ABORIGINAL HEALTH

Peter M. Moodie

ABORIGINES IN AUSTRALIAN SOCIETY

From this study of Aboriginal health a depressing picture emerges. The death rate for Aborigines from almost all causes, and the incidence of communicable disease, is much higher than for white Australians. Much of Aboriginal ill-health is directly associated with poverty and poor living conditions—and therefore hygiene—and with malnutrition, particularly among the children. On health grounds alone, the Aborigines are shown to be severely handicapped in almost every aspect relative to white Australians, and to other indigenous minorities such as the Maoris and the American Indians.

Though it is recognised that an Aboriginal 'health problem' exists and a good deal of factual material has been collected, no systematic survey of the available data has ever been made. If the problem is to be solved, the available knowledge must be collated and interrelated. That is the aim of this study. The book covers a wide range of diseases and patterns and causes of death among Aborigines and part-Aborigines throughout Australia; it shows many gaps in knowledge, in particular the lack of ordered statistics with which those concerned with Aboriginal health must contend.

The problem of Aboriginal health will not be solved quickly, but Dr Moodie's work, and the suggestions he makes, provide a basis on which future policy may be developed.

This book is essential reading for all concerned with the quality of life in Australia and with the plight of the Aborigines.

ABORIGINAL HEALTH

Aborigines in Australian Society 9

A series sponsored by
The Academy of the Social Sciences in Australia

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Peter M. Moodie

AUSTRALIAN NATIONAL UNIVERSITY PRESS CANBERRA 1973

First published in Australia 1973

Overseas

United Kingdom, Europe, Middle East, Africa and Caribbean:
Angus and Robertson (U.K.) Ltd, London
North, South, and Central America: International Scholarly
Book Services, Inc., Portland, Oregon
Southeast Asia: Angus and Robertson (S.E. Asia)
Pty Ltd, Singapore

Japan: United Publishers Services Ltd, Tokyo

Registered in Australia for transmission
by post as a book.
Printed in Australia for the
Australian National University Press, Canberra
Library of Congress Catalog card no 73-76201
ISBN 0 7081 0094 5

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NOTE ON THE SERIES

The Social Science Research Council of Australia (now The Academy of the Social Sciences in Australia), which was founded in its present form in 1952, is the national organisation of social scientists. Some of its major functions are:

to encourage the advancement of the social sciences in Australia;
to act as a co-ordinating group for the promotion of research and teaching in the social sciences;
to foster research and to subsidise the publication of studies in the social sciences.

To these ends the Council has sponsored a number of major research projects. The first related to the role of women in public and professional life in Australia and was carried out by Mr Norman MacKenzie. His report, together with the associated study of the legal status of women in Australia by Dr Enid Campbell, was published in 1962 in a book, *Women in Australia* (F.W. Cheshire Pty Ltd, Melbourne).

The second major project, carried out by a group of economists, was concerned with the Australian taxation structure and under the authorship of R.I. Downing, H.W. Arndt, A.H. Boxer, and R.L. Mathews, the results were published in 1964 in *Taxation in Australia: Agenda for Reform* (Melbourne University Press, Melbourne).

In 1963 the Council approved its third and most ambitious major project, Aborigines in Australian Society, with the broad objectives of:

elucidating the problems arising from contacts between Aborigines and non-Aborigines and formulating policy implications from these;

drawing together existing knowledge in various parts of Australia and undertaking such further original research as can be carried out over a period of three years.

In May 1964, Mr C.D. Rowley, formerly Principal of the Australian School of Pacific Administration in Sydney, was appointed Director of the Project, to work under the general guidance of a Project Committee appointed by the Council. The volumes now being published represent a major research enterprise in which many social scientists collaborated over the length and breadth of Australia.

However, the whole enterprise depended in very large measure on the magnificent support received, from the outset, from the Myer Foundation of Australia and the Sidney Myer Charity Trust. The Council wishes to acknowledge its gratitude for their generosity.

W.D. Borrie
Canberra

PREFACE

> Any man's *death* diminishes *me*, because I am involved in *Mankinde*; And therefore never send to know for whom the *bell* tolls; it tolls for thee.
>
> John Donne, *Devotions*

The principal aim of this study has been to identify, classify, evaluate, and interrelate current Aboriginal 'health problems'. In the process, a picture has been built up of a minority group in Australia which is severely disadvantaged in most aspects of health relative to non-Aboriginal Australians, and relative to other indigenous minorities such as the American Indians or the Maoris of New Zealand.

It is all too easy to see these disadvantages, in retrospect, as the outcome of mismanagement of the Aboriginal situation by governments, government departments and other authorities, past and present. But, if the situation was mismanaged, is this not apparent only in the light of subsequent events and new knowledge? Is it possible to know with certainty what *must* be done in the future in the field of health action? Would all the health disadvantages of Aborigines disappear if their socio-economic environment altered dramatically?

If these questions are to be answered, a thorough knowledge of the present Aboriginal health situation is the first step, followed by an analysis of the mechanisms producing or controlling the situation, and a clear idea of the rationality, practicability and effects of intervention—not only now, but as the situation changes.

The present study has attempted to go a little beyond the first step. In the attempt, it was immediately apparent that there were large and important gaps in the available data with respect to current Aboriginal health status, and particularly with respect to health trends, to 'health behaviour', to attitudes to illness and health services, and to the modern Aboriginal ecosystem in general.

The most valid criticism of past activities in the field of Aboriginal health, in the writer's view, relates to the failure to fill these gaps in

knowledge and understanding, resulting in a very conservative and ethnocentric approach to the Aboriginal 'health problem'. Lacking concrete data and a proper perspective, it had seemed sufficient to offer Aborigines a health system which, at most, was a slightly modified form of the system offered to Australians in general, according to priorities determined by and for non-Aboriginal observers, and implying that Aborigines have—or should have—the same 'health values' as white Australians. Whether by design or in simple response to demand, the Australian health system is an extension of the white Australian social and economic system—as it should and must be. If the social and economic systems of Aborigines and part-Aborigines differ from those of white Australians, it is hardly surprising that the best-intentioned efforts to ameliorate Aboriginal ill-health with 'traditional' solutions have often failed, and that the situation has now become identified as 'the Aboriginal health crