North America.

Aboriginal smallpox in South Australia 1866–67

In 1866 active smallpox was seen in Aboriginals and several Europeans on the south-west coast of Eyre's Peninsula, hundreds of kilometres west of Narrinyeri territory, and south of Diyari land. At the end of the year, when local residents alerted government officials in distant Adelaide, the Colonial Surgeon recommended that Dr Robert Gething should visit infected districts. Gething migrated to South Australia in 1854 when he was twenty-eight. He had studied in London, then Edinburgh, where he gained a doctorate in Medicine. He had also travelled in Asia. He would have seen smallpox and pockmarks, and known more about it than colonial colleagues who had never seen active cases.⁴⁷ On 31 December 1866 he was told to visit Venus and Streaky bays to report on the disease said to be smallpox among Aboriginals. In the new year 'I left Port Adelaide per S. S. *Lubra* for Port Lincoln on the 5th inst and arrived there on the following day—I immediately on arrival placed myself in communication with Inspector Searcey of the police and Dr Lawson, who gave me every information they could'. The next day he set off by mail for Streaky Bay, 320 kilometres north-west of Port Lincoln.

Gething arrived on 9 January 1867, vaccinated children in the vicinity, and continued his way on horseback, with a police corporal to assist him. They explored surrounding country, looked

unsuccessfully for Aboriginals who had been at the police station, then returned to the Bay and vaccinated more children. On 12 January they rode forty-eight kilometres,

and there found the natives who had been suffering from an eruptive 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 3 or 4 deaths directly traced to that cause. At this station (Oliver's) I vaccinated all who had not been previously. The next day (Sunday) the natives I had seen the previous day came down to me at the Bay bringing with them others who 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 another occasion. On the Monday I vaccinated some who had not been seen previously. The next day (Tuesday) I left Streaky Bay for Venus Bay accompanied by Corporal Provis with a view of vaccinating between the two bays. About midway I stopped at Mr Schlunke's station where I saw a young man and his sister, relatives of Mr Schlunke, the only Europeans who had been infected, having been previously vaccinated, its effect had been comparatively mild, leaving only a few traces upon the neck and face. I proceeded from thence to Venus Bay . . . hearing there were some blacks in the neighbourhood I visited them, and found a young female aboriginal named 'Chilminga' suffering unmistakably from pure Variola then having the secondary fever, this the most recent case I saw quite decided the opinion I had formed from seeing the first cases. I could gain no reliable information respecting the length of time it had been remarked nor its probable origin among the natives, but the Police at Streaky and Venus Bays informed me that the first cases they had noticed, were about the latter end of October or beginning of November last, and that it was confined to the adult natives most of whom had had it to the number of 200 or 300 at Streaky Bay, and 60 or 70 at Venus Bay, and that there had been seen 30 or 40 ill at the same time. I could only ascertain that 6 or 8 had died directly from Variola. I may also add that my information was very imperfect as to the early stages of the Epidemic owing to their utter inability of the natives to express their symptoms. I of course vaccinated all I could meet with during my journey of 21 days. I left instructions, vaccine,

lancets, points etc with Corporal Provis at Streaky Bay, an exceedingly intelligent person; and in my opinion quite capable of undertaking such a duty; he promised to communicate with me should he require a fresh supply of vaccine—It may not be superfluous for me to add that I attribute the comparatively excessively small mortality among the natives in this instance, to some peculiarity of diet or habits of life and that the Europeans have suffered so little I imagine may be from the slight communication they have with the blacks, their very scattered population and nearly all adults having been previously vaccinated I am quite unable to decide as to its origin and therefore was at first in some doubt as to its character.⁴⁸

Back in Adelaide on 30 January 1867, Gething sent his report to the Vaccine Board, and offered to give any more information he could. Within a few days the report went to the Chief Secretary, the Colonial Surgeon, the Commissioner of Crown Lands and the press. It was years before there was other information from him, but before Gething died in 1883, Professor Tate expressed interest in his investigation of Aboriginal smallpox. When the Royal Society of South Australia met in 1882, Tate said he had talked to Gething, who was unsure of his earlier opinion: ‘he was not prepared to deny that it was not smallpox’, there was not enough data to trace its spread or settle the question of its origin. However, ‘The Natives at Streaky Bay declared that the disease came to them from the North’.⁴⁹

Curr’s informants at Streaky Bay were D. K. Richardson, and Gething’s companion of 1867, Charles Provis. Their vocabularies did not include words for smallpox, nor did Curr’s remarks. Mrs Richards of Fowler’s Bay, a corresponding member of the Royal Society of South Australia, had seen the outbreak and remembered Dr Gething.⁵⁰ In 1882 she said:

at the end of 1866 and the early part of 1867 the natives of Streaky Bay and Fowler’s Bay had what was supposed to be smallpox, great numbers of them dying. A few of the affected were still living, and very much pitted, more especially an old lubra, who was blind; although constantly with them, no white person was known to have taken the disease.

Another South Australian who remembered the epidemic among the Aboriginals was Henry Cowell Hawson, who settled at Port Adelaide in 1837 and became prominent in the Port Lincoln district. He had probably never seen active smallpox until he saw it at Streaky Bay:

Having occupied a station at Streaky Bay, in the colony of South Australia, in 1865, when the disease was very prevalent in the nature of an epidemic among the natives of the western district of that colony, many instances came under my notice, and one in particular, which could go far to show that the disease was wholly different from smallpox, though similar in some respects. The one instance I would offer as an illustration was that of a native lad of about eighteen years of age, who had been employed about the station, who was attacked by this disease at shearing time, when there were many whites about, and who had not recovered when the shearing was over, and who at one time was wholly unable to move or to assist himself in any way. During the whole of the time of this lad's illness my second son attended to him, carrying him each evening into a hut, and taking him out (as was his own wish) to his camp every morning. As he was in close contact with my son on these occasions, and he escaped the infection, I should suppose the conclusion to be that this disease could not possibly have been small-pox, or my son would not have escaped. I may here observe that owing to the care taken of him, the native lad recovered. Many of the natives died, but I think more from neglect and want of care by their own friends, than from the effects of the disease. I may also say that the natives were about at many stations suffering from the above-mentioned disease, that there were children at many of the stations, and that in no instance did the infection extend to any of the whites.⁵¹

Stirling saw unequivocal evidence of smallpox among Aboriginals in southern localities in South Australia in the late nineteenth century. In 1896 he said:

On the Horn Expedition I saw no marks which could be said to be those of smallpox, but I have frequently seen, in more southern localities in South Australia, where small pox has never gained admittance from beyond the sea, such pittings of the face as could only have been caused by variola.⁵²

Commentary

There was an outbreak of smallpox among Aboriginal people at Streaky Bay and Fowler's Bay on the south coast of South Australia in 1866 and 1867. Victims said it came from the north, and it is reasonable to speculate that contacts continued from south of the Centre until it reached denser populations on the coast. Stirling saw survivors in southern districts whose disease probably spread south from Lake Eyre. It was not recognized by many Europeans, but Gething called it 'pure variola', and immediately vaccinated blacks, who accepted treatment because they were afraid of smallpox. It spread on the coast at Streaky Bay and Fowler's Bay for six months.

The incidence was high, with about 4000 known Aboriginal cases. It was affected only marginally by vaccination. Sick station Aboriginals probably benefited from distributions of food. Chilminga and perhaps other young people survived, and pockmarked survivors were still seen thirty years later. However, many were neglected by others who were sick themselves. Incidence and case-fatality rates were underestimated by Gething because his investigation was very limited, but Mrs Richards and Hawson later said many had died, a conclusion in keeping with mortalities in naive subjects. Beyond the frontier, mortalities in affected groups were probably higher. The incidence among Europeans was negligible, because they had little close contact with infected Aboriginals, and most had previously been vaccinated. As far as we know, smallpox did not occur among settlers in Queensland, the Northern Territory or South Australia before the 1880s,⁵³ except at Schlunke's station between Streaky Bay and Fowler's Bay.

Conclusion

Unlike other infectious diseases to which Aboriginals were exposed after 1788, smallpox came from infected non-Europeans from the north. The presence of active smallpox in eastern Australia after 1860 was unknown to most colonists at the time, except in far-off settlements on the coast of Eyre's Peninsula. By the 1870s its recent presence among Aboriginal people was apparent in newly occupied

parts of Queensland and the Northern Territory. For the first time there were Europeans in remote parts of the interior, in north-eastern, northern and central Australia who saw traces of smallpox among Aboriginals, and kept records that establish that in the 1860s smallpox spread from the north coast to the east coast and the south coast, as it evidently did in the 1820s, and very likely did in the 1780s. In the next chapter, we will find that it also spread down the west coast in the 1860s.

The Aftermath

Except in Western Australia, the destruction of the lives of indigenous Australians caused by smallpox in the 1860s was never recognized. Europeans had scarcely penetrated north and west Queensland, and Curr had few informants there who were familiar with pockmarks. Foelsche and Giles were his only informants in the Northern Territory, and he did not mention smallpox in South Australia. Nor did he acknowledge it occurred ‘after 1845’ in eastern Australia. Like Curr, Radcliffe-Brown ignored its impact in Queensland, the Northern Territory and South Australia in the 1860s. Using Radcliffe-Brown’s *minimum* population estimates, White and Mulvaney overlooked its final appearance in eastern Australia.⁵⁴

The sheer loss of numbers due to smallpox jeopardized the survival of clans such as the Unalla, who were nearly exterminated. Others tribes lost elders soon after Europeans occupied tribal lands. Settlement followed, preventing the restoration of traditional life. Smallpox, and the loss of numbers and morale, coincided with the pastoral invasion of marginal lands in eastern Australia after the Burke and Wills journeys.

Frontier violence, like the violence between Aboriginals and Europeans in the 1850s and early 1860s in central Queensland, subsided after smallpox attacked the invaded people. Aboriginal reactions were often subdued when settlers arrived after smallpox outbreaks; some Aboriginals in Queensland and South Australia, who had survived smallpox in 1830, were infected with tuberculosis after contact with Europeans. As Palmer knew, tuberculosis, common in settlers, followed smallpox again into Queensland in the

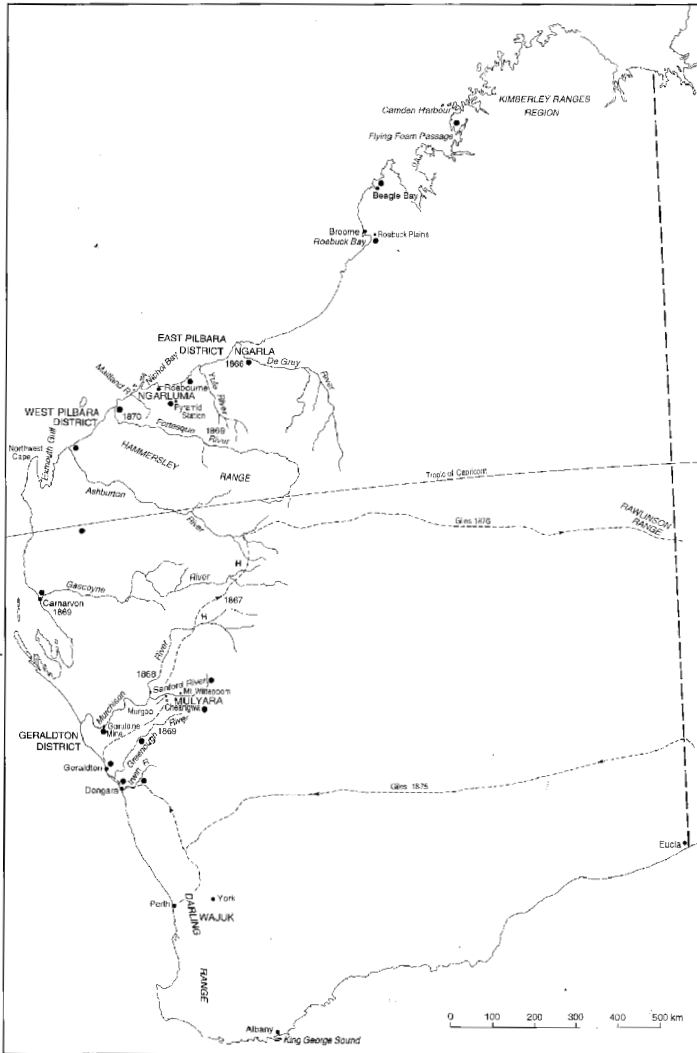
1870s, while in the Northern Territory, British newcomers working on the Overland Telegraph Line and Chinese miners transmitted it to Aboriginals.⁵⁵ Yet resistance continued, with a new generation of warriors, decades after smallpox occurred. Kalkatungu tribesmen fought settlers near Cloncurry in 1884. There were more attacks on settlers, and 15 European deaths between 1878 and 1884 in the Top End.⁵⁶ In the Victoria River district, some families survived smallpox and resisted invasion. But by the 1880s, survivors of smallpox had lost most of their land.

9

Western Australia 1860–1870

There were no cases of smallpox related to occasional introductions in colonial ports apart from 16 cases after the *Commodore Perry* arrived at Melbourne in 1857, and the European population had remained free of it. For decades, the origin of smallpox among Aboriginals in eastern Australia in 1789 and 1830 eluded people who saw its results. Active smallpox had not been seen in Aboriginals anywhere, except by a few settlers on the remote south coast of South Australia in the 1860s. Its unexpected presence in indigenous populations in eastern Australia was unknown to most of the European population. However, during the same period in Western Australia, active smallpox and pockmarked people were seen in various parts of the colony by concerned people in the small settler population. They knew something about smallpox, provided care, and talked with survivors.

In Western Australia the arrival of ships at Fremantle, the occasional presence of fishing-fleets on the north-west coast, and the presence of smallpox in the islands of the Indonesian archipelago in the near north provoked anxiety about introductions of it well before active smallpox was seen in Aboriginal populations. The presence or absence of smallpox or its traces was always a matter of concern to the isolated colonists. In the 1840s a Swan River settler reported seeing several old Aboriginals who were deeply pitted with



Key: ● outbreak H hypothetical outbreak - - - Giles' route

Western Australia: smallpox outbreaks 1862–70

Outbreaks lasted at least a month in any one locality, and could continue for six months with related outbreaks in adjacent areas. In sparse populations, outbreaks consisted of a few scattered cases; in dense populations, they affected hundreds of people. Between 1865 and 1870, there were outbreaks at Beagle Bay, Broome, on the De Grey River, at Roebourne, Geraldton, Dongarra, Exmouth Gulf, on the Fortesque and Maitland rivers.

what he thought was smallpox. The Colonial Surgeon speculated that it might have been introduced by the French, or by some whaler, at the end of the eighteenth century.¹ But there is no other evidence of the early presence of smallpox in Western Australia, there were no sure signs of it among the indigenous people in the 1830s, and Benedictine missionary Salvado said it was ‘quite unknown to the Australian native’ when he was at New Norcia in 1850.²

There were 5000 Europeans in Western Australia in 1850,³ and there had not been any shipboard introductions to cause outbreaks of smallpox. Nevertheless, members of the medical profession were anything but complacent about its absence. In 1854 the surgeon H. Burnham Bryan of Busselton said he intended to vaccinate Aborigines. He thought it would be difficult to collect the separate tribes, but he believed that ‘Humanity itself should of course do all in its power to avert the scourge inevitably pending’. The Perth and York Guardians arranged for Aborigines to be vaccinated.⁴ In 1858 Colonial Surgeon John Ferguson recommended that a supply of good cowpox virus should be obtained from the South Australian Government, and the virus should be ‘carefully put up . . . into carefully sealed tubes’. He and his colleagues thought of getting a supply from Marseilles. One surgeon got vaccine from London by mail.⁵ Early in 1860 the Principal Medical Officer of the Convict Department submitted an ambitious plan to the Governor, and stressed:

the urgent necessity of having established ‘Vaccine Depots’ in all the principal towns of this Colony, with the view of having the important operation of vaccination effectively performed, thereby warding off the dreadful scourge of Small Pox—should such a loathsome disease make its appearance in this Colony. I confess my apprehension that should Small Pox visit this Country, its ravages would indeed prove generally destructive—In British Africa—a climate similar to this, Small Pox has proved imminently fatal to the colored population.⁶

Six months later, when plans to contend with the likely arrival of smallpox in the colony were under way at Perth, Aborigines in the south-west were exposed to measles, probably for the first time, after a case on a passenger ship was missed by quarantine officials at Albany.⁷ In August 1860 the Guardian at Albany reported that great numbers of Aboriginal people were suffering from measles,

and several had died. In December he said 'the epidemic is still making great destruction amongst the natives . . . I consider that more than two hundred thro' the whole district have died.' Farther north at York, the *Guardian* reported that Aboriginal people with measles were supplied with food by settlers and their shepherds throughout the epidemic, and none had died from neglect or want of food.⁸ But 'Several have lost their lives from their friends pouring water over them in the heat of the fever, and from plunging into the pools when in a state of delirium'. He warned that it would be more fatal in unsettled districts.⁹ According to settler Mrs Edward Millett, 'It spread widely and rapidly, assuming a very virulent character, more especially among the natives, of whom so many died that both they and the colonists in alluding to the visitation spoke of it in terms that would have been almost applicable to a time of pestilence'.¹⁰ Mortalities were probably like those in islands in the Pacific where, as a new disease, it killed a quarter of its victims. It was less destructive than smallpox.¹¹

In the second half of the nineteenth century, smallpox was increasingly prevalent in the Indonesian archipelago, and the forebodings of anxious members of the medical profession in Perth about the damage it could cause were justified. Indonesian fleets had already been seen on the north coast. Fleets that visited the north-west coast occasionally included fishermen who had been infected in their home ports. Some trepanging praus seen off the Kimberley coast sailed from Sumbawa, where members of the Bugis population were engaged in the Australian trepang industry. Others came from Sumba or neighbouring islands, and some came from Macassar. They were known as 'Malays', as they were on the north coast. The voyage to the north-west coast was more hazardous than the voyage to the Top End, but the trepang was superior.¹² Trepangers probably brought smallpox to Western Australia less often than to the north coast, because the perilous voyages were less frequent, and the exceptionally rugged and indented Kimberley coast limited face-to-face contact between the Australians and their visitors.

All the same, it was inevitable that 'Malays' would eventually bring smallpox to the Kimberley when the fleets were large and smallpox was endemic in the islands. Once it was established on the Kimberley coast in the early 1860s, it spread into unexplored and

newly settled districts from far north-western Western Australia at least as far as Dongara, several hundred kilometres north of Perth, and it was clear that fears about Aboriginal smallpox expressed by members of the medical profession were well founded. Frightful outbreaks were seen in the northern districts of the colony from 1866 to 1870. The descriptions that followed, of deserted camps and unburied dead, of many dead at some camps and starving victims at others, were typical descriptions of virgin soil smallpox. A small number of people in the European population of the colony, which had grown to about 20 000, reported widespread destruction. Some officials and settlers were eye-witnesses of outbreaks, some provided support at the time, and talked with survivors years later, as Daisy Bates did in the twentieth century.

These outbreaks were not publicized outside Western Australia, but several accounts of Aboriginal smallpox eventually appeared when E. M. Curr published *The Australian Race* in 1886. The only professional medical account was published by Cumpston in 1914 after research in Western Australia. Much of the Australia-wide historical evidence of Aboriginal smallpox, including Western Australian evidence, has never been readily available. In this chapter, the evidence and its interpretation is discussed in the appropriate context of the epidemiology and history of smallpox in Native Americans. Because of the great size of Western Australia and the time it took for smallpox to spread from north to south, evidence from four distinct geographical regions is considered in chronological sequence.

Aboriginal smallpox in the Kimberley 1862–65

Writing in 1881, Foelsche said smallpox had travelled down the west coast from Darwin after being introduced there in 1861,¹³ but there is no reliable evidence to support this speculation. The only literate observers in the Kimberley who could have seen Aboriginal smallpox in the 1860s were a few settlers and a government party at Camden Harbour in 1864–65. In April 1865 they were surprised to see seven praus, thirty canoes and 300 Indonesian fishermen, who stayed for some days to fish for trepang. Although west coast settlers later said Aboriginal smallpox had been introduced by these

visitors, neither the Resident, R. J. Sholl, nor his Medical Officer, C. S. Bompas, reported Aboriginal smallpox, and there is no evidence that Sholl saw it before the settlement was abandoned in October 1865, or at Roebuck Bay farther down the Kimberley coast later in the year.¹⁴

However, Alexander Robert Richardson later reported that he had seen Aboriginal smallpox on the north-west coast soon after that time. He had left Victoria with relatives in 1865 to take advantage of land opportunities in Western Australia. They established Pyramid Station on the George River, forty kilometres south-east of Roebourne, where Richardson lived until 1876. His memoirs, written in 1909, recorded: ‘that deadly epidemic smallpox, which about the year 1867, coming down the coast from perhaps Camden Harbour where Malays in search of beche-le-mer visiting the coast had inoculated [i.e. infected] them with the disease, carried them off in hundreds if not thousands’. It might have been 1866.¹⁵

Another west coast settler who linked Aboriginal smallpox with ‘Malays’ was John Perks from Cheangwa. He travelled north up the Kimberley coast years after seeing instances in the settled south. Curr quoted Perks.

the Blacks of the Victoria District say the disease came to them from the north; that he has been as far north as Camden Harbour, and that the further he went in that direction the more disastrous he found the disease had been, and the more prevalent the marks which it leaves behind; that at Roebuck Bay nearly all who have survived are scarred and pitted more or less by it. Between the various tribes from the De Grey River to Champion Bay frequent communication is kept up. The outbreak of smallpox . . . in this part of Western Australia, is, I learn, generally attributed by the Whites to the Malays, who are known to have frequented the west coast about that time. In 1864, also, several Malay proas entered Camden Harbour, and the Whites located there prevented their crews from landing.¹⁶

Daisy Bates recognized pockmarks decades later, on Aboriginals at the Trappist Mission at Beagle Bay, south of Camden Harbour. She noticed traces of smallpox again when she stayed at Broome, and when she lived on a station on the Roebuck Plains for a year. She believed it was introduced in the 1860s, and ‘carried off numbers

of victims'. She heard about *walmbing*, which was 'a kind of chicken-pox or smallpox, introduced from eastward or north-eastward'; and that sea bathing was the only remedy available. She said no more about smallpox.¹⁷

Commentary

After Foelsche saw Indonesian fishermen at Darwin in the 1870s, he assumed they had introduced smallpox on the north coast, and that it spread down the west coast, while settlers assumed that fishermen introduced it somewhere on the north-west coast of Western Australia in the 1860s. The differences are insignificant, because whenever smallpox was prevalent in South Sulawesi or other islands, face-to-face contacts between trepangers who became sick on the voyage or after arrival and Aboriginal people they happened to meet would have caused smallpox to break out in families in wet-weather camps. After it spread slowly in the wet season, infectious contacts would have increased dramatically in the dry season, with its ritual, social and economic obligations. This may have happened in the north of Western Australia in the mid-1860s. Smallpox would have spread readily in relatively dense Kimberley populations in coastal districts and on major waterways such as the Fitzroy River and tributaries. At such times, sickness, death and fear disrupted customary life, and people travelled to escape impending disasters. Like others who fled from smallpox, panic-stricken Kimberley victims took it with them when they sought friends and kinsmen at times of crisis and bereavement.

No outbreaks were seen during Sholl's sojourn at Camden Harbour before he left for Roebourne at the end of 1865, but in the next three decades, pockmarked survivors were seen from Camden Harbour in north Kimberley to Roebuck Plains, Beagle Bay and Broome in south Kimberley. Judging from what Perks saw, the incidence of smallpox was high on the north Kimberley coast, and farther south at Roebuck Bay, where 'nearly all' the survivors were scarred and pitted. High mortalities in adults who provided food caused economic disruption and starvation. Richardson's account of disastrous mortalities was based on the Roebourne district, but he later visited the Kimberley, and his rough estimate that smallpox

killed ‘hundreds if not thousands’, applies there too, as Daisy Bates later confirmed.

Aboriginal smallpox in the East Pilbara 1866

One of the first settlers to see active smallpox among Aboriginals in the north-west was Charles Harper, who saw it on the De Grey River. A native-born settler from the south-west, he sailed for Roebourne in 1866 and spent a year exploring, boat-building and learning the local Aboriginal language. During a smallpox outbreak in August 1866, Harper visited Roebourne and reported that a European on a neighbouring station was ‘laid up with small pox’.

Much later, when Curr asked about smallpox in the Ngarla tribe on the De Grey River, Harper said that in 1865–66, he was an eye-witness of an outbreak of smallpox that came from the north and passed over the De Grey River country; that large numbers of the Ngarla died of it; that many survived its attack; and that a few Europeans suffered from it lightly. Many of the Aboriginals who died were left unburied, and Mr Harper saw camps long afterwards in which their bones lay bleaching on the ground. The tribe called the smallpox *boola*, a term applied to anything nasty or poisonous.¹⁸ Another De Grey settler wrote to the Perth *Inquirer* at the time, advising the vaccination of Aboriginals, ‘who, a short time before, had been dying in great numbers from small-pox . . . the Whites on the station did what they could to relieve them’.

In May 1866 C. H. Elliott, Medical Officer of the Victoria District, and A. Durlacher, the Resident of the District, informed the Colonial Secretary that smallpox was prevalent at Nickol Bay on the newly settled north-west coast, and some settlers refused to have their children vaccinated. The Resident was sufficiently alarmed to enforce the provisions of the Vaccination Ordinance, and the penalty, in all cases of neglecting to have children vaccinated.¹⁹

The Nickol Bay tribe was several hundred kilometres west of the De Grey River. Its territory, which stretched from the Maitland River to the Yule River and inland, was occupied by European settlers in 1864. When smallpox broke out in 1866, Richardson saw it at close quarters. He told Curr that the Nickol Bay tribe consisted of about 250 or 300 people when he first knew them, but they had

decreased considerably as a result of smallpox the following year. The disease had come from tribes farther east:

there can be no doubt of its having been genuine small-pox, often confluent, and that the Blacks, terrified at the horrible and unknown malady, fled from those amongst them stricken with it; that many of the deserted sufferers were supplied with food and drink by the Whites, none of whom, however, took the disease, except a few children who had not been vaccinated and had it in a mild form. Many Blacks are still living who display the marks commonly left by the disorder.²⁰

When he published his reminiscences, Richardson described the Aboriginals' fear of infection and the abandonment of sufferers, and recalled the astonishment of some who were looked after by Europeans and recovered. He recollected that the Europeans did not know how long immunity conferred by vaccination lasted, but expected that childhood vaccination would give some protection. The 1866 outbreak 'certainly gave one a very realistic idea of what a smallpox outbreak is like where neither medical skill, nursing comforts and accessories—nor vaccination—was possible'.²¹

As a student in ethnology at Cambridge in 1911, A. R. Radcliffe-Brown investigated three tribes of Aboriginals in the Pilbara. One was the Ngarluma tribe, whose territory extended from the Maitland River to the Sherlock River, and inland. He used Richardson's account of the Nickol Bay tribe in *The Australian Race*. He acknowledged that a large number of them died during an epidemic of smallpox in 1866, and referred to an outbreak of measles that followed. He concluded that the 250–300 people described by Richardson as the Nickol Bay tribe were only a part of the tribe, and that the whole tribe numbered at least twice as many. The tribe was divided into local groups, with not less than 30 people in each. The minimum for a tribe was about 750. In 1911 there were employed on sheep stations, 60 people who were the only remaining members of the Ngarluma tribe. Radcliffe-Brown again referred to smallpox in the Ngarluma in his 1930 estimate of former numbers of Aboriginal people.²²

Members of the Sholl family who were in the Roebourne district between 1866 and 1870, in particular the Resident himself, left

records of smallpox outbreaks. A family background of medicine and journalism heightened their concern. They had emigrated to Western Australia in 1840. The oldest of the Sholl brothers, William Horatio Sholl, was appointed to act as Colonial Surgeon in 1841. Acrimonious relations with the Government prevented his permanent appointment, and he edited the weekly *Inquirer* from 1847–49. Another brother was Robert John Sholl, the Roebourne Resident, who had abandoned his medical studies when he left London. In 1849 he joined the *Inquirer*, then edited the *Commercial News and Shipping List* at Fremantle. He was secretary of the Geraldine Mining Company and the Roebuck Bay Pastoral Company before being appointed Resident at Camden Harbour and Roebourne. His son Horace Sholl, a pastoralist at Yule River, told Curr about the Nickol Bay tribe.²³

Smallpox was Robert Sholl's foremost concern in 1866. One of his reports was about the numbers of Aboriginals who were dying from an unnamed disease, and whose bodies were lying about the countryside.²⁴ When a party of settlers, including the Resident's son Trevarton, returned to Roebourne on 8 August 1866 after an expedition to Exmouth Gulf, they were told that there were cases of smallpox at the settlement. The next day, Trevarton found the settlers were *not* all well, and that one young lady was having her beauty spoilt with 'this wretched disease'.

In September, Sholl advised the Colonial Secretary that: 'The small pox which has heretofore been confined to the Aboriginal population has I regret to state attacked some of the children of the settlers'. He elaborated. A previously vaccinated child of the settler John Withnell was first attacked, then another unvaccinated child of his, the assistant surgeon Wedge's unvaccinated infant, and two previously vaccinated Woodhouse children. The only adult attacked was Emma Withnell's sister, Miss Hancock. Emma, who managed their station when John went pearling, helped him to run a butcher's shop on the outskirts of the township. Apparently she was already vaccinated when she looked after her sister and the children, and nursed and vaccinated many Aboriginals.

Speaking of European patients, Sholl reported that 'The disease was in every case of a mild type, the patient easily recovering and not being in any way disfigured'. But he knew that several children

were unvaccinated. He feared that more virulent attacks might occur, and asked for a supply of vaccine lymph. At the end of September he added that there had been 7 cases in the total European population of 124 men, women and children, that only one was an adult; and 'I am not aware of any cases of Small pox at this stage among either the white or black population'. Several years later Sholl said that when the disease occurred in 1866 many natives, but no Europeans, had died. In 1872 early settler A. S. Roe remembered Aboriginals with pockmarks in the Roebourne District, and north-west settler McKenzie Grant said: 'We lost one half of them through it'.²⁵

Commentary

In 1866 smallpox spread from the Kimberley to districts on the north-west coast that are now known as the Pilbara. Infectious contacts from south Kimberley triggered outbreaks on the De Grey River, which Harper saw after he arrived early in 1866. Shocked and fearful people abandoned camps and deserted victims. After frequent contacts between Aboriginal people, infection spread some distance inland on waterways, and several hundred kilometres westwards on the coast as far as the Roebourne district. The worst fears of the medical profession were realized when smallpox broke out in the Withnell family on the Harding River, and on the George River south-east of Roebourne the year after Richardson established Pyramid Station. It attacked Europeans as well as Aboriginals on stations, spreading between the two races. Richardson realized that Aboriginal people were terrified by the unknown disease and fled from those stricken with it, so continuing its spread. By the end of September 1866 the Roebourne Resident did not know of any new cases of smallpox among Aboriginals or Europeans in his district.

The incidence of smallpox was high east of Roebourne in 1866, so were mortalities. McKenzie Grant's description of its impact in the north-west was close to recent demographic estimates of smallpox mortalities. Sholl, who was familiar with smallpox, thought the victims most likely to survive were children and young men and women, a feature also seen in modern epidemics. Mortality was reduced to a small extent when settlers provided food and care. Emma

Withnell's nursing saved some, so did vaccinations that she and a few other settlers performed in east Pilbara.

The first European cases in the Roebourne district, which were also the first settler cases in Western Australia, were caused by contacts with sick Aboriginals and infection spread to other susceptible members of settlers' families. There were few European cases in 1866, because many had been vaccinated in infancy, but several suffered mild attacks. The small number of European cases also reflected a lack of close contact between the two races.

There were no literate observers in unexplored semi-arid and arid country from the De Grey River to the Hammersley Range to leave records of Aboriginal smallpox in the 1860s. Its impact was probably less severe there, where it would have spread slowly in small scattered desert populations on intermittent watercourses, than it was in districts with better resources. Nevertheless, there evidently were some cases in both unexplored and newly explored districts in the interior, where infection followed the communication networks that Perks mentioned between the De Grey and Champion Bay (as Geraldton was once known). Clans exchanging goods and sharing rituals made contacts on waterways and at desert wells.

Aboriginal smallpox in the Geraldton District 1868–69

Perks was Curr's informant at Geraldton and in the surrounding district. Perks arrived in Western Australia as a convict in 1854, and was employed in the Victoria District when William Burges was Resident, and held positions of trust for his family. He was a shepherd and overseer on their stations, searched for new land and moved stock for them in the 1860s. A well-read man, Perks later supplied Curr with a vocabulary of language spoken at Cheangwa on the Sanford River, and with dialectic differences, from the Murchison to the Irwin. It was used at Geraldton. Perks worked on Bowes and Yuin stations, north-east of Geraldton, and saw Aboriginals with smallpox for the first time there about the end of 1868 (not 1858, as Curr reported). Quoting Perks, Curr said that:

They called the disease *Moolya errill-ya-rill-ya*; had no remedy for it but incantations, and no fear or knowledge of its infectiousness. Numbers of those attacked died, and were buried. Others were de-

serted in dismay by their friends before death, and their bones still bleach in the sun. Some found nurses in the Whites on the stations, none of whom contracted the disease. Of the individuals who survived, a moiety bear the marks of smallpox; their faces, in many cases, being fearfully pitted, scarred, and furrowed. To judge by the marks left, more men seem to have survived than women, and more women than children. Mr Perks notes that he never saw White men so fearfully marked. He adds that on the occasion of this outbreak of smallpox, many of the Blacks were induced, and even compelled, by the colonial authorities, to present themselves to the medical officer stationed at Champion Bay, and were there vaccinated.²⁶

In January 1869 A. Durlacher, the Resident at Geraldton, advised C. H. Elliott, the Medical Officer, that:

Small pox in a virulent form is among the Aborigines to the Northward and Eastward, and that numbers have died therefrom. This fearful disease is now within 50 miles of the Geraldine Mine.

I trust you will make this known to the Central Vaccine Board at Perth so that you may be at once provided with Vaccine Matter, as I estimate there are at least two hundred children in the District who have not been vaccinated.

In March 1869 Elliott wanted the Government to find out what the disease was. He had consulted colleagues, and ‘I believe from what Dr Bompas has stated to me personally, that the disease is a mild form of Smallpox’. The 1870 *Western Australian Almanack* reported that in May 1869 smallpox was very prevalent among indigenous people in the Geraldton district.²⁷

South of Geraldton and east of the Irwin River, Police Constable James Watson reported to the Resident on 10 March 1869, when some Aborigines were getting better:

there are not so many sick at present, but a good many of them are in a very low state. There are no white people sick with it up here. Nor any Natives on this side of the Darling Range, 30 miles from my station. I continue sending the flour tea and sugar around to them with the old pack horse. There is now left a little more than a bag of flour 35 lbs of Sugar and 7 lbs of Tea. The pack starts again tomorrow with another load for them. These supplies have saved the lives of a great

many Natives who could not have got their living otherwise as many of them were left to die by those that were able to walk away and leave them.²⁸

In the following weeks, Watson saw the Geraldton Resident and the Magistrate in the Greenough office, and told them that a disease resembling smallpox was raging among the Aboriginals 100 kilometres to the eastward of the Irwin. They were in a deplorable state, and ‘he describes them as dying daily from small pox, their end hastened by want of food, as the Natives not affected leave them to their fate’. The Resident and the Magistrate issued flour, tea and sugar for Watson to distribute immediately. At the same time, Bompas informed the Resident that Mr Pascoe’s children were suffering from ‘a complaint which he declares to be a modified form of “Small Pox”’. His temporary appointment was urged.²⁹

On 27 March 1869 more supplies arrived at Watson’s station:

I at once sent off a Packhorse loaded with tea sugar and flour to a camp of Natives sick on the North Branch at the same time I told him where to meet me on the 30th. On the 29 I started with my team with rations for the Sick Natives and took another Pack horse to convey it to them through the thickets. On the 30 at 9 am the Native sent on the 29 returned with the Pack horse. Stated that he had given the rations to the Sick Natives also stated that a great Number was dead and that there was a great number sick and that some had got better and was gone Back in the thicket far away and state that there was two more lots sick. I at once started two Pack horses loaded with flour tea Sugar to them and one on foot to a Station where there was 5 Sick. On the 31 they returned to me with the Packs. they told me . . . where there was another lot Sick. I packed them off again with another last week. At 5 pm they returned and stated that all the sick they knew of had got flour tea and sugar to last them for some time. I then proceeded to one of Mr [Foster’s] cattle stations. there I found 35 Natives and about 15 of them sick.

Watson and an Aboriginal continued rounds with rations in April:

from what the Natives tell me there is but little doubt but what it is the Small Pock. Some that has got better it as left them much Marked . . . two White men as told me the same thing that as had the Small

Pock themselves. I have not seen any myself since nor do I wish to see them. Dokter Bompas as been twice to my Station and vaccinated one of my children and in neither cases as it taken . . .

I shall travel around the Natives again with the Pack horse again with rations to the Sick Natives. I think it is decreasing. I do not think it is spreading.³⁰

When Watson's report was forwarded to the Colonial Secretary, the Magistrate from Greenough added that on 5 April 1869 the Constable mentioned one instance in which 'a whole camp of 17 in number died before assistance could reach them'. In his opinion the mortality would have been greater but for the provisions with which they were supplied for the benefit of the sick.³¹

Durlacher and Magistrate Maitland Brown also reported that a ticket-of-leave man named Maughan on a station at Dongara was suffering from smallpox: 'He is a mass of ulcers from head to foot, save his face which is merely discoloured'. When Maitland Brown saw him, he was 'prostrated by the disease and in a very precarious condition'. The magistrate thought that if there were more cases, a 'Small Pox' Depot might be set up on the Greenough Flats. It would have proper officers and nurses to attend to the sick. All persons attacked who could not otherwise get proper attention were to be sent there immediately. Bompas, who set off to see Maughan at 9 p.m. on 7 April, said he was suffering from 'unmistakeable [*sic*] Small pox', and, instructed by the Resident, hired a man to look after Maughan and to prevent any contact with the hut where he lived. By 13 April Maughan was rapidly recovering, no further cases among Europeans had been reported, 'and the Natives are better', according to Watson's weekly report. Medical officers performed more vaccinations.³² Through the Government of Western Australia, Curr heard of Natingero people, 'two hundred miles north-east of Newcastle', whose country was occupied in 1869. A few of them were also pitted with smallpox, which was called *bilabunin*.³³

When he was the Colonial Secretary of Western Australia from 1880 to 1883, Lord Gifford³⁴ informed Curr about the Mulyara tribe on the upper Sanford River near Mt Wittenoom, several hundred kilometres north-east of Dongara. Their land was eighty kilometres or more square, and was between Mount Luke, Mount Parr,

Cheangwa and Warra-warra. Europeans arrived in 1874, and found a tribe of about 250 people. Curr reported:

The Mulyara tribe, in about 1864, was visited by a terrible outbreak of small-pox, which they call *noongoola*. So many died that the living were unable to bury the dead . . . Many of those who survived this disease are still alive, covered with pock-marks, and some of them blind.³⁵

In 1878 and 1879 Curr corresponded with Perks, whose sentence had expired. Curr's difficulties with his informant's handwriting contributed to confusion about when Perks had seen Aboriginals with smallpox. Perks was now a telegraph operator at Cheangwa Station, and manager at Murgoo on the lower Sanford, over 300 kilometres north-east of Geraldton, and over 200 kilometres from the west coast (Cheangwa Hill, 27° latitude, 116° longitude). He had seen active smallpox there.

many natives died of it, chiefly matured and old men. It was pitiful to see them dying unaided, and depending only on their own superstitious charms for recovery. They did not seem to fear contagion, although in some instances the diseased were left to die alone, and remained unburied. I have been among the same natives ever since, and I think I may safely aver that no instance of the disease has occurred since that time. It appears to come like a plague or the cholera amongst them at long intervals . . . I cannot say that I ever saw any of the Whites affected, although we were unavoidably among them during the last-mentioned outbreak, the second I have seen.

Perks said two-thirds of the male adults at Cheangwa were pitted with smallpox, many of them 'one mass of seams and scars. I have never seen Whites so badly marked'; not so many of the females were scarred, and none of the children.³⁶ Perks had known the survivors ever since. Frank Wittenoom, who knew Perks, described bushman's holidays and shooting excursions undertaken by him in the 1870s, with Aboriginal companions only.³⁷

Commentary

As smallpox declined in East Pilbara late in 1866, it spread south-west, and appeared in the interior south of Hammersley. It then

spread slowly on the upper reaches of waterways that terminated on the west coast, where it was seen nearly two years later, in 1868/69. After infectious contacts between people farther north and farther east, smallpox appeared near the Geraldine Mine on the outskirts of Geraldton. Infection spread to stations in the district, where Perks saw Aboriginal smallpox for the first time, and at the end of 1868 it was on the Murchison and Greenough rivers. The Geraldton Resident reported the outbreaks in January 1869, and smallpox was still active near Geraldton in May 1869, according to the 1870 *Almanack*.

As Watson found, it was spreading in clans east of the Darling Range at the same time, and by March 1869 smallpox was raging less than a hundred kilometres from the Irwin. It did not spread much farther south, but it broke out farther east in Sanford clans at about that time. On the Upper Sanford near Mt Wittenoom it struck the Mulyara tribe some years before settlers arrived in 1874, when new settlers observed pockmarked survivors. Perks saw Aboriginal smallpox for the second time at Cheangwa on the Lower Sanford in 1869 or 1870, when more cases occurred among Aboriginals on stations.

The incidence of smallpox was high in affected families, and was especially high in relatively dense river populations, as Watson's rough and hasty reports of it on the Irwin show. Between February and April 1869 the policeman with his packhorses visited or sent supplies to at least a dozen camps and several stations, where most Aboriginal people were sick. On just one of Foster's cattle stations, nearly half of 35 Aboriginal workers were sick. Many cases had occurred in 500 or more people in the groups he saw. His Aboriginal helper said a great number were sick, and that those who got better departed. In Watson's district, victims were dying daily, 'a great number' had died, and 17 dead people were seen at one camp. Accounts of unburied dead followed when indigenous communities were badly affected, and the remainder distressed and disoriented. Many deaths were recorded by people who saw the sick and knew pockmarked survivors. The mortalities in outbreaks Perks saw were similar and severe. His comment that more men survived than women is consistent with modern records of higher smallpox mortalities in women than men.

The impact of smallpox in indigenous societies was exceptionally severe because it was a new and demoralizing disaster in unsettled and settled districts in the west. Measles in 1860 was almost

certainly the first virgin soil epidemic to have occurred in living memory, but smallpox spread over a larger area was much more severe, and the after-effects on those who survived were frequently blindness, pockmarks and bacterial infections. Relatively dense populations on the coast and permanent inland waterways, where the incidence was highest, suffered the greatest damage.

Europeans on stations and in settled districts who nursed the sick and provided food saved some victims. Watson's supplies of tea, flour and sugar, meagre as they were, meant that some people who were too sick to get food for themselves stayed alive. Among Aborigines who did not suffer smallpox during these outbreaks were those who had been compelled to see Elliott at Geraldton, and were vaccinated when news of the Roebourne outbreaks reached the Resident. But judging from Watson's account of the vaccination of his children, vaccine was not always effective, perhaps because of the warm climate. Except for Maughan, the one adult victim from a station outside Dongara, the white incidence was again limited to a few of the settlers' unvaccinated children, who had mild attacks. Most Europeans at Cheangwa, Geraldton and Dongara owed their immunity to infant vaccination.

Smallpox disappeared somewhere south of Dongara and died out before it reached the south-west corner of the continent, where there are no records of it. Nor did settlers in remote southern communities at Eucla and Eyre's Sand Patch see it.³⁸ Perks, who had seen pockmarked Aborigines in northern districts and near Geraldton, said it was rare to meet any Aboriginal pitted with smallpox in the southern districts. Its impact was unquestionably greater in newly settled northern districts than in the old settled south.

Aboriginal smallpox in West Pilbara 1869–70

Neither E. T. Hooley, who established an overland route from older west coast settlements to Roebourne,³⁹ nor Trevarton Sholl, who explored 1600 kilometres of pastoral country west of Roebourne, saw smallpox among Aboriginal people they encountered in West Pilbara in 1866, and it was absent from adjacent coastal districts for three more years. However, later reports show it occurred in

West Pilbara about 1869. In the early 1880s, when Lord Gifford was Colonial Secretary, Curr heard of a tribe whose territory extended from North West Cape to about fifty kilometres south of the Gascoyne River and inland. It included Carnarvon. They were known as the Kakarakala tribe, and they were said to number 2000 people when Europeans arrived in 1877. According to Lord Gifford, 'Many of the tribe are strongly marked with small-pox, which they call *moonmangno*, and say reached them from the eastward'. In 1930 Radcliffe-Brown said the area included four tribes, with a total population of 2500–3000, and that Curr's estimate of 2000 was made after a very heavy mortality from small-pox.⁴⁰ Its recent ravages on the Gascoyne River were unmistakable.

In 1885 Protector C. Gale was at Carnarvon, where the Gascoyne enters the sea on the west coast, and said there was 'a clear history of small-pox among the natives . . . many of the older men showed definite pock marks'. When he asked them how long it was since the outbreak, 'he was shown a lad, about 16 years old, and was told that it occurred when that boy was a baby', an estimate that Cumpston accepted. Settlers did not see pockmarks in an isolated tribe at Shark's Bay in 1874.⁴¹

An outbreak early in 1870 among Kanakas on the ship *Kate Kearney* was widely reported. They had caught smallpox at Exmouth Gulf and some had died. The *Kate Kearney* was a pearling craft, which had sailed from Sydney to the north-west with Kanaka fishermen. On 16 February the outbreak was reported in the *Inquirer*. When the ship returned to Sydney, her crew spread news of the outbreak among the pearl-fishers and described the destructiveness of the disease. In June 1870 the news was circulated by the press in Melbourne and Adelaide as well as Perth. Cumpston investigated it much later. The *Kate Kearney* arrived at Sydney from Nickol Bay on 30 April 1870 and was quarantined for smallpox. There were 26 cases of which 6 were fatal; and 16 cases were landed at the Quarantine Station. Cumpston found no record of the pearler's movements, and said smallpox was then an epidemic in the north-western districts.⁴²

The last outbreak in the Pilbara was west of Roebourne. On 1 January 1870 Sholl said smallpox was present again.

The small pox having appeared among the natives to the Westward and fear being entertained that it will spread among the white population I have been requested to apply for a small supply of vaccine matter. If favourable in Perth I shall be glad to get some of the virus in glasses as being less likely to be affected by climate.

At the end of January 1870 Sholl heard smallpox was prevalent on the Maitland River, fifty-six kilometres west of Roebourne. Two had died and another was sick, and he feared that before long the disease would travel in the direction of Roebourne. It had steadily advanced from Exmouth Gulf, and was carried from place to place by travellers. He was apprehensive that it would break out among the Aboriginal pearl-shellers, who came from all parts of the coast. In that case, there would be great loss of life, and the stoppage of the fishing. Sholl recalled the 1866 outbreak, and said it might be advisable for Roebourne to be declared an infected port. He was authorized to act on shipping, but 'the chief danger is from overland communication by natives, for which the ordinance makes no provision'. He wanted instructions from the Colonial Secretary.⁴³

On 16 February 1870 the *Inquirer and Commercial News* ran the story in Perth, where the press had long-standing connections with the Sholls. Smallpox had broken out among the Aboriginals on the Fortesque, 'where they were dying off', and a case had been reported on the Maitland. There was no vaccine lymph, many unvaccinated children, and some alarm among the settlers. On 16 March there was more news of smallpox. By then, smallpox had disappeared at the Fortesque, but not before attacking Mr Hooley's children in a mild form. When he had an opportunity, Mr Hooley bled the Aboriginals when they first became ill, and claimed that those who were thus treated recovered. Others died. At the Maitland the disease was still prevalent, and several Aboriginals, some of whom Sholl said were useful men, had died. Roebourne was free of it for the time being, but Sholl feared it would travel there sooner or later.⁴⁴ The 1871 *Western Australian Almanack* chronicled the attacks suffered by Hooley's family.

The Resident was more confident when the disease had still not appeared at Roebourne early in April 1870. He had visited the Maitland and the Fortesque rivers to find out to what extent the dis-

ease had affected Aborigines. On the Fortesque it had quite disappeared, but not before many had died. On the Maitland some 7 or 8 natives had died. Sholl saw several in different stages of the disease, 1 of whom had since died. He thought Mr Venn on the Maitland was most attentive to them, giving food and medicine, and he left medicine for the sick with Venn and Hooley. When Venn was in town later, he said there were no fresh cases, that one of the native shepherds had died and others were convalescing. He hoped the disease had disappeared from his district. A half-caste lad who was attacked recovered. According to Sholl, 'children and young men and women almost invariably recover'. The disease was apparently no longer active, but more vaccine lymph was needed in order that Aborigines might be vaccinated and protected from future attacks.⁴⁵

Commentary

Smallpox spread to the Gascoyne and Ashburton and tributaries. It travelled west towards Carnarvon, north to Exmouth Gulf and the North West Cape, and an epidemic hit West Pilbara in 1869 and 1870. Kanakas on the *Kate Kearney* were infected during contacts with local people. Refugees fleeing from infected districts were blocked by the inaccessible Hammersley Range, and smallpox spread to hitherto unexposed people on the Fortesque and Maitland west of Roebourne. It was active there for at least four months, from January to April 1870. Its ravages in West Pilbara ended in 1870. The impact of smallpox in West Pilbara was comparable with the damage Sholl had seen farther east in 1866. As we might expect, young people often recovered, and in newly settled districts the severity of smallpox was mitigated by settler support. Hooley and Venn were among those who supplied food and care, which saved some sick victims, although many died. Without modern data about smallpox mortalities in naive populations, Radcliffe-Brown underestimated mortalities in tribal populations near the North West Cape. The mortality in Kanakas at Exmouth Gulf in 1870 was less than it was among Aborigines, only 6 of 26 Kanaka cases being fatal. The economic value of Kanaka divers ensured they were fed. The incidence of smallpox among Europeans in West Pilbara was lower than it had been farther east in 1866. Sholl had obtained

vaccine lymph, and vaccinations would have been performed on most unvaccinated children, although Hooley's children had mild attacks after contacts with infectious blacks on the Fortesque.

Brief records of smallpox among Aboriginal people in Western Australia in the 1860s are similar to more plentiful records of virgin soil outbreaks of smallpox among Native Americans in British North America in the seventeenth and eighteenth centuries, and in the United States in the nineteenth century. Both populations were wholly susceptible. Like Aboriginal Australians, Native Americans spread it by terror-stricken flight when it appeared among them. Both attributed the disease to supernatural causes. Native Americans thought cruel demons caused the sickness, and Aboriginal people used charms and incantations to protect themselves. Indigenous 'treatments', which included plunging into cold water, may have made them worse. Epidemics swept across the Rocky Mountains, and most of the continent, on several occasions. Smallpox swept through Western Australia from north to south in the 1860s.

Vaccination of Native Americans began in the first decade of the nineteenth century, and was promoted by Congress in 1832, but it was too late to prevent disastrous mortalities in the first half of the nineteenth century. After the 1850s decidedly smaller indigenous populations were better vaccinated and were less affected by smallpox epidemics than other American populations.⁴⁶ Vaccination of Aboriginal people in the south-west of Western Australia had begun by the 1850s, and was extended to the Pilbara when smallpox appeared there in 1866. It was performed by colonial surgeons and dedicated people such as Emma Mary Withnell. As Sturkey relates, their services were appreciated; but as Sholl realized, distance and climate adversely affected the supply, storage and distribution of vaccine.

Conclusion

Smallpox was the most important cause of demographic changes in Aboriginal societies in Western Australia between 1860 and 1870. Curr's information from the Government of Western Australia was a reliable but incomplete record of where smallpox had occurred and its impact. Radcliffe-Brown used Curr's work, but referred to only two of many outbreaks in the west. He did not use Cumpston's

more recent work, or other substantial evidence available in Western Australia. His estimates of mortalities fall short of modern knowledge of its impact in naive populations.

Topography and terrain affected the spread of smallpox in Western Australia in unexpected ways, and dated observations imply that the last outbreak was in West Pilbara. Much of the Western Australian evidence is from observers who were there during outbreaks, it is thus more detailed than evidence of outbreaks in eastern Australia in the 1860s. It is later and more carefully preserved than evidence of earlier outbreaks elsewhere in Australia, and covers a bigger area. Western Australian evidence is thus important for an overall interpretation of the introduction, spread and impact of Aboriginal smallpox. Its undeniable impact in northern and Western Australia casts doubt on Diamond's finding that Aboriginal societies in land unsuited to European food production in the north and the west remained more or less intact.⁴⁷

The Aftermath

Several years before smallpox attacked Aboriginal people, their resistance to European incursions in the north-west of Western Australia shocked settlers and provoked reprisals by them; for instance, on the Boolu Boolu river at La Grange Bay in 1864, and a few years later, when tribal people killed a party of Aboriginals and Europeans near Flying Foam Passage. In the aftermath of smallpox in the early 1870s, when indigenous numbers and morale were low, pastoralists occupied land on the Murchison and Upper Sanford rivers, apparently without difficulty. But a decade later, Aboriginal hostility forced some new settlers to sell out, and in the early 1880s, Aboriginal resistance also alarmed settlers in the Kimberley. Smallpox outbreaks were a major cause of the fluctuating strength of Aboriginal resistance, and the way was left open for settlers.⁴⁸

The invasion of the Pilbara by pastoralists and pearlers in the 1860s did not include convicts or ticket-of-leave men, so Aboriginal labour was crucial in both industries. In Sholl's opinion, the shell-seeker owed his wealth to the intelligence of the local people, who had not yet realized the value of their services. When smallpox proved fatal to Aboriginals in 1866 and again in 1870, settlers

lost the scarce labour they needed, and from 1872, they imported 'Malays', as they described Timorese divers from Kupang.⁴⁹ Non-Aboriginal labour became important: by 1901, there were as many European and Asian workers as Aboriginal workers in the pastoral north-west, and non-Aboriginal labour was also essential for the pearling industry.⁵⁰

Before the twentieth century, the European population of Western Australia was too small to maintain childhood diseases, but measles, introduced in 1860, was re-introduced twenty years later, when settlers and indigenous people alike described it as disastrous.⁵¹ Tuberculosis attacked the Wajuk in the south-west when their land was occupied in 1834. It was the first European disease noticed among the local population, and it was a constant menace. In 1879 an early settler said 'consumption' was still common in the Wajuk, and that their numbers had decreased from 80 to 40 people in the previous fifty years. Tuberculosis would have spread in the south-west in indigenous communities in the nineteenth century, and was spread by settlers in the north-west in the 1860s. Tuberculosis and measles prevented any demographic recovery from smallpox in small Aboriginal populations.

10

The Diseases that Killed

As Cleland realized, indigenous Australians shared the ancient disease patterns of South-East Asia. They suffered from, and still suffer from, trachoma, yaws, endemic syphilis and hepatitis B, all chronic diseases, which, notwithstanding the discomforts and disadvantages they cause, are not lethal. Like Native Americans, who were isolated until the sixteenth and seventeenth centuries, the Australians were isolated from Eurasia for millennia before voyagers from the outside world began sustained contact with them in the late eighteenth century. The impact of Old World diseases on indigenous populations on the two continents was comparable, and their susceptibility to the invisible invaders comes as no surprise.

However, in 1994 Francis Black surveyed evidence of high death rates among New World peoples in contact with Old World diseases, and expanded the sequence of factors that caused their biological catastrophes. New World populations have a different and much more restricted suite of major histocompatibility antigens than Old World populations. The major histocompatibility antigens play an essential role in generating immune responses, and the greater heterogeneity of Old World populations is believed to be due to negative selection by the wide range of infectious diseases to which they, but not New World populations, have been exposed. This genetic susceptibility was compounded by the social disruption caused by outbreaks of 'new' diseases such as smallpox and tuberculosis.¹

There were significant differences in the history of Old World diseases on the American and Australian continents. The voyage across the Atlantic to North America was short enough for travellers infected by fellow passengers to arrive in America apparently well, then sicken and become infectious after landing. By comparison, although travellers with chronic diseases (in particular tuberculosis) arrived in Australia in 1788, acute infectious diseases, such as whooping cough and measles, died out on the longer sea voyage. The newcomers established a quarantine service at Sydney by 1828, and subsequently quarantine was regularly practised in Australian ports. Acute infections were occasionally missed by quarantine officials, but were not maintained for long in the small colonial populations.

Measles was not endemic in populations under 200 000, and was not a common problem among Europeans at Sydney or other colonial capitals before the 1860s, when the immigrant Australian population reached about 1 000 000. Close contacts between Aborigines and settlers that might result in transmission of acute infectious diseases were rare, although tuberculosis was more easily transmitted.

One interesting difference between disease patterns in Native Americans and native Australians after European contact is that Australians were exposed to diseases by visitors from Asia and immigrants from Europe almost simultaneously. Infection with tuberculosis was introduced into Australia by British colonists from the time the First Fleet arrived. Later newcomers introduced measles and venereal infections, but they did not introduce the most deadly of the Euro-Asian diseases: smallpox. By coincidence, this was introduced from the north by Macassan trepangers, at the same time as British settlement on the east coast, late in the eighteenth century.

Smallpox had invaded the Indonesian archipelago in the late eighteenth century, when severe outbreaks occurred in Sumatra in 1780–83, in the eastern archipelago about the same time, in the Palau group in 1783, and probably also in South Sulawesi. When it broke out near the busy port of Macassar, it infected some of the islanders who made short voyages to Australia, and sometimes persisted in the fleet for a whole trepang season. This may have happened when Pobassoo made his first voyages to Australia in

the early 1780s. Smallpox occurred again in fishing-fleets bound for Australia in the 1820s, the 1860s and 1870s, and appeared in New Guinea in the 1890s. Outbreaks in south-east Asian mainland countries and islands continued into the twentieth century.

No signs of pockmarks or smallpox were seen among any of the native peoples of Australia or the Pacific Islands during Cook's voyage in 1770. We know it did not occur among members of the First Fleet during the voyage or after their arrival in Australia. It is interesting to find that as a precaution against the possible occurrence of smallpox during the voyage to Australia, or after arrival, the surgeons of the First Fleet had been provided with variolous matter. Arriving in January 1788 there was no need for it until April 1789, when smallpox broke out among local Aboriginals. None of the Europeans caught it, either because few of them had close contact with sick Aboriginals, or because they were already immune. When it occurred on later occasions, it was largely confined to Aboriginal people, because most Europeans had been vaccinated before they emigrated. Except for unvaccinated children, and inadequately vaccinated adults, who caught it from sick Aboriginals, smallpox did not occur among Europeans in eastern Australia before 1857, or in Western Australia before 1893. However, as Cumpston relates, it was introduced in port cities on twenty-two occasions, and 146 vessels were quarantined because of smallpox between 1828 and 1914.

Smallpox spread by face-to-face contacts in the course of daily activities. Aboriginal people also travelled long distances for ceremonial occasions. The incubation period could end during such ceremonies, and visitors become acutely ill and highly infectious. Some tribal people might have taken the disease with them when they fled from infected districts to more distant relatives or friends. Cleland's account of introductions of smallpox, and how it spread through Aboriginal bands across Australia was first published in 1912. Half a century later, after investigating how ecology influenced disease patterns in Aboriginal societies, he reaffirmed his early opinion, and said that smallpox was introduced on the north coast several times, probably by 'Malays', and swept right across the continent to Sydney in 1789 and the lower Murray before settlement.² We know from Mair's report that it did indeed spread from the interior to the east coast in 1831.

Almost certainly, smallpox was occasionally introduced along the north coast without spreading beyond one or two families. Later introductions, which were recorded, spread a considerable distance. Between 1780 and 1790, it penetrated eastern Australia, reaching Port Jackson in 1789, Port Phillip soon after, and perhaps other districts before it died out in the early 1790s. In the next fifty years, the presence of older pockmarked people in New South Wales, South Australia, Victoria, and even Queensland, revealed the paths smallpox had followed in the 1780s, before the British arrived.

It swept through eastern Australia again between 1824 and 1832. Observers on the Cobourg Peninsula in the 1820s and 1830s recognized pockmarks and left records, as others did when they saw it in far north-western New South Wales early in 1829, in eastern New South Wales in 1830 and 1831, and on the east coast at the end of 1831. When it was introduced a third time, in the early 1860s, it spread in north-eastern and northern Australia, through the Centre and south to the South Australian Bight in 1867. Between 1860 and 1870, another introduction was followed by its spread past the Kimberley to the Pilbara, the north-west and down the west coast to the Dongara district.

Thus in the ninety years between about 1780 and 1870, smallpox was active somewhere on the continent, for various periods, totalling some thirty years; and it attacked people in every mainland colony. Previously unexposed populations were in the path of infection when it spread out with travellers from South Sulawesi and invaded northern Australia. The outbreaks of smallpox in the 1780s were probably the first to occur in this country. It probably took eight years for it to travel from northern to southern Australia before it arrived on the east coast in 1789, and probably eight years for it to cross the continent to the east in 1830 and 1831. It reached the Bight more quickly in 1867, but took just as long to spread past the Kimberley and down the west coast in 1870.

This interpretation of the introduction and spread of smallpox in Aboriginal Australia on three or four occasions is in direct contrast to explanations of the same events put forward for the first time by Curr a century ago, by economic historian Noel Butlin in 1983, and again in 1997 by American physiologist Jared Diamond, who claimed that epidemics of diseases introduced by Europeans

within a year of their arrival at Sydney in 1788 were a principal cause of Aboriginal deaths. Diamond's killers include chickenpox, and others not found in the records, as well as smallpox and tuberculosis.³

Curr's extensive publications in the 1880s lent authority to his mistaken opinion that smallpox was introduced at Sydney in 1788, and was endemic among Aboriginals until 1845. Like Curr, Butlin claimed that smallpox in Aboriginals followed an introduction at Sydney, and attributed the outbreak to the surgeons' supply of bottles of virus material. The one possibility Butlin did not consider was that the bottles were never opened. His opinions differed from Curr's, in that he proposed that a second introduction in 1829 occurred through blankets being sent ashore from a ship at Sydney. In 1991 he again considered the possibility that Indonesian fishermen introduced smallpox, but he came to the same conclusions that he had reached in 1983, saying: 'The two early outbreaks in 1789 and 1829 are timed with known incidents connecting colonists [in Sydney] with the virus.'⁴

Butlin underestimated the connection Macknight had made between Macassan visitors to northern Australia and introductions of smallpox. Nor did he recognize that smallpox spread through indigenous communication networks. Continued speculation about introductions of smallpox on the east coast now seems unnecessary, although Diamond still regards Sydney as the place where smallpox, as well as other diseases, entered the country in 1788. Smallpox was the only epidemic recorded in 1789, soon to be followed by tuberculosis, but there are relatively few records of other epidemics until the European population increased later in the nineteenth century. Smallpox was occasionally mistaken for chickenpox in the records, but chickenpox itself was not a recorded killer. It has recently been recognized as an ancient disease of hunter-gatherers, and was maintained in groups of less than 100 people by latency and reinfection after a first attack.⁵ Unlike measles, chickenpox does not cause severe symptoms in modern hunter-gatherers.

We know that the eradication of smallpox was achieved only after intensive research into factors that affected its spread, including the effects of temperature and humidity on variolous matter. Butlin did not cite the WHO publication by Fenner and others in 1988, and evidently was unaware that smallpox virus, present in the

surgeons' variolous matter, was unlikely to be active after exposure to temperatures and humidities recorded at Sydney in 1788 and 1789. Bultin's reconstruction of events that followed the arrival of the *Bussorah Merchant* in 1828 is flawed for similar reasons.

Like Curr, Butlin also dismissed the possibility that smallpox was introduced in the Gulf in the 1860s. He said that if it had been introduced by Macassans, it was extraordinary that the strongest pockmarking was not reported near the Gulf. Curr's anonymous informant at Cowan Downs had seen only lightly pitted Aborigines, evidence that Butlin said would not support a strong case for a Macassan introduction in Queensland.⁶ Nevertheless, research and photographic evidence, published by WHO in 1988 establish that although numbers of victims acquired severe and permanent pockmarks, on some faces they faded in a decade.⁷ Historical evidence of smallpox on the north coast in the 1860s is consistent with this published evidence. Cloncurry Aborigines on Cowan Downs would have acquired pockmarks in the early 1860s, which had faded by the time Curr's correspondent described them in 1879. However, at Darwin in 1881, Foelsche knew Mangminone, alias Mr Knight, who had acquired severe and permanent pockmarks about 1861, when he was five years old.

Everything we know about smallpox in the Sydney district, where active disease was seen in 1789, suggests that early outbreaks were virgin soil outbreaks. There is no other explanation for plentiful evidence of newly dead seen in all the coves and inlets of the harbour, on the path to Broken Bay, and at Botany Bay, and for working-parties needed to bury them. Fear of the appalling new disease and shock caused those still well enough to flee, and so spread infection. Abandoned victims, prostrated by smallpox, starved or died of thirst. The Sydney outbreaks, with high incidence, and very high mortalities recorded by people who knew smallpox well, were like outbreaks that occurred in previously unexposed Native American populations, where there were no immune survivors of previous outbreaks to aid victims. With no such support in unsettled Australia, the first outbreaks were among the most damaging that occurred.

When smallpox returned to eastern Australia in 1824–32, virgin soil outbreaks occurred again in rarely exposed populations. Sturt

and his party were the only observers in the unsettled south-east who saw them in 1829; then in 1830–31 smallpox appeared in newly settled districts in eastern New South Wales. A few older Aboriginals were immune because of previous attacks, but many more previously unexposed people were infected. Settlers provided food, which saved some from starvation, and army surgeons vaccinated others and distributed vaccine and lancets. A few old colonists who knew smallpox variolated some groups. In 1838 Sturt would recall the extensive damage caused by smallpox that he had seen in the south-east.

In the 1860s smallpox struck rarely exposed people in eastern Australia for the third time. In Queensland, where it had never occurred among Europeans, not many of some 30 000 new settlers would have recognized it, even if they had seen it. There are no records of active smallpox, and very few newcomers in the small population of the large new colony knew of its recent presence in central and north Queensland. Exceptions were a small number that included telegraph officer ‘W’, the Cowan Downs informant, Palmer, Hodgkinson and a middle-aged Wonduri. Knowledge of smallpox was fading fast, and it is likely that its incidence and impact on Queensland Aboriginals in the 1860s will never be known. It did not spread into the settled south-east on that occasion.

In the Top End, reliable informants, such as Foelsche, Robinson, Wildey, the German traveller Eylmann in the 1880s, and more recently Allan Young, spoke of violent outbreaks in the 1860s. In the Centre, ‘W’, Lutherans on the Finke River, Gillen, Eylmann, explorers Giles and Tietkins and Mounted Constable Samuel Gason unexpectedly saw pockmarked people who had recovered in the 1860s. In western South Australia, eye-witnesses saw active Aboriginal smallpox in 1867, and victims said it had come from the north. The South Australian Government provided vaccine so that Aboriginals at Streaky Bay and Fowler’s Bay could be vaccinated.

In Western Australia, pockmarked survivors in the Kimberley had smallpox in the 1860s. There is no evidence of earlier outbreaks or of elderly pockmarked survivors in the colony. Evidence of active smallpox in the Pilbara, the north-west and on the west coast, followed by evidence of unburied dead, abandoned camps and starving people, suggests outbreaks in the 1860s were the first to occur in

Western Australia, and that mortalities were severe. Aboriginals in coastal districts benefited marginally from settler support on local stations, and from supplies distributed by police. Members of the medical profession were well prepared. Some Aboriginals were vaccinated by medical officers, or by settlers such as the Withnells.

Generations of Aboriginals were devastated by this new disease that killed a high proportion of those who were infected. Victims often transmitted infection to family members, and to neighbouring families they traded with. Exchanges of valued and scarce commodities, such as pituri, facilitated the spread of infection over amazingly long distances, as did obligations of kinship and friendship. Yet whole Aboriginal populations were never exposed, because opportunities for infection to spread depended on unpredictable social behaviour, and the chances of close contact between susceptible and infectious people. It did not spread between hostile clans, so it was never uniformly distributed, and was always patchy.

The impact of smallpox varied more predictably with the seasons, as happened in northern Australia. It probably invaded Aboriginal communities away from the coast for the first time after the departure of fishermen in the early 1780s, when people who had gathered together in the wet season dispersed in the dry season after the monsoon. Infection would have spread readily when fearful people travelled to get away from the new disease. The incidence of smallpox would have been high among Aboriginals in the Top End, and in coastal clans who had close contact with infectious visitors, and with each other. Mortalities would have been high in prostrated, dehydrated and starving victims at a time of social and economic crisis. Children and young adults often survived, but in previously unexposed populations, where most people caught smallpox, mortalities of 60 per cent would have been likely. When smallpox was introduced again in 1824 and 1861, it behaved in the same way. On those occasions, a few old Aboriginal people were immune, namely those who had recovered from infection forty years earlier. But smallpox may have killed over half the population of monsoonal northern Australia between 1780 and 1870.

However, the impact of smallpox varied considerably with the size and density of populations it invaded, and Australian populations in different parts of the continent fared differently when

smallpox broke out. Each time it was introduced, smallpox spread from centres of infection on the coast along rivers and inland waterways. When it penetrated sparse and scattered populations in the arid zone, only one or two cases at a time occurred, and it would often have died out, or it might have spread slowly in the interior for several years on each occasion. Its incidence in those small bands would have been low, unless they joined others for ceremonies or to gather harvests after rain. Mortalities in small scattered families in the harsh environment would have been high; but probably fewer than 25 per cent of inland populations in north-eastern and northern Australia died of smallpox between 1780 and 1870.

Chains of infection from the north in the 1780s and the 1820s extended south to the northern reaches of the Darling River, and smallpox then spread readily in relatively dense riverland populations on the Murray and tributaries, causing high mortalities on both occasions. During the 1820s epidemic, the presence of some old immune survivors, vaccination of some Aboriginals, and food and milk provided by some settlers saved some lives. Nevertheless, it is not unreasonable to think smallpox had probably killed half the population of the Murray–Darling Basin by 1840.

South of the Murray–Darling Basin, settlers in South Australia and Victoria identified older people who had recovered from smallpox in the 1780s, as well as survivors of the 1829–32 epidemic. Its incidence would have been high in denser populations in the 1780s, and mortalities might have reached 60 per cent during the first onslaughts of the new disease. In 1830 smallpox spread to riverside and coastal populations in southern Australia for the second time before settlement in South Australia and the Port Phillip District. On that occasion, smallpox attacked denser populations in well-endowed districts on the lower Murray, the coast and inland that were already depleted in much the same way that it would have in the 1780s. Early in the twentieth century, a South Australian victim recalled that some clans were exterminated. Others in Victoria suffered severely on both occasions. As we would expect, smallpox spread readily in ‘semi-settled’ localities in the Western District, and Robinson’s reference to a Hopkins River clan who died of disease suggests that extinction was a real possibility there also. By 1877 only a few old pockmarked people were seen in the semi-arid fringes

of the Western District. These clans in South Australia and Victoria suffered long-lasting depopulation, and dispossession prevented their recovery.⁸ In the absence of other lethal diseases among Aboriginals at that time, smallpox was its cause. Although there is no good evidence of its occurrence there, the absence of smallpox in Gippsland seems unlikely. Thus its impact in denser populations in southern Australia in 1830 would have been at least as severe as it was in eastern New South Wales, and it had probably killed over half the population of southern Australia by 1840.

East of the Great Dividing Range, smallpox spread to the coastal strip in the late 1780s, and broke out among Byelée people near the future site of Rockhampton. It probably occurred in most coastal populations in eastern Australia, and would have severely damaged moderately dense populations with riverside and marine resources. Clans on the coast were vulnerable to infection by travellers from west, north or south. By 1789 smallpox was ravaging Aboriginals in the new colony, where it was seen around Sydney Harbour, and on the east coast between Port Hacking and Broken Bay. According to observers who knew smallpox, incidence and mortalities were high. Its violence shocked colonists, and when they heard about it, European visitors were surprised. Bennelong's estimate, that half of those in that part of the country had died, is a credible estimate of its impact in the one epidemic seen at Sydney. However, only 3 of the Cadigal clan survived; 4 of 6 victims treated at the settlement died; and there are many reports of dead victims. Although some local Aboriginals probably escaped it, mortalities among those who did catch smallpox were at least 60 per cent, and were among the worst in eastern Australia between 1780 and 1870. Accounts of elderly survivors at Port Macquarie and Dungog suggest severe mortalities were not confined to Sydney in 1789.

Smallpox attacked Aboriginals on the coastal strip again in 1830. Settlers later heard about severe outbreaks, and described many pockmarked people in districts from Rockhampton to Brisbane. In 1831 active smallpox was seen at Trial Bay and Port Macquarie, and experienced observers Smyth and Mair reported die-offs. With an interval of about forty years between outbreaks, high mortalities probably occurred twice in relatively dense populations in coastal districts with good resources. In the 1860s small-

pox invaded populations on the Burdekin and Dawson rivers, and elsewhere in north Queensland for the third time. It is reasonable to assume that it had killed half the population on the east coast by 1840, and more in Queensland in the 1860s.

In Western Australia, smallpox might have occurred more than once before settlers saw its traces in the Kimberley, but it occurred only once elsewhere in the colony. In the 1860s, virgin soil outbreaks ravaged Aboriginal communities in coastal and riverside districts from Roebourne to Dongara. After outbreaks in newly settled districts, settler McKenzie Grant said half the Aboriginal population had been destroyed. His opinion resembles Bennelong's estimate of early losses on the other side of the continent, and is consistent with overall losses of 60 per cent in virgin soil outbreaks in denser populations in eastern Australia. However, losses in scattered bands in the harsh interior, where sparse populations converged on intermittent waterways and desert wells, were less significant, and smallpox probably died out in western deserts. It never reached the far south-west, or the coast along the Bight.

When smallpox spread from the north coast to the south coast between 1780 and 1870, it inflicted the worst damage in denser populations in eastern Australia, and the least damage in sparser populations in Western Australia. In northern Australia, coastal clans, such as the Unalla, cannot be identified today, after suffering severely on three occasions. At those times smallpox seems to have persisted for several years without inflicting major damage in the interior of northern and north-eastern Australia or northern South Australia. Populations in adjacent western South Australia suffered at least once. In the Murray–Darling Basin, relatively dense populations suffered extensively twice before 1840, as did those in eastern South Australia and Victoria. Those in eastern Queensland suffered three times, those in eastern New South Wales twice.

Long-standing debates about the demographic impact of Aboriginal smallpox have continued for over a century. In 1886 Curr said smallpox introduced at Sydney in 1788, and again in Western Australia in the 1860s, killed one-half or one-third of the Aboriginal population south of the Tropic of Capricorn. His rough estimate has since influenced the more recent opinions of Tidswell, Radcliffe-Brown and Butlin, who assumed that the impact was greater in the

south than the north. However, information from several of Curr's informants, and further evidence available after his death, showed that smallpox had occurred on three separate occasions in northern Australia, in the Northern Territory and Queensland; and that in southern Australia, it had occurred at least once in western South Australia. The heavy mortalities of smallpox in the south-east, which Curr attributed to an introduction at Sydney, resulted from the relatively large size of the local populations.

The demographic impact of smallpox in Mexico five centuries ago, as analysed by Robert McCaa in 1995, was several times greater than its impact in Europe, and over all broad demographic destruction exceeded 50 per cent, ranging upwards beyond 75 per cent, even to 90 per cent.⁹ Like other evidence reviewed by Fenner, and mentioned earlier, records of smallpox in Mexico suggest that epidemics in previously unexposed Native American populations produced much higher mortalities than those in Asia, owing to disruption and starvation. Similarly, Australian records suggest that mortalities in denser populations, where tribal territories were small and many languages were spoken, were higher than those reported by Rao in hospital patients in Madras. But in scattered, sparse populations in the interior of Queensland, the Northern Territory, northern South Australia and Western Australia, over half the continent, the incidence of smallpox was lower and its impact was less severe, which limited overall demographic destruction.

Wherever smallpox occurred, populations suffered damaging losses of productive adults, warriors and tribal elders. In Australia the shock of the new disease, and the disruption of customary life, coincided with crises of dispossession. Smallpox reached Port Jackson after the British ships left, when their passengers' intention of staying was unmistakable.

When it reappeared forty years later, it coincided with the newcomers' incursions far into the north-west and south-west of the colony of New South Wales; and within a decade settlers were pasturing sheep on the tribal territory of populations that had twice suffered heavy losses from smallpox. Finally, after smallpox had depleted remote clans in the outback and the west, miners and pastoralists occupied the sparsely populated lands. Aboriginal people continued to associate smallpox with invaders, as they had in Went-

worth's day. Resistance was impossible during outbreaks, and for some years the loss of warriors and morale made life less hazardous for settlers. The time it took to recover from outbreaks was one reason why resistance was erratic. Victims blamed enemies, Aboriginal or European, and might have retaliated in tribal wars or clashes with settlers. But between 1780 and 1870 smallpox itself was the major single cause of Aboriginal deaths. The consequences of Aboriginal smallpox are an integral part of modern Australian history. It hindered resistance by Aboriginals, and opened the way for settlement by vaccinated Europeans, who were untouched by it.

From 1789 until the 1920s Europeans who were familiar with smallpox and pockmarks saw Aboriginal people who had suffered from it as children or young adults, with pockmarks severe enough in some cases to identify them as its victims for the rest of their lives. There are many written records of them in colonial literature. In nineteenth-century photographs of Aboriginal people there are blemishes on some faces that look little worse than acne. Nineteenth-century newspaper correspondents in Victoria called them 'native pock'. But in 1932, after examining cases of acne in Aboriginal people, Basedow said some had genuine and typical pockmarks. Of the diseases seen in Aboriginals before 1870, smallpox caused the most dramatic die-offs, and was the known cause of large accumulations of skeletal remains seen on the mainland in the nineteenth and twentieth centuries.

During that period, the treatment of Aboriginal people in newly settled districts, who were seen to be at risk of smallpox stands out in contrast to the troubled consciences of humanitarians and the somewhat ineffectual remedies for the problems of the day. A small but significant number of concerned and well-informed surgeons and settlers undertook the only appropriate course of action to prevent and control smallpox when they arranged the vaccination of indigenous people to confer immunity to smallpox, and so prevent it spreading to other Aboriginals or to European children.

From the beginning of European settlement, colonists reported other diseases they thought they had seen among Aboriginals. Many believed venereal diseases were prevalent among them, and (with reason) assumed that contact with Europeans was the cause. The records are confusing, because few observers distinguished between

gonorrhoea, which was not lethal, and venereal syphilis, which could be. Moreover, whites who recorded these diseases among Aboriginals in the eighteenth and nineteenth centuries knew nothing about related indigenous diseases, such as yaws and non-venereal endemic syphilis, which they may have mistaken for venereal syphilis.

The worst disease transmitted to Aboriginals by Europeans between 1789 and 1880 was tuberculosis. The 'white plague' was most severe in first contacts, and occurred at Sydney, Perth, Brisbane, Melbourne and Adelaide. By the time the ravages of smallpox had ended, tuberculosis was a major problem in most Aboriginal communities. The European population, over 1 000 000 in 1870, climbed to over 2 000 000 in 1880, and to over 3 000 000 in 1890. In this changing environment, where, after 1906, they had less contact with Indonesians, many Aboriginals were affected by European diseases. Tuberculosis, which can be maintained in small populations, thrived in large ones. Measles appeared more often and, after many contacts with Europeans, the pattern of infectious diseases among Aboriginals came to resemble that of the European population.

Modern health care now benefits the entire population. Tuberculosis is being successfully treated, and many other communicable diseases that have threatened Aboriginals can be prevented. Vaccination is available for diphtheria, whooping cough, measles, mumps, rubella, polio, influenza, pneumonia, hepatitis and tetanus. Yet infectious diseases still persist, periodically plague Aboriginal populations and contribute to low life-expectancy. The most promising method of attacking problems of Aboriginal health was officially recognized in 1998. Using approaches to health questions employed elsewhere in Australia and the Asia-Pacific region, the National Centre for Epidemiology and Population Health at the Australian National University launched an Applied Epidemiology (Indigenous Health) programme. In the year 2000 most of the first 8 graduates of the programme were Aboriginals. They are in a better position than most health workers to address the present urgent need for trained personnel in that field. Familiar with the needs of Aboriginal communities, they can anticipate the appropriate medical services required by their own people. Their importance in the health network of Australia, New Zealand, the Pacific and Asia is now being acknowledged.¹⁰

Glossary

- bilabunin*: WA west inland, 320 kilometres north-east of Newcastle. Curr, *The Australian Race*, vol. 1, p. 380.
- boola*: WA north-west coast, mouth of the De Grey River, referred to anything nasty or poisonous: Ngurla tribe. Curr, *The Australian Race*, vol. 1, pp. 287–92.
- boulol*: NSW inland, Namoi River, near Nandewar Range. J. Mair, Assistant Surgeon, 39th Regiment, ‘Observations’, NSWA 4/2130
- chin-chin*: Qld east coastal range, headwaters of the Burdekin River, about 160 kilometres inland from Townsville: Breeaba tribe. Curr, *The Australian Race*, vol. 2, p. 432.
- danna-danna*: smallpox; *gulgog-gulgog*, smallpox scars, Wiradjuri language, NSW inland, Bogan River: Awabakal tribe. Threlkeld, *An Australian Language*, Appendix, pp. 79, 80, 88. Donaldson, note 20, in Campbell, ‘Smallpox in Aboriginal Australia, The Early 1890s’, p. 341.
- deum*: Qld east coast 160 kilometres north-west of Brisbane, Boyne River: Toolooa tribe. Curr, *The Australian Race*, vol. 3, pp. 121–2.
- dunnerh-dunnerh*: NSW inland west of Great Divide, Narran-Barwon rivers: Euahlayi tribe. Parker, *The Euahlayi Tribe*, p. 39.
- gal-gal-la* (smallpox): NSW east coast, Port Jackson, 1789. Collins, *An Account of the English Colony in New South Wales*, p. 598.
- goobimwah*: NT Port Darwin. Foelsche, ‘Notes on the Aborigines of North Australia’, p. 8.
- mean warann*: Vic., Western District, Hopkins River, Port Fairy, Portland. Dawson, *Australian Aborigines*, pp. 60–1. The words mean ‘chopped root’, perhaps *myrnong*, which was common in Victoria. Smallpox was similarly associated with vegetable food eaten by its victims elsewhere. Some Bornean tribes were afraid to eat corn during a smallpox epidemic because of its

- resemblance to pustules, and Sea Dyaks on Borneo called smallpox 'Jungle Fruits'. Hopkins, *Princes and Peasants*, p. 115.
- meeha-meeha*: NT north coast, Cobourg Peninsula–Port Essington: Unalla tribe. Foelsche. 'Notes on the Aborigines of North Australia', p. 8.
- mingi*: SA south coast, Mount Remarkable, Spencer Gulf: Doora tribe. Curr, *The Australian Race*, vol. 2, p. 136
- monnangno*: WA North West Cape to 48 kilometres south of Gascoyne River and 48–80 kilometres inland: Kakarakala tribe. Curr, *The Australian Race*, vol. 1, pp. 302–3.
- monola mindye*: smallpox, the dust of the *mindye*; *lillipook mindye*, scars of smallpox, the scales of the *mindye*. Vic., inland north-west, Loddon and Avoca rivers. Braim, *A History of New South Wales*, vol. 1, p. 245.
- moo-nool-e-mindye*: smallpox, dust, *lillipp-e-mindye*, pox-marked, north-west Vic., Loddon River: Ja-jow-er-ong tribe. Parker, 'Dialect of the Ja-jow-er-ong race', in R. Brough Smyth, *The Aborigines of Victoria*, vol. 2, p. 163; Radcliffe-Brown, 'The Rainbow Serpent Myth in South-East Australia', pp. 344–6.
- moora-moora*: SA inland, Lake Eyre, Lake Hope: Dyari tribe. Gason, in *The Native Tribes of South Australia*, ed. J. D. Woods, p. 283. Gason was not to know that *moora-moora* referred not to the disease of smallpox, but to a mythical ancestor said to have caused it.
- mooyla errill-ya-rill-ya*: WA west inland, 220 kilometres from the coast, Irwin and Murchison rivers, Cheangwa country: one of the Minung tribes. Curr, *The Australian Race*, vol. 1, pp. 369–71.
- nguya*: pustules, or smallpox; *nguya palti*, smallpox song, used as a remedy SA south coast, Adelaide. Teichelmann and Schurmann, *Outlines of the Aboriginal Language of South Australia*, p. 34.
- noongoola*: WA west inland, upper Sanford River between Mount Luke, Mount Parr, Cheangwa and Warra-warra: Muliarra tribe. Curr, *The Australian Race*, vol. 1, pp. 375–8.
- oie-boie*: NT north coast, Cobourg Peninsula, Raffles Bay: Unalla tribe. Wilson, *Narrative of a Voyage Round the World*, p. 170.
- pania*: NT inland, upper and middle Finke River; Aranda tribe. Louis Schulze, 'The Aborigines of the Upper and Middle Finke River', p. 218.

- poc* (dead), *mittayon* (smallpox): NSW east coast, Lane Cove, 1790. *The Journal of Lt. Ralph Clark 1787–1792*, p. 109. Scarred survivors of that epidemic were later seen by settlers who recorded Aboriginal words for smallpox in New South Wales, Victoria, South Australia and Queensland. Words for smallpox were also recorded by settlers after subsequent outbreaks in eastern Australia, 1824–30, and again in the 1860s after further outbreaks in Queensland, the Northern Territory, South Australia and Western Australia.
- purrer-purrer*: NT Darwin area, Port Woolner district: Larrakiya tribe. Anonymous vocabulary 1869, Curr, *The Australian Race*, vol. 1, p. 231. Macknight points out that this is a version of the Macassarese word *puru-puru*, meaning pustules, which can include smallpox, *Archeology in Oceania*, vol. 21, no. 1, 1986, p. 71.
- thunna-thunna*: Wiradjuri language, NSW inland, Lachlan River, Bathurst. J. Mair, Assistant Surgeon, 39th Regiment, ‘Observations on the Eruptive Febrile Disease’, NSWA 4/2130.
- tingal*: Qld east coast, Bustard Bay, Rodd’s Bay, and back to Many Peak Range. Curr, *The Australian Race*, vol. 3, p. 126.
- walmbing*: WA Broome and Beagle Bay, a kind of chickenpox or smallpox. Bates, *The Native Tribes*, p. 295.
- wanboy*: Qld east coast near Rockhampton–Keppel Bay, Caliope River, Curtis Island: Byellee tribe. Curr, *The Australian Race*, vol. 3, p. 114.
- weeteen*: Qld mountain ranges some 320 kilometres west of Rockhampton on the Barcoo River and Ravensbourne Creek: Torraburri tribe. Curr, *The Australian Race*, vol. 3, pp. 78–9.

Notes

1 Aboriginal Australians and Old World Diseases

- ¹ Banks, *Endeavour Journal*, vol. 2, p. 126; Worgan, *Journal*, p. 19.
- ² McNeill, 'Migration Patterns and Infection', p. 28; Fenner, 'Sociocultural Change and Environmental Diseases', Stanley and Joske (eds), *Changing Disease Patterns*, pp. 8–26; Black, 'Modern Isolated Pre-Agricultural Populations', pp. 38–53.
- ³ Fenner, 'Sociocultural Changes and Infectious Disease', pp. 22–3, 28–9.
- ⁴ Moodie, *Aboriginal Health*, pp. 29, 161–2.
- ⁵ White, 'Pitfalls to Avoid', pp. 269–92; Dampier, *A Voyage to New Holland*, vol. 1, pp. 453, 457, vol. 2, pp. 440, 442; Cunningham, *Two Years*, vol. 1, pp. 184–5; Smyth, *The Aborigines*, vol. 1, p. 253; Bates, *The Native Tribes*, pp. 288–9.
- ⁶ McNeill, *Plagues and Peoples*, pp. 177–80; Chambers, *Yaws*, pp. 1–3, geographical distribution, pp. 28–35, stages of the disease, with plates of lesions, pp. 89–90; Hackett, 'The Human Treponematoses', pp. 152–69; Hackett, 'Treponematoses in Aboriginal Bones', with references and plates of lesions, pp. 387–405; Moodie, *Aboriginal Health*, pp. 165–8; Garner *et al.*, 'Treponemal infection in Australian Northern Territory Aborigines', pp. 285–93.
- ⁷ Hackett, *Boomerang Leg*; Moodie, *Aboriginal Health*, pp. 235–6.
- ⁸ Chambers, *Yaws*, p. 89. The name used for yaws may refer to its origin in French West Africa; Radolf, 'Treponema', p. 473.
- ⁹ Hackett, 'Origin of the Human Treponematoses', pp. 7–41; Radolf, 'Treponema', pp. 471–2.
- ¹⁰ Cornell (trsl.), *Journal of Baudin*, p. 345; Hackett, 'Possible Treponemal Changes', pp. 436–43; personal communication, Hackett, 31 Dec. 1980.
- ¹¹ Ryan, *The Aboriginal Tasmanians*, pp. 171–2.
- ¹² Black, 'Geographic and Sociologic Factors', pp. 28–9; Blumberg, Alter and Visnich, 'A "new" antigen', pp. 541–6; Barwick, 'Changes in Population', pp. 305–6.
- ¹³ Blainey, *The Triumph of the Nomads*, pp. 95–100, 227–8; Fenner, 'Sociocultural Change and Environmental Diseases', p. 14.
- ¹⁴ Petrie, *Tom Petrie's Reminiscences*, pp. 4, 178–9; Curr, *Recollections of Squatting*, p. 42 and *passim*; Curr, *The Australian Race*, vol. 1, p. 268 and *passim*; Dawson, *Aborigines*, p. 62; Bates, *The Native Tribes*, pp. 90–1.
- ¹⁵ Burnet and White, *Natural History of Infectious Disease*, pp. 204–15.
- ¹⁶ Hackett, 'The Human Treponematoses', pp. 162–7.
- ¹⁷ Fenner, 'Sociocultural changes', p. 157; Fenner *et al.*, *Smallpox and its Eradication*, p. 119; Fenner, 'Socialcultural changes and infectious disease', pp. 23–4; Diamond, *Guns, Germs and Steel*, pp. 206–7.
- ¹⁸ Fenner, *Smallpox and its Eradication*, pp. 210–11; Hopkins, *Princes and Peasants*, pp. 14–15, 165, plate 2, p. 172; Diamond, *Guns, Germs and Steel*, p. 205.
- ¹⁹ Diamond, *Guns, Germs and Steel*, pp. 205, 202; Ziegler, *The Black Death*, p. 231.
- ²⁰ McNeill, *Plagues and Peoples*, pp. 203–4; Hopkins, *Princes and Peasants*, pp. 204–15.

- ²¹ Macknight, 'Macassans and the Aboriginal Past', p. 70; Macknight, *The Voyage to Marege*, pp. 94–7, *passim*. Macknight's chronology of the Macassan trepang industry in northern Australia, based on literary evidence, is conservative, but allows for contacts and transmission of disease by about 1750.
- ²² Hunter, *An Historical Journal*, pp. 133–4.
- ²³ Ailments common before European settlement were considered 'natural' rather than the result of sorcery, White (ed.), *The Native Tribes*, p. 288.
- ²⁴ Some observers left records of active Aboriginal smallpox in 1789, 1828–31 and 1866–70. Others who did not see active smallpox left records of pockmarked survivors they had seen in the eighteenth, nineteenth and early twentieth centuries. The rough ages of the pockmarked people have been estimated using age-specific case-fatality rates recorded in the smallpox eradication campaign, which are the only reliable guide to the changing impact of smallpox in different age-groups (see ch. 2). The observers' presence after outbreaks in affected districts was established by using relevant information in their publications, and in the *ADB*. Page references to entries in the *ADB* are included below for important or little-known observers.
- ²⁵ Stirling, 'Preliminary Report on . . . Native Remains . . . and the Alleged . . . Pandemic', pp. 4–15; Dixon, *Smallpox*; Campbell, 'The Early 1830s', pp. 350–1; in South Australia, Kimber, personal communication, 2 Oct. 1986.
- ²⁶ Kerr to Thomas, in Thomas, 'On Variola and Varicella', pp. 47–8.
- ²⁷ Butlin, *Our Original Aggression*, pp. 143–8.
- ²⁸ Wentworth, *Description*, 1819, p. 44; Cunningham, *Two Years*, vol. 1, p. 183; Lesson, *Voyage Medical*, p. 110; R. Dawson, *The Present State*, p. 323; *Sydney Gazette*, 8 Oct. 1831.
- ²⁹ Reece, *Aborigines and Colonists*, p. 128.
- ³⁰ Population estimates, Vamplew (ed.), *Statistics*, p. 26.
- ³¹ Minutes taken before the Select Committee on the Aborigines and Replies to a Circular Letter from the Select Committee, Appendix to the *Report from the Select Committee on the Condition of the Aborigines*, pp. 1–59, Legislative Council, New South Wales, *V&P*, 1845, hereafter Appendix, *Select Committee 1845*.
- ³² Dubos, *The White Plague*, ch. 1, 6, 14, 16; Fenner, 'Sociocultural Change and Environmental Diseases', pp. 14–15.
- ³³ Cook, 'The Significance of Disease', pp. 259–60.
- ³⁴ Dubos, *Man Adapting*, pp. 173–5.
- ³⁵ Black, 'Modern Isolated Populations', p. 49.
- ³⁶ Abrahams, 'Tuberculosis in Indigenous Australians', in A. Proust (ed.), *History of Tuberculosis*, pp. 69–71.
- ³⁷ Hackett, 'Treponematoses (Yaws and Treponarid)', pp. 387–8; Moodie, *Aboriginal Health*, pp. 163–8; Radolf, 'Treponema', pp. 464–73.
- ³⁸ *Encyclopaedia Britannica*, 1911 edn, vol. 13, pp. 939–44, vol. 27, pp. 983–5; Curson, *Times of Crisis*, p. 12; Cunningham, *Two Years*, vol. 1, p. 185.
- ³⁹ Hackett, 'A Critical Survey', pp. 733–4; Butlin, *Our Original Aggression*, p. 39.
- ⁴⁰ Clark, *A History of Australia*, vol. 1, p. 198 and *passim*.
- ⁴¹ Sturt, *Two Expeditions into the Interior*, vol. 2, pp. 154–5; Waterhouse (ed.), *Narrative of . . . Exploration by . . . Eyre*, p. 208.
- ⁴² Collins, *An Account of the English Colony*, vol. 1, pp. 596–7.
- ⁴³ For Sturt's account of diseases in Aborigines, see ch. 6.
- ⁴⁴ Baker, *Days of Wrath*, pp. 115–16, 119–20.
- ⁴⁵ Appendix, *Select Committee 1845*: Polding, Sydney, pp. 6–12, Schmidt, Moreton Bay, pp. 15–16.

- ⁴⁶ Appendix, *Select Committee 1845*: Thomas, Port Phillip District, diseased Aboriginal infants and children, p. 55; Robinson reported Aboriginal victims of European diseases, syphilis, p. 48; syphilis in Aboriginals in New South Wales, pp. 28, 37, 38, 58; promiscuous behaviour, European men and Aboriginal women, *passim*.
- ⁴⁷ Hackett, 'Origin of the Human Treponematoses', pp. 22–3.
- ⁴⁸ Curr, *The Australian Race*, vol. 1, p. 208.
- ⁴⁹ 'F. J. Gillen', *ADB*, vol. 9, pp. 6–7, 'W. Spencer', *ADB*, vol. 12, pp. 33–6; Mulvaney *et al.*, 'My Dear Spencer', pp. 152, 160, 190, 502; Hackett, 'A Critical Survey', pp. 740–3.
- ⁵⁰ Hackett, 'Origin of Human Treponematoses', pp. 740, 735.
- ⁵¹ Basedow, 'Diseases', p. 194; *ADB*, vol. 7, pp. 202–3.
- ⁵² Morgan, *William Buckley*, ed. Sayers, p. 68; White (ed.), *The Native Tribes*, p. 290; Wentworth, *A Description*, p. 44; Lesson, *Voyage Medical*, p. 110; Cunningham, *Two Years*, vol. 1, p. 183; Dawson, *The Present State*, p. 323; Foley, *In Quarantine*.
- ⁵³ Black, 'Geographic and Sociologic Factors', p. 30; size of main cities and towns in Australia in 1851; Blainey, *A Land Half Won*, p. 119; Curson, *Times of Crisis*, pp. 14–15, 54–72.
- ⁵⁴ Curr, *The Australian Race*, vol. 1, p. 342; Moodie, *Aboriginal Health*, pp. 31, 157.
- ⁵⁵ White (ed.), *The Native Tribes*, p. 34; Rowley, *The Destruction*, vol. 1, pp. 102–3; Rowley, *A Matter of Justice*, p. 38; Reynolds, *The Other Side of the Frontier*, pp. 102–4.
- ⁵⁶ 'J. H. L. Cumpston', *ADB*, vol. 7, pp. 174–6; Cumpston, *The History of Smallpox*, pp. 27–8.
- ⁵⁷ Cumpston, *The History of Smallpox*, pp. 9–11, 30–43; Hopkins, *Princes and Peasants*, pp. 89–91, 125–6; *Argus*, 16 and 17 Jan. 1877.
- ⁵⁸ *Argus*, 19 Jan. 1877; 'Thomas Embling', *ADB*, vol. 4, pp. 140–1.
- ⁵⁹ Cumpston, *The History of Smallpox*, pp. 11–23, 43–64, 66–9, 72–3, 75; Curson, *Times of Crisis*, pp. 90–119; Roe, 'Smallpox in Launceston', pp. 111–48.
- ⁶⁰ 'Curr', *ADB*, vol. 3, p. 508.
- ⁶¹ Stirling, 'Preliminary Report', pp. 4–45; Mulvaney and Calaby, *So Much That is New*, pp. 118, 131.
- ⁶² Cleland, 'Diseases Peculiar to Australia'; Cumpston, *Smallpox*, pp. 163–70; Cleland, 'Diseases Among Australian Aborigines', pp. 54–5, 56, 67, 70.
- ⁶³ For Cumpston's account of Aboriginal smallpox, see *The History of Smallpox*, pp. 1–6.

2 'the most dreadful scourge of the human species'

- ¹ Fenner *et al.*, *Smallpox and its Eradication*, pp. 1208–9.
- ² *Ibid.*, ch. 10, *passim*.
- ³ Birdsell, 'Some Factors Influencing Aboriginal Populations', p. 197.
- ⁴ Butlin, *Our Original Aggression*, pp. x–xii, ch. 2–5; demographic modelling of smallpox in south-eastern Australia, pp. 63–70; 'Some General Inferences', pp. 175–6; Butlin, *Economics and the Dreamtime*, p. 125.
- ⁵ Curson, *Times of Crisis*, pp. 48–51; Wilson, *Australia: 1788–1988*, pp. 75–84; Frost, *Botany Bay Mirages*, pp. 190–210.
- ⁶ White and Mulvaney, 'How Many People?', pp. 115–17.
- ⁷ Fenner *et al.*, *Smallpox and its Eradication*, pp. 2–4, 361–2; Cumpston and McCallum, *Smallpox in Australia, 1909–1923*, ch. 3; Foley, *In Quarantine*, pp. 106–9.

- ⁸ Hopkins, *Princes and Peasants*, pp. 25, 168.
- ⁹ Fenner *et al.*, *Smallpox and its Eradication*, pp. 70–5, 96.
- ¹⁰ *Ibid.*, pp. 4–5.
- ¹¹ *Ibid.*, pp. 5–6.
- ¹² *Ibid.*, pp. 19–22; plates 1.4–1.14, pp. 8–18.
- ¹³ *Ibid.*, plates 1.16–1.18, pp. 23–5.
- ¹⁴ *Ibid.*, p. 22.
- ¹⁵ *Ibid.*, pp. 31–2, 37–8, 62–3.
- ¹⁶ *Ibid.*, pp. 27, 228.
- ¹⁷ *Ibid.*, pp. 47–9; plate 1.26.
- ¹⁸ *Ibid.*, pp. 49–50; plate 1.27, p. 57; plate 13.8, p. 657; plate 24.4, p. 1117.
- ¹⁹ Hopkins, *Princes and Peasants*, pp. 42, 69, 75, 80, 131–2, 153–4; Fenner *et al.*, *Smallpox and its Eradication*, p. 50.
- ²⁰ Hopkins, *Princes and Peasants*, pp. 131, 150, 237; Wood, ‘The Impact of Smallpox’, p. 34.
- ²¹ Fenner *et al.*, *Smallpox and its Eradication*, pp. 51–2.
- ²² *Ibid.*, table 1.13, p. 54.
- ²³ *Ibid.*, pp. 54–5; Rao, *Smallpox*, p. 40.
- ²⁴ Hopkins, *Princes and Peasants*, pp. 153–4.
- ²⁵ Fenner *et al.*, *Smallpox and its Eradication*, pp. 169–70.
- ²⁶ *Ibid.*, pp. 173–4.
- ²⁷ *Ibid.*, pp. 179–82.
- ²⁸ *Ibid.*, pp. 96–7, 175–8.
- ²⁹ Hopkins, *Princes and Peasants*, pp. 179–80; Fenner *et al.*, *Smallpox and its Eradication*, pp. 234–5.
- ³⁰ Hopkins, *Princes and Peasants*, pp. 195–7.
- ³¹ Fenner *et al.*, *Smallpox and its Eradication*, pp. 182–90.
- ³² *Ibid.*, pp. 191–4.
- ³³ *Ibid.*, pp. 194–6.
- ³⁴ *Ibid.*, pp. 196–8, 224.
- ³⁵ *Ibid.*, p. 199.
- ³⁶ *Ibid.*, pp. 199–201.
- ³⁷ *Ibid.*, pp. 201–5.
- ³⁸ *Ibid.*, p. 205, table 4.13.
- ³⁹ *Ibid.*, pp. 117–18.
- ⁴⁰ *Ibid.*, pp. 245–58; Razzell, *The Conquest of Smallpox*.
- ⁴¹ Fenner *et al.*, *Smallpox and its Eradication*, pp. 258–62; Cumpston, ‘Public Health in Australia’, p. 497.
- ⁴² Fenner *et al.*, *Smallpox and its Eradication*, pp. 263–5, 283.
- ⁴³ *Ibid.*, p. 271.
- ⁴⁴ *Ibid.*, pp. 273–5; Foley, *In Quarantine*.
- ⁴⁵ McNeill, *Plagues and Peoples*, pp. 120–1; Boccaccio, *The Decameron*, p. 7; Thucydides, *History of the Peloponnesian War*, pp. 151–6.
- ⁴⁶ Fenner *et al.*, *Smallpox and its Eradication*, pp. 235–8; Hopkins, *Princes and Peasants*, ch. 6; Hemming, *Red Gold*, *passim*.
- ⁴⁷ Dobyns, *Their Number Become Thinned*, pp. 14–16.
- ⁴⁸ Fenner *et al.*, *Smallpox and its Eradication*, pp. 235–6; Hopkins, *Princes and Peasants*, p. 234.
- ⁴⁹ The following account is based on Fenner *et al.*, *Smallpox and its Eradication*, pp. 238–40, and Hopkins, *Princes and Peasants*, ch. 7, except when other sources are specified.

- ⁵⁰ Vaughan, *New England Frontier*, pp. 21–2.
- ⁵¹ Duffy, ‘Smallpox and the Indians’, pp. 324–41; S. Cook, ‘The Significance of Disease’, pp. 485–508.
- ⁵² Stearn and Stearn, *The Effect of Smallpox*, pp. 13–16, 42–5.
- ⁵³ Oswalt, *This Land was Theirs*, pp. 267–9, 566.
- ⁵⁴ *Ibid.*, pp. 54, 144, 380.
- ⁵⁵ Fenner *et al.*, *Smallpox and its Eradication*, pp. 165–6.
- ⁵⁶ Stearn and Stearn, *The Effect of Smallpox*, pp. 15, 95, 133; Oswalt, *This Land was Theirs*, pp. 403, 435; Dobyns, *Their Number Become Thinned*, Essay One, pp. 13–16.
- ⁵⁷ Crosby, ‘Virgin Soil Epidemics’, pp. 289–99.
- ⁵⁸ Wood, ‘The Impact of Smallpox’, pp. 35–6; Dobyns, *Their Number Become Thinned*, Essay Two, p. 41; McNeill, *Plagues and Peoples*, pp. 203–5.
- ⁵⁹ Fenner and Fantini, *Biological Control of Vertebrate Pests*, p. 308.

3 Myths

- ¹ Fenner *et al.*, *Smallpox and its Eradication*, p. 64.
- ² Black, ‘Geographic and Sociologic Factors’, p. 28.
- ³ Moodie, *Aboriginal Health*, p. 157; Jezek *et al.*, ‘Facial scarring after varicella’, pp. 798–803; Diamond, *Guns, Germs and Steel*, p. 320.
- ⁴ Fenner *et al.*, *Smallpox and its Eradication*, pp. 199–200.
- ⁵ Jamieson, ‘General Observations’, 1 July 1804, in Cumpston, *The History of Smallpox*, pp. 176–7; J. Mair to Col. Sec., 14 Feb. 1831; J. Bowman, Medical Department Sydney, to Col. Sec., 10 Oct. 1831, Archives Office of NSW, ref. 31/10001 with 32/34, 4/2130, hereafter NSW 4/2130; Bates, *The Native Tribes*, pp. 296–7.
- ⁶ G. Busby, Bathurst, to Inspector of Colonial Hospitals, Sydney, 19 Oct. 1831, NSW 4/2130; Cumpston, *The History of Smallpox*, pp. 148–150; *Sydney Gazette*, 29 Nov. and 10 Dec. 1831.
- ⁷ Dixon, *Smallpox*, pp. 205–7; Crosby, *Ecological Imperialism*, pp. 309–11.
- ⁸ Urry, ‘Beyond the Frontier’, pp. 6–7, n.27; Curson, *Times of Crisis*, pp. 43–4; Frost, *Botany Bay Mirages*, p. 205.
- ⁹ King in Hunter, *An Historical Journal*, p. 406, Fitzhardinge (ed.), *Sydney’s First Four Years*, pp. 146–51; J. Mair to Col. Sec., 14 Feb. 1831, NSW 4/2130.
- ¹⁰ Bennett, *Wanderings in NSW*, vol. 1, ch. 1; *ADB*, vol. 1, pp. 85–6. Cumpston’s access to official records was limited, and he used Bennett’s findings. Cumpston, *The History of Smallpox*, pp. 150–4.
- ¹¹ J. Mair to Col. Sec., 26 Sept. 1831; A. C. Imlay to J. Bowman, 5 Oct. 1831; J. Mair to Col. Sec., 10 Dec. 1831, with enclosure: ‘Observations on the Eruptive Febrile disease which prevailed among the Aborigines during 1830 and 1831’, pp. 1–26; NSW 4/2130; Minutes of the Executive Council, vol. 3, 3 Jan. 1832, item 7, pp. 420–1, NSW 4/1517; personal communication, Frank Fenner, 21 April 1983.
- ¹² Curr, *The Australian Race*, vol. 1, p. 218.
- ¹³ Barwick, ‘Changes in the Aboriginal Population’, pp. 1, 307; Barwick, ‘Mapping the Past’, pp. 103, 109, n.13; Cumpston, *The History of Smallpox*, pp. 2–6.
- ¹⁴ *Argus*, 22, 24, 25, 29 Jan. 1877; Clark, *History of Australia*, vol. 1, pp. 128–9; Ann Blainey and Mary Lazarus, ‘George William Rusden’, in *ADB*, vol. 6, pp. 72–3.
- ¹⁵ Critchett, *A Distant Field of Murder*, ch. 5, p. 84.
- ¹⁶ Taplin (ed.), *Folklore*, pp. 44–6.
- ¹⁷ Basedow, ‘Diseases of the Australian Aborigines’, p. 183.
- ¹⁸ Bates, *The Native Tribes*, p. 296.
- ¹⁹ Fitzhardinge (ed.), *Sydney’s First Four Years*, p. 146.

- ²⁰ 'W. C. Wentworth', in *ADB*, vol. 2, pp. 582–9; Wentworth, *A Statistical, Historical and Political Description*, 1819 edn, p. 44.
- ²¹ Debenham (ed.), *The Voyage of Captain Bellingshausen*, vol. 2, pp. 336, 352.
- ²² Lesson, *Voyage medical autour du monde*, pp. 110–11.
- ²³ Wentworth, *A Statistical, Historical and Political Description*, 3rd edn, 1824, vol. 1, pp. 310–11.
- ²⁴ *The Australian Race*, vol. 1, ch. 8, pp. 225–6, 216–17; reports of sickness and deaths of convicts on the *Alexander* transport, Phillip to Sydney, 5 June 1787, *HRNSW*, vol. 1, no. 2, pp. 107ff; Phillip to Sydney, 30 Aug. 1787, *ibid.*, pp. 111ff; Frost, *Botany Bay Mirages*, pp. 195–6.
- ²⁵ Butlin, *Our Original Aggression*, pp. 32–3 and *passim*.
- ²⁶ Moorehead, *The Fatal Impact*, p. 145; Docker, *Simply Human Beings*, p. 51; Abbie, *The Original Australians*, p. 93; Butlin, *Our Original Aggression*, pp. 175, 21–2; Curson, *Times of Crisis*, pp. 46–7, 52–3.
- ²⁷ Fenner *et al.*, *Smallpox and its Eradication*, pp. 115–16.
- ²⁸ Hunter, *An Historical Journal*, pp. 72–5; Worgan, *Journal of a First Fleet Surgeon*, pp. 23–4; Fitzhardinge (ed.), *Sydney's First Four Years*, pp. 69–70, 117, 221, 264–7.
- ²⁹ Docker, *Simply Human Beings*, p. 52; Moorehead, *The Fatal Impact*, p. 171.
- ³⁰ Cumpston, *The History of Smallpox*, p. 77.
- ³¹ Butlin, *Our Original Aggression*, pp. 34–6; Butlin, *Economics and the Dreamtime*, pp. 113, 116.
- ³² Darling to Huskisson, 28 Aug. 1828, with enclosure, Minutes of the Proceedings of the Executive Council, 29 July 1828, with Appendix S and Appendix T, *HRA*, vol. 1, no. 14, pp. 348–50; Foley, *In Quarantine*, pp. 18–19.
- ³³ Fenner *et al.*, *Smallpox and its Eradication*, pp. 238–9; Fenner, 'Smallpox, "the most dreadful scourge of the human species"', pp. 730–1; Hopkins, *Princes and Peasants*, pp. 204–14, 235–41.
- ³⁴ Mair, 'Observations', p. 14.
- ³⁵ Cumpston, *The History of Smallpox*, pp. 65–7.

4 The Indonesian Archipelago

- ¹ India: Fenner *et al.*, *Smallpox and its Eradication*, pp. 211, 214, 217–19, plate 5.2; Hopkins, *Princes and Peasants*, ch. 4. China: Fenner *et al.*, *Smallpox and its Eradication*, pp. 215–16, 225–6; Hopkins, *Princes and Peasants*, ch. 3; McNeill, *Plagues*, pp. 293–302.
- ² Fenner *et al.*, *Smallpox and its Eradication*, pp. 219, 223–5; Hopkins, *Princes and Peasants*, pp. 153–4.
- ³ Fenner, 'Smallpox in Southeast Asia', pp. 34–48; Fenner *et al.*, *Smallpox and its Eradication*, p. 228; A. Reid, 'Low Population Growth'.
- ⁴ Hopkins, *Princes and Peasants*, pp. 123–4.
- ⁵ *Ibid.*, p. 113; Boomgaard, 'Morbidity and Mortality in Java', pp. 60–4; Lovric, 'Bali: Myth, Magic and Morbidity', pp. 125–3; Fenner *et al.*, *Smallpox and its Eradication*, pp. 631–2, 654–5.
- ⁶ Hopkins, *Princes and Peasants*, pp. 113, 115; Evans, *The Religion of the Tempusak Dusuns*, pp. 39, 151–5, 193–5; St John, *Life in the Forest of the Far East*, pp. 61–2.
- ⁷ Hopkins, *Princes and Peasants*, p. 113.
- ⁸ *Encyclopaedia Britannica*, 1910–11 edn, vol. 5, pp. 596–9; South Sulawesi and Macassar 1838: van der Velde, *Gezigten uit Neerlands Indië*, 1979 edn, p. 56. I am indebted to Macknight for this information.

- ⁹ Mundy, *Narrative of Events in Borneo and Celebes*, vol. 1, pp. 60, 154–6; Macknight, ‘Macassans and the Aboriginal Past’, p. 74.
- ¹⁰ Fenner *et al.*, *Smallpox and its Eradication*, pp. 635, 652–3, table 13.7.
- ¹¹ Macknight, ‘Macassans and the Aboriginal Past’; Fox, *Harvest of the Palm*, pp. 166–7.
- ¹² Miklouho-Maclay, *New Guinea Diaries 1871–1883*, pp. 91, 179.
- ¹³ Sack and Clark, *German New Guinea: The Annual Reports*, pp. 81, 101, 110, 122, 186; Hahl, *Governor in New Guinea*, pp. 13–14.
- ¹⁴ Fenner *et al.*, *Smallpox and its Eradication*, p. 242.
- ¹⁵ Mulvaney, *The Prehistory*, ch. 1; Mulvaney, *Encounters in Place*, ch. 4.
- ¹⁶ Macknight, *The Voyage to Marege*, Introduction, ch. 1 and 2; Macknight, ‘Macassans and the Aboriginal Past’, p. 70.
- ¹⁷ Macknight, *The Voyage to Marege*, pp. 28–9.
- ¹⁸ *Ibid.*, ch. 4 and 6, plates 3, 11; Mulvaney, *Encounters in Place*, pp. 22–8.
- ¹⁹ For Flinders’ meeting with Pobassoo, see Mulvaney, *Encounters in Place*, p. 23; ‘Flinders’, *ADB*, vol. 1, pp. 389–91; Flinders, *A Voyage to Terra Australis*, vol. 2, pp. 229–33.
- ²⁰ Smallpox in South Sulawesi: ‘Robert Brown’, *ADB*, vol. 1, pp. 166–7; Brown, Diary, 1801–5, 18 Feb. 1803, mfm, c.10, 022. He noted faint traces of smallpox on the faces of Pobassoo’s crew: Macknight, ‘Macassans and Aborigines’, p. 292.
- ²¹ Campbell, ‘Smallpox in Aboriginal Australia, 1829–31’, pp. 546–7; Macknight, *The Voyage to Marege*, pp. 129–32; Wilson, *Narrative of a Voyage*, Appendix, ‘Remarks on transportation’, p. 334. Wilson, RN, was Surgeon Superintendent on convict transports between 1822 and 1836. Vaccination was available, so smallpox did not affect his convict charges, and he recorded deaths of under 2 per cent among them, *ADB*, vol. 2, p. 612.
- ²² de la Rue, *Pearl-shell and Pastures*, pp. 71–83.
- ²³ R. Sholl, Government Resident, Roebourne, to Col. Sec., 7 Dec. 1875, with enclosure: Bompas, ‘Vessels visited under the Quarantine Act’, WAA/Roebourne/809.
- ²⁴ de la Rue, *Pearl-shell and Pasture*.
- ²⁵ Fremantle, Port Topics, *Western Australian Times*, 7 Mar. 1876.
- ²⁶ Berndt and Berndt, *Arnhem Land*, p. 75.
- ²⁷ ‘Foelsche’, *ADB*, vol. 4, pp. 192–3. Revaccination of German military recruits: Hopkins, *Princes and Peasants*, pp. 88–91; Foelsche, ‘Notes’, *TPRSSA*, p. 8.
- ²⁸ Cumpston, *The History of Smallpox*, pp. 76, 102–3; Searcy, *In Australian Tropics*, pp. 331–3; Cook, ‘Medicine and the Australian Aboriginal’, pp. 559–65.
- ²⁹ Cumpston, *The History of Smallpox*, p. 76.
- ³⁰ Fenner *et al.*, *Smallpox and its Eradication*, pp. 1073–5.
- ³¹ Flannery, *The Future Eaters*, pp. 334–6.
- ³² Kettle, ‘Tuberculosis in the Northern Territory’, in Proust, *History of Tuberculosis in Australia*, p. 71.
- ³³ Moodie, *Aboriginal Health*, p. 152.
- ³⁴ Diamond, *Guns, Germs and Steel*, p. 314.
- ³⁵ Macknight, *The Voyage to Marege*, ch. 8.

5 Hidden History

- ¹ Curr, *The Australian Race*; Butlin, *Our Original Aggression*; Diamond, *Guns, Germs and Steel*, p. 320.
- ² See ch. 3, pp. 10–12; Wilson, *Australia: 1788–1988*, pp. 77–9.

- ³ Foelsche, 'Notes', *TPRSSA*, p. 8.
- ⁴ *New South Wales Medical Gazette*, vol. 2, 1871, Aug. 1872, editorial.
- ⁵ See ch. 7 and 8; Curson, *Times of Crisis*, pp. 51–3.
- ⁶ Phillip to Sydney, 12 Feb. 1790, *HRNSW*, vol. 1, no. 2, pp. 298, 308–9.
- ⁷ *ADB*, vol. 1, pp. 236–40; Collins, *An Account of the English Colony*, vol. 1, pp. 65–6.
- ⁸ *Ibid.*, Appendix, pp. 597–8.
- ⁹ Scott, *Remarks on a Passage*, p. 47.
- ¹⁰ Fitzhardinge (ed.), *Sydney's First Four Years*, pp. 146–50.
- ¹¹ *Ibid.*, pp. 186, 229–30, 285.
- ¹² Hunter, *An Historical Journal*, pp. 133–4, 138–40.
- ¹³ 'William Bradley', in *ADB*, vol. 1, pp. 145–6; Bradley, *A Voyage to New South Wales*, pp. 161–6, 181–2.
- ¹⁴ Gunn, *Links with the Past*, pp. 17–18. Arrival of the Second Fleet on 3 June 1789, in Clark, *A History of Australia*, vol. 1, p. 122.
- ¹⁵ Fidlon and Ryan (eds), *The Journals of Lt Ralph Clark, 1787–1792*, p. 109; Lt Fowell to his Father, 31 July 1790, *HRNSW*, vol. 1, no. 2, pp. 276–7.
- ¹⁶ Barrington, *A Voyage to Botany Bay*, pp. 66–7; Price Journal, f. 87; Peron, *A Voyage of Discovery*, pp. 290–1.
- ¹⁷ Wentworth, *A Statistical, Historical and Political Description*, 1819 edn, p. 44.
- ¹⁸ Appendix, *Select Committee 1845*, p. 3.
- ¹⁹ Frost, *Botany Bay Mirages*, pp. 203–5.
- ²⁰ Fenner *et al.*, *Smallpox and its Eradication*, pp. 191–3, 194.
- ²¹ Curson, *Times of Crisis*, p. 52.
- ²² Fenner *et al.*, *Smallpox and its Eradication*, pp. 152–4.
- ²³ Collins, *An Account of the English Colony*, vol. 1, pp. 23–4, 30–1, 43, 57–8.
- ²⁴ Clark, *A History of Australia*, vol. 1, p. 90.
- ²⁵ Curson, *Times of Crisis*, p. 49.
- ²⁶ *New South Wales Medical Gazette*, vol. 2, 1871, Aug. 1872; Hiatt, 'Mystery at Port Hacking', pp. 313–17.
- ²⁷ Fitzhardinge (ed.), *Sydney's First Four Years*, pp. 137, 151, 176–81.
- ²⁸ Proust (ed.), *History of Tuberculosis in Australia*, pp. 5–6.
- ²⁹ Collins, *An Account of the English Colony*, vol. 1, appendix, p. 607.
- ³⁰ Clark, *A History of Australia*, vol. 1, p. 151.
- ³¹ Lesson, *Voyage Medical*, pp. 110, 112, 113; Proust (ed.), *History of Tuberculosis in Australia*, p. 7.
- ³² Black, 'An Explanation of High Death Rates', pp. 292–4.
- ³³ Vamplew (ed.), *Australian Historical Statistics*, p. 26.

6 The Frontiers of Eastern Australia 1824–1830

- ¹ 'T. B. Wilson', in *ADB*, vol. 2, p. 612; Wilson, *Narrative of a Voyage*, pp. 1–2, 71–2, 151, 157.
- ² *Ibid.*, pp. 80–2, 155.
- ³ *Ibid.*, pp. 169–70.
- ⁴ *Ibid.*, pp. 112, 170, 319.
- ⁵ Macknight questions claims by Wilson and others that Macassan activities extended beyond the bottom of the Gulf of Carpentaria. Macknight, *The Voyage to Marege*, p. 152, note 56.
- ⁶ Bremer to Beaufort, 7 Dec. 1838, in Allen, Archeology, and the History of Port Essington, vol. 1, p. 399.
- ⁷ Curr, *The Australian Race*, vol. 2, p. 234; vol. 3, pp. 78–9; vol. 2, p. 182.

- ⁸ Ibid., vol. 3, pp. 96–7.
- ⁹ Gunn, *Links with the Past*, p. 18.
- ¹⁰ Curr, *The Australian Race*, vol. 3, p. 114.
- ¹¹ Ibid., vol. 3, pp. 121–2.
- ¹² Ibid., vol. 3, pp. 126, 144–5.
- ¹³ Petrie, *Tom Petrie's Reminiscences*, pp. 5, 65; ADB, vol. 5, pp. 400–1.
- ¹⁴ Cilento and Lack, 'Wild White Men' in *Queensland*, pp. 10–11; Lang, *Queensland*, p. 340.
- ¹⁵ Curr, *The Australian Race*, vol. 3, pp. 222, 231.
- ¹⁶ Gibbney, 'Charles Sturt', in ADB, vol. 2, pp. 495–8; 'Hamilton Hume', in ADB, vol. 1, pp. 564–5.
- ¹⁷ Sturt, *Two Expeditions*, vol. 1, pp. 84–101, esp. 93–4.
- ¹⁸ Ibid., pp. 102–5, 107.
- ¹⁹ Bennett, *Wanderings in New South Wales*, p. 153, note.
- ²⁰ Sturt, *Two Expeditions*, vol. 1, pp. 108–39, 140–50.
- ²¹ Baker, 'Thomas Mitchell', ADB, vol. 2, pp. 238–42.
- ²² Mitchell, *Three Expeditions into the Interior*, vol. 2, p. 218.
- ²³ Ibid., pp. 240, 257, 261.
- ²⁴ Ibid., pp. 262–3.
- ²⁵ Ibid., p. 307.
- ²⁶ Parker, *The Euablayi Tribe*, p. 39.
- ²⁷ Baker, *The Civilised Surveyor*, *passim*.
- ²⁸ Sturt, *Two Expeditions*, vol. 2, pp. 28–9, 37, 45–6.
- ²⁹ Ibid., pp. 50–1, 55, 57–8.
- ³⁰ Ibid., pp. 69–73, 81, 86–91.
- ³¹ Ibid., pp. 94–7, 100.
- ³² Ibid., pp. 102–7, 110–11, 114, 115–16.
- ³³ Ibid., pp. 120–1, 124–5.
- ³⁴ Ibid., pp. 125–9.
- ³⁵ Ibid., pp. 130–2, 134–5, 138.
- ³⁶ Ibid., pp. 148–9.
- ³⁷ Ibid., pp. 154–5, 157.
- ³⁸ Ibid., pp. 197–9, 200–2, 214–15, 223.
- ³⁹ Moodie, *Aboriginal Health*, pp. 151–4.
- ⁴⁰ Chambers, *Yaws*, pp. 89–90, 112–16; for yaws and non-venereal endemic syphilis, see ch. 1, pp. 3–6; Hackett, 'Treponematoses in Exhumed Bones', pp. 387–8.
- ⁴¹ Hackett, 'A Critical Survey', p. 734.
- ⁴² I am indebted to John Rudder, AIATSIS, for information about the use of white during mourning.
- ⁴³ See Chapter 7, p. 147.
- ⁴⁴ Eyre, *Journals*, vol. 2, p. 379.
- ⁴⁵ Hawker, *Early Experiences in South Australia*, p. 19.
- ⁴⁶ 'Angas', in ADB, vol. 1, pp. 18–19; Angas, *Savage Life and Scenes*, vol. 1, p. 206.
- ⁴⁷ T. Moulden, *Argus*, 24 Jan. 1877.
- ⁴⁸ Stirling, 'Preliminary Report', p. 16.
- ⁴⁹ G. Clapham, in Smyth, *The Aborigines of Victoria*, vol. 1, p. 254.
- ⁵⁰ Woods (ed.), *The Native Tribes of South Australia*, Introduction, p. xiv; Taplin, 'The Narrinyeri', in *ibid.*, pp. 44–5.
- ⁵¹ Stirling, 'Preliminary Report', pp. 16–19; Gilbert, *Living Black*, pp. 25–31.
- ⁵² *Register*, Adelaide, 5 July 1911; Stirling, 'Preliminary Report', pp. 40–1; Welinyeri clan on the Murray, in Taplin, *Folklore*, p. 34.

- ⁵³ Stirling, 'Preliminary Report', p. 38.
- ⁵⁴ TPRSAA, vol. 5, 1881–82, 2 May 1882, Abstract of Proceedings, p. 110; *South Australian Gazette*, no. 77, 11 July 1839.
- ⁵⁵ Hahn, *Reminiscences of Captain Hahn, 1838–9*, p. 129; Teichelmann and Schurmann, *Outlines of Language of South Australia*, p. 34; Moorehouse, *Vocabulary of the Murray River*, p. 47; Adams, *My Early Days*, p. 8; Stirling, 'Preliminary Report', p. 19.
- ⁵⁶ *Ibid.*, p. 29; Curr, vol. 2, p.136; Thomas, 'On variola and varicella', pp. 47–8.
- ⁵⁷ Butlin, *Our Original Aggression*, p. 27.
- ⁵⁸ Fenner *et al.*, *Smallpox and its Eradication*, p. 191.
- ⁵⁹ Kettle, 'Tuberculosis in the Northern Territory', in Proust (ed.), *History of Tuberculosis*, p. 71.
- ⁶⁰ E. Abrahams, in *ibid.*, p. 46; Petrie, *Tom Petrie's Reminiscences*, pp. 65–6.
- ⁶¹ Mulvaney, *Encounters in Place*, p. 80.
- ⁶² Moodie, *Aboriginal Health*, p. 148.
- ⁶³ Blainey, *A Land Half Won*, p. 78.

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- ¹ *Sydney Gazette*, 14 Sept. 1830; J. Mair to Col. Sec., 14 Feb. 1831, NSWA 4/2130.
- ² J. Bowman, Medical Department, to Col. Sec., 14 Nov. 1831, NSWA 4/2130.
- ³ *Sydney Gazette*, 9 Aug., Sept., Oct., Nov., Dec. 1831. *Commissioned Officers in the Medical Services of the British Army, 1660–1960*, vol. 1: Alexander Imlay, no. 4228, p. 283, J. Mair, no. 4039, p. 270, J. Mair to Col. Sec., 26 Sept. 1831, NSWA 4/2130; Hopkins, *Princes and Peasants*, p. 87; Fenner *et al.*, *Smallpox and its Eradication*, p. 231.
- ⁴ Minute no. 1, 3 Jan. 1832, Minutes of the Executive Council, vol. 3, NSWA 4/1517.
- ⁵ Campbell, 'Smallpox in Aboriginal Australia, 1829–31', pp. 536–56; Campbell, 'Smallpox in Aboriginal Australia, the Early 1830s', pp. 336–58.
- ⁶ Mair, 'Observations', pp. 4–7, NSWA 4/2130.
- ⁷ *Ibid.*, pp. 2–3.
- ⁸ *Ibid.*, pp. 21–2; J. T., *Argus*, 9 Feb. 1877.
- ⁹ Mair, 'Observations', NSWA 4/2130, p. 4.
- ¹⁰ Threlkeld, *An Australian Language*, pp. 79, 80, 88; Wilkes, *Narrative of the United States Exploring Expedition*, vol. 2, p. 197.
- ¹¹ Mair, 'Observations', NSWA 4/2130, p. 4.
- ¹² *Ibid.*, pp. 11–13.
- ¹³ *Ibid.*, p. 3.
- ¹⁴ J. Mair to Col. Sec., 26 Sept. 1831; A. C. Imlay to J. Bowman, 5 Oct. 1831; NSWA 4/2130.
- ¹⁵ George Busby, Bathurst, to Inspector of Colonial Hospitals, 19 Oct. 1831, NSWA 4/2130; NSW *Medical Gazette* 1874–75; Cumpston, *Smallpox*, pp. 148–50.
- ¹⁶ Mair, 'Observations', NSWA 4/2130, pp. 15–21.
- ¹⁷ Mitchell, *Three Expeditions*, vol. 1, p. 26.
- ¹⁸ Captain Smyth, Port Macquarie, to Col. Sec., 24 Nov. 1831, NSWA 01/9834.
- ¹⁹ J. Mair & A. C. Imlay to Col. Sec., 24 Dec. 1831, NSWA 4/2130.
- ²⁰ Mair, 'Observations', NSWA 4/2130, pp. 13–15.
- ²¹ *Ibid.*, p. 3.
- ²² *Ibid.*, pp. 9–10.
- ²³ *Ibid.*, pp. 10–11.

- ²⁴ Ibid., pp. 22, 24.
- ²⁵ Ellis, *Braidwood*, p. 36.
- ²⁶ Sturt Papers, p. 167; Cumpston, *Charles Sturt*, pp. 84–5.
- ²⁷ Mackaness (ed.), *George Augustus Robinson's Journey*, p. 32, note; 'George Augustus Robinson', *ADB*, vol. 2, pp. 385–7; 'George Henry Haydon', *ADB*, vol. 4, pp. 362–3.
- ²⁸ Mackaness (ed.), *George Augustus Robinson's Journey*, pp. 13, 19, 28.
- ²⁹ Ibid., pp. 22–3; Imlay brothers, *ADB*, vol. 2, pp. 2–3.
- ³⁰ Mackaness (ed.), *George Augustus Robinson's Journey*, p. 26.
- ³¹ Ibid., p. 28.
- ³² Appendix, *Select Committee 1845*, p. 27; Fraser, *The Aborigines of New South Wales*, p. 62; Mullins, 'A Brief History of Smallpox and Vaccination', p. 492.
- ³³ For example, Scone, Dungog, Brisbane Water, Shoalhaven, Wollongong, Windsor, McLeay River, Port Macquarie, Raymond Terrace, Newcastle, Broulee, Murrumbidgee, Maneroo, Appendix, *Select Committee 1845*, *passim*.
- ³⁴ Hopkins, *Princes and Peasants*, pp. 153–4.
- ³⁵ Bonwick, *Port Phillip Settlement*, pp. 15, 28.
- ³⁶ Beveridge, *Argus*, 27 Jan. 1877; Beveridge, 'Of the Aborigines Inhabiting', p. 35.
- ³⁷ Kirby, *Old Times*, pp. 77–8; *Argus*, 7 Feb. 1877.
- ³⁸ Hamilton, *Pioneering Days*, p. 100; Trangmar, 'The Aborigines of Victoria', p. 8; Hawdon, *Journal 1838*, pp. 31, 27–8.
- ³⁹ Kirby, *Old Times*; Reece, *Aborigines and Colonists*, pp. 24–5.
- ⁴⁰ Morrison, *Early Days*, p. 36.
- ⁴¹ Kerr, *Glimpses of Life*, p. 16.
- ⁴² Stone, 'Lake Boga', pp. 433–4, 462.
- ⁴³ Curr, *Recollections*, pp. 230–6; Curr, *The Australian Race*, vol. 3, pp. 566–7.
- ⁴⁴ Curr, *Argus*, 29 Jan. 1877; Curr, *Recollections*, pp. 236–9; Curr, *The Australian Race*, vol. 3, p. 569.
- ⁴⁵ Albert A. C. Le Soeuf to Curr, 14 Feb. 1877, in Curr, *The Australian Race*, vol. 1, pp. 217–18.
- ⁴⁶ *Age*, 16 Sept. 1993, p. 8.
- ⁴⁷ Campbell, 'Smallpox in Aboriginal Australia, The Early 1830s', pp. 350–1; Hamilton, *Pioneering Days*, p. 48; Trangmar, 'A Short Talk'.
- ⁴⁸ Appendix, *Select Committee 1845*, p. 48.
- ⁴⁹ Presland (ed.), 'Journals of G. A. Robinson, 1841', no. 11, p. 168; no. 6, p. 48; J. Dawson, *Australian Aborigines*, pp. 60–1.
- ⁵⁰ Thomas, 'On Variola and Varicella', pp. 47–8; Kerr, *Glimpses of Life*, p. 16.
- ⁵¹ Withers, *The History of Ballarat*, p. 10.
- ⁵² Morgan, *Life and Adventures of William Buckley*, ed. Sayers, pp. 68, 72–5.
- ⁵³ Peter Snodgrass, in Bride (ed.), *Letters from Victorian Pioneers*, pp. 213–16.
- ⁵⁴ William Thomas, in Bride (ed.), *Letters from Victorian Pioneers*, pp. 397, 420–6; Braim, *A History*, vol. 2, p. 245; Radcliffe-Brown, 'The Rainbow-Serpent Myth', pp. 342–7.
- ⁵⁵ Haydon, *Five Years Experience*, p. 23.
- ⁵⁶ Curr, *The Australian Race*, vol. 3, p. 545; Pepper and De Araugo, *The Kurnai of Gippsland*, pp. 118–19ff.
- ⁵⁷ Critchett, *A Distant Field of Murder*, p. 78.
- ⁵⁸ Mulvaney, *Cricket Walkabout*, pp. 13–17.
- ⁵⁹ Flannery, *The Future Eaters*, p. 326; Critchett, *A Distant Field of Murder*, pp. 79–80, Appendix 3, pp. 242–55.

- ⁶⁰ Curr, *The Australian Race*, vol. 1, p. 213, vol. 3, pp. 539, 545; McBryde, 'Kulin greenstone quarries', pp. 267–85.
- ⁶¹ Mackaness (ed.), *George Augustus Robinson's Journey*, p. 64; Pepper and De Araugo, *The Kurnai of Gippsland*, p. 80.
- ⁶² Watson, *Caledonia Australis*, pp. 80, 163.
- ⁶³ Hopkins, *Princes and Peasants*, pp. 270–4.
- ⁶⁴ Diamond, *Guns, Germs and Steel*, pp. 310, 320.
- ⁶⁵ Pepper and De Araugo, *The Kurnai of Gippsland*, p. 172.
- ⁶⁶ Appendix, *Select Committee 1845*, pp. 26, 33, 35–6; Barwick, 'Aboriginal Population', p. 305.
- ⁶⁷ W. Thomas, *Guardian*, Victoria, Parliamentary Papers, vol. 39, 1861, p. 27.

8 Eastern Australia 1860–1867

- ¹ Curr, *The Australian Race*, vols 1, 2, 3, *passim*; *ibid.*, vol. 1, pp. 208–27; Tidswell, 'History of Small-pox and Vaccination', pp. 1058–66.
- ² Butlin, 'Macassans and Aboriginal Smallpox', pp. 315–35.
- ³ Mulvaney, *Encounters in Place*, p. 26.
- ⁴ For provisions for shelter and food gathering on Cape York Peninsula, see Wharton (ed.), *Uningan Guide*, pp. 10–14.
- ⁵ The occurrence and spread of smallpox in north-eastern and northern Australia was affected by the amount and seasonal distribution of rainfall, which limited the frequency of infectious contacts: summer maximum, Nov.–April, *Macquarie Illustrated World Atlas*, p. 162; Rose, *Hidden Histories*, pp. 7ff. Foelsche associated outbreaks of smallpox with the dry season, 'Notes', *TPRSSA*, p. 7.
- ⁶ Mulvaney, 'The Chain of Connection', in Mulvaney and White (eds), *Australians to 1788*, pp. 72–94.
- ⁷ Earlier studies emphasized mortalities in children under ten in eighteenth-century Europe and twentieth-century Bombay. In 1986 Crosby stressed the susceptibility of the young and the loss of an entire generation in a few weeks, *Ecological Imperialism*, pp. 39–40; studies by Rao establish that children and young adults from five to nineteen years were more likely to survive smallpox than any other age-group, as the evidence of Foelsche and Sholl suggests.
- ⁸ Foelsche, 'Notes', *TPRSSA*, pp. 7–9.
- ⁹ Curr, *The Australian Race*, vol. 2, pp. 330–5. Settlers probably knew Mayi-Yapi speakers on the Flinders and Cloncurry rivers; Palmer, *Early Days*, pp. 101–4.
- ¹⁰ 'W. O. Hodgkinson', in *ADB*, vol. 4, pp. 404–5; Curr, *The Australian Race*, vol. 2, p. 432.
- ¹¹ 'W', *Argus*, 25 Jan. 1877.
- ¹² G. Reid, *A Picnic with the Natives*, pp. 28–40.
- ¹³ Foelsche, 'Notes', *TPRSSA*, pp. 7–8, and in Foelsche, Appendix, *Bulletin of the Northern Territory*, vol. 1, 1912, pp. 65–6.
- ¹⁴ Curr, *The Australian Race*, vol. 1, pp. 221, 250, 270–3.
- ¹⁵ Searcy, *In Australian Tropics*, pp. 19–20; Allen, 'Archeology, and the History of Port Essington', vol. 1, p. 399.
- ¹⁶ Macknight, 'Macassans and the Aboriginal Past', pp. 72–3; Escape Cliffs, *Australian Encyclopaedia*, 1983, vol. 3.
- ¹⁷ Curr, *The Australian Race*, vol. 1, pp. 250–7.

- ¹⁸ Foelsche, 'Notes', *TPRSSA*, pp. 15–16; Curr, *The Australian Race*, vol. 1, p. 253.
- ¹⁹ 'Ralph Tate', in *ADB*, vol. 6, pp. 243–4; *TPRSSA*, vol. 5, 1881–82, Abstract of Proceedings, 2 May 1882, p. 110; 13 June 1882, p. 112.
- ²⁰ Berndt and Berndt, *Arnhem Land*, pp. 78, 218; Coppinger, *Cruise of the 'Alert'*, p. 206; Basedow, 'Diseases of the Australian Aborigines', p. 183.
- ²¹ Wildey, *Australasia and the Oceanic Region*, pp. iv, 96, 98, 115–16.
- ²² Eylmann, *Die Eingeborenen der Kolonie Sudaustralien*, p. 439.
- ²³ Foelsche, 'Notes', *TPRSSA*, p. 8; Macknight, 'Macassans and the Aboriginal Past', pp. 72–3.
- ²⁴ Rose, *Hidden Histories*, pp. 74–8, 5, 113–18.
- ²⁵ Diamond, *Guns, Germs and Steel*, p. 320.
- ²⁶ Frost, *Botany Bay Mirages*, pp. 207–8; Fenner *et al.*, *Smallpox and its Eradication*, ch. 17, 'Nigeria', pp. 880–8.
- ²⁷ For smallpox as a new disease in South Africa, see Hopkins, *Princes and Peasants*, pp. 178–80; Fenner *et al.*, *Smallpox and its Eradication*, pp. 234–5; in the Americas, Hopkins, *Princes and Peasants*, ch. 6 and 7, *passim*; Fenner *et al.*, *Smallpox and its Eradication*, pp. 235–40.
- ²⁸ 'W', *Argus*, 25 Jan. 1877.
- ²⁹ Personal communication, T. Donaldson and J. Simpson, Feb. 2000.
- ³⁰ G. Reid, *A Picnic with the Natives*, pp. 129–30.
- ³¹ Schulze, 'The Aborigines of the Finke River', p. 218.
- ³² Strehlow, *Die Aranda und Loritja*, p. 1361.
- ³³ Eylmann, *Die Eingeborenen der Kolonie Sudaustralien*, p. 439.
- ³⁴ 'W. H. Tietkins', in *ADB*, vol. 6, pp. 275–6; *TPRSSA*, Abstract of Proceedings, 13 June 1882, p. 112; Curr, *The Australian Race*, vol. 1, pp. 214–15; 'Ernest Giles', in *ADB*, vol. 4, pp. 246–7; Giles, *Geographic Travels in Central Australia*, p. 183.
- ³⁵ Curr, *The Australian Race*, vol. 1, pp. 416–17; 'F. J. Gillen', in *ADB*, vol. 9, pp. 6–7; Mulvaney, *So Much That Is New*, pp. 162–3; Stirling, 'Preliminary Report . . . on the Alleged Occurrence of a Pandemic', p. 29.
- ³⁶ Gason, *The Dieyerie Tribe*, p. 28.
- ³⁷ Fenner *et al.*, *Smallpox and its Eradication*, p. 205, and ch. 22, pp. 1058–60.
- ³⁸ Clark, *A History of Australia*, vol. 4, pp. 150–62.
- ³⁹ Howitt, 'Notes on the Aborigines of Cooper's Creek', pp. 300–9.
- ⁴⁰ McBryde, 'Goods from Another Country', ch. 13, in Mulvaney and White (eds), *Australians to 1788*, pp. 253–73.
- ⁴¹ Kimber and Smith, 'An Aranda Ceremony', ch. 11, in Mulvaney and White (eds), *Australians to 1788*, pp. 221–36.
- ⁴² In 1908, when Erhard Eylmann described them, victims who had smallpox in the 1820s would have been not just *alt*, but *uralt*, or extremely old.
- ⁴³ Schulze, 'The Aborigines of the Finke River', pp. 234–5.
- ⁴⁴ Richardson, *The Pioneers of the North-West*, p. 95.
- ⁴⁵ Schulze, 'The Aborigines of the Finke River', pp. 210–55.
- ⁴⁶ Hopkins, *Princes and Peasants*, pp. 183–8; Dobyns, *Their Number*, pp. 13–14.
- ⁴⁷ Loyal, *Notable South Australians*, pp. 131–3; Cumpston, *Smallpox*, p. 72.
- ⁴⁸ R. Gething to Chairman Vaccine Board, 30 Jan. 1867; Dr Gething, Report of trip to Venus and Streaky Bays, 5 Feb., Letter 30, 1867, SAA, GRG 24/6.
- ⁴⁹ *TPRSSA*, Abstract of Proceedings, 13 June 1882, pp. 112–13.
- ⁵⁰ Curr, *The Australian Race*, vol. 2, pp. 3–7; *TPRSSA*, Abstract of Proceedings, 5 July 1882, p. 113, 13 June 1882, p. 112.
- ⁵¹ *Pastoral Pioneers*, vol. 2, pp. 152–3; H. C. Hawson, *Argus*, 2 Feb. 1877.

- ⁵² Stirling, 'Anthropology', part 4, *Report on the Work of the Horn Expedition*, p. 128.
- ⁵³ Cumpston, *The History of Smallpox*, pp. 75–6, 72.
- ⁵⁴ Radcliffe-Brown, 'Former Numbers', pp. 689–90, 693–5; White and Mulvaney, 'How many people?' pp. 115–17.
- ⁵⁵ Ellen Kettle, 'Tuberculosis in the Northern Territory', pp. 71ff, in Proust (ed.), *History of Tuberculosis in Australia*.
- ⁵⁶ Mulvaney, *Encounters in Place*, pp. 171–5; G. Reid, *A Picnic with the Natives*, pp. 85–101.

9 Western Australia 1862–1870

- ¹ 'An Old Australian', *Argus*, 5 Feb. 1877. This is the only reference known to the writer that Aboriginal smallpox in Western Australia before 1866. It would not have persisted long enough in crews of European vessels that reached the southern ocean in the eighteenth century to be transmitted to Aborigines.
- ² W. Milligan, 'Abstract of a Report', p. xi; Storman, *The Salvado Memoirs*, p. 173.
- ³ For estimates of the European population, see Vamplew (ed.), *Australian Historical Statistics*, p. 26.
- ⁴ H. Burnham Bryan, Surgeon Busselton, to Guardian of Natives, Perth, 17 Feb. 1854; N. Cowan, Guardian York, to Col. Sec., 7 Feb. 1854; C. Symmons, Guardian Perth, to Col. Sec., 13 Sept. 1854, WAA 282.
- ⁵ J. Ferguson, Col. Surgeon, to Col. Sec., 7 Mar. 1858, WAA 410; J. Ferguson, Perth, to Col. Sec., 18 Feb. 1861; A. Waylen, Surgeon, Guildford, to Col. Sec., 25 Mar. 1861, WAA 479.
- ⁶ William [S], MD to Col. Sec., 22 Feb., 2 and 6 Mar. 1860, WAA 455.
- ⁷ Cumpston, *The History of Measles in Australia, 1788–1925*, pp. 200–1, 248.
- ⁸ A. Trimmer, Guardian Albany, to Col. Sec., 1 Aug. 1860, 4 Dec. 1860, WAA 445; N. Cowan, Guardian York, to Col. Sec., 14 Jan. 1861, WAA 470.
- ⁹ N. Cowan, Guardian York, to Col. Sec., 14 and 28 Jan. 1861, 7 Oct. 1861, WAA 470.
- ¹⁰ Millett, *An Australian Parsonage*, pp. 257–8.
- ¹¹ Cliff and Peter Haggett, *The Spread of Measles in Fiji and the Pacific*, pp. 54–6.
- ¹² Mulvaney, *The Prehistory of Australia*, p. 21.
- ¹³ Foelsche, 'Notes', TPRS SA, pp. 7–9.
- ¹⁴ Kimberly, *History of West Australia*, p. 208; R. Sholl to Col. Sec, 1865, Camden Harbour, WAA 552; Roebuck Bay, WAA 553; 'Robert John Sholl', in *ADB*, vol. 6, pp. 121–2.
- ¹⁵ Richardson, *Early Memories*, p. 63; 'Alexander Robert Richardson', in *ADB*, vol. 11, p. 377.
- ¹⁶ Curr, *The Australian Race*, vol. 1, pp. 370–1.
- ¹⁷ Bates, *The Native Tribes*, pp. 5, 295–7, 'The Kimberley Nation', pp. 59–61.
- ¹⁸ Journals of Trevarton C. Sholl, 1865–66, Friday 10 Aug., in WAA; Curr, *The Australian Race*, vol. 1, pp. 220, 287–8, 290.
- ¹⁹ C. H. Elliott, District Vaccinator, Champion Bay, to Col. Sec., 31 Mar. 1864, WAA 451; C. H. Elliott, Medical Officer, Victoria District, per A. Durlacher to Col. Sec., 21 May 1866; A. Durlacher, Geraldton, to Col. Sec., 21 May, 1866, WAA 580.
- ²⁰ Curr, *The Australian Race*, vol. 1, p. 296.
- ²¹ Richardson, *Early Memories*, p. 64.
- ²² Radcliffe-Brown, 'Three Tribes', pp. 1, 146, 170–1; 'Former Numbers', p. 689.
- ²³ *Australian Encyclopaedia*, 4th edition, vol. 9, 1983, p. 86.

- ²⁴ Resident Roebourne to Col. Sec., 1866, in Hasluck, *Black Australians*, p. 104.
- ²⁵ Journals of Trevarton C. Sholl, 1865–66, Thursday 9 Aug.; Douglas Sturkey, ‘Emma Mary Withnell’, in *ADB*, vol. 6, pp. 430–1; *Bicentennial Dictionary*, vol. 4, p. 3362; R. Sholl to Col. Sec., 22 and 29 Sept. 1866, WAA 582; Hasluck, *Black Australians*, p. 104; Cumpston, *The History of Smallpox*, p. 66.
- ²⁶ *Bicentennial Dictionary*, vol. 3, p. 2465; ‘William Burges’, in *ADB*, vol. 1, p. 180. Mary Albertus Bain referred to James Perks in *Ancient Landmarks*, pp. 202–3; Curr referred to John Perks in *The Australian Race*, vol. 1, pp. 368–71. Using vocabulary from his informants, Curr said that *moolya errill-ya-rill-ya* meant stone (in the) nose, Curr, *The Australian Race*, vol. 3, p. 97, note.
- ²⁷ *Australian Encyclopaedia*, vol. 4, 1983, p. 281; A. Durlacher, Geraldton, to Col. Sec., 25 Jan. 1869, with enclosure, WAA 645; C. H. Elliott, Geraldton, to Col. Sec., 13 Mar. 1869, WAA 638; *Western Australian Almanack and Directory 1870*, pp. 38–9. Perks referred Curr to the 1870 *Almanack* because it reported the Geraldton outbreak, the first one he saw.
- ²⁸ Jas. Watson to Corporal Lucas, 10 Mar. 1869, WAA 638.
- ²⁹ A. Durlacher and Maitland Brown, Greenough, to Col. Sec., 24 Mar. 1869, WAA 638.
- ³⁰ P. C. Watson, Upper Irwin Station, 27 Mar.–5 Apr. 1869. The report is hard to read. A. Durlacher, Geraldton, to Col. Sec., 7 Apr. 1869, with enclosures, WAA 638.
- ³¹ Maitland Brown, Perth, to Col. Sec., 8 Apr. 1869, WAA 638.
- ³² A. Durlacher, Geraldton, to Col. Sec., 13 Apr. 1869; Maitland Brown, Perth, to Col. Sec., 17 Apr. 1869; C. H. Elliott, Geraldton, to Col. Sec., 19 May 1869, WAA 638.
- ³³ Curr, *The Australian Race*, vol. 1, p. 380. Newcastle is not used as a place name in Western Australia now. The Natingero were over 300 kilometres north-east of Newcastle, perhaps near Dongara.
- ³⁴ ‘Edric Frederick, Baron Gifford of St. Leonards, V. C., 1849–1911’, in George Cokayne, *The Complete Peerage*, London, 1910–40, vol. 5, p. 655.
- ³⁵ Curr, *The Australian Race*, vol. 1, pp. 375–6.
- ³⁶ *Ibid.*, vol. 1, pp. 219–20, 368–71.
- ³⁷ Australia 1: 250 000. Printed 1969. Murgoo; Richardson, *Early Memories*, pp. 53–4.
- ³⁸ Curr, *The Australian Race*, vol. 1, pp. 220, 342, 338, 402, 395.
- ³⁹ Kimberly, *History of Western Australia*, p. 210; T. Hooley to R. Sholl, 4 Sept. 1866; R. Sholl, Port Walcott, to Col. Sec., 10 Sept. 1866, WAA 582.
- ⁴⁰ Curr, *The Australian Race*, vol. 1, p. 302; Radcliffe-Brown, ‘Former Numbers’, p. 689.
- ⁴¹ Cumpston, *Smallpox*, pp. 65–6; Curr, *The Australian Race*, vol. 1, p. 306.
- ⁴² *Inquirer and Commercial News*, 15 June 1870; *Western Australian Almanack and Directory 1871*, Mar. 1870, p. 32; *Australian Medical Gazette*, 1870, p. 81 in Cumpston, *Smallpox*, p. 78.
- ⁴³ R. Sholl, Roebourne, to Col. Sec., 1 and 27 Jan. 1870, WAA 665.
- ⁴⁴ Roebourne, Nickol Bay, *Inquirer and Commercial News*, 16 Feb. and 16 Mar. 1870.
- ⁴⁵ R. Sholl to Col. Sec., 8 Apr. 1870, WAA 665.
- ⁴⁶ Hopkins, *Princes and Peasants*, pp. 270–4.
- ⁴⁷ Diamond, *Guns, Germs and Steel*, p. 320.
- ⁴⁸ Kimberly, *History of Western Australia*, p. 206; Hasluck, *Black Australians*, pp. 188–9; Richardson, *Early Memories*, pp. 56–8, 64–6.
- ⁴⁹ R. Sholl, Roebourne, to Col. Sec., 13 Mar. 1869, WAA 646 (Malays) imported: 30 Apr., 17 May, 21 May, 29 May and 24 Aug. 1872, WAA 714.
- ⁵⁰ Biskup, *Not Slaves Not Citizens*, pp. 30, 19.
- ⁵¹ Cumpston, *A History of Measles*, p. 248; Hasluck, *Black Australians*, pp. 104–6.

10 The Diseases that Killed

¹ Black, 'An Explanation of High Death Rates', pp. 292–307.

² Cleland, 'Ecology, Environment and Diseases', pp. 155–8.

³ Diamond, *Guns, Germs and Steel*, p. 320.

⁴ Butlin, *Our Original Aggression*, pp. 19–37; Bultin, *Economics and the Dreamtime*, p. 120.

⁵ Fenner, 'Sociocultural changes and infectious diseases', p. 22.

⁶ Butlin, *Economics and the Dreamtime*, p. 119.

⁷ Fenner *et al*, *Smallpox and its Eradication*, pp. 57, 657, 1117.

⁸ In the 1991, Aboriginal populations in South Australia and Victoria were each less than 20 000, and were the smallest on the mainland.

⁹ McCaa, 'Smallpox and Demographic Catastrophe in Mexico', pp. 397–431.

¹⁰ The Australian Immunisation Procedures Handbook, 1994. *National Graduate*, The ANU Society, Autumn 2000, 'Indigenous health graduates in demand', pp. 8–9.

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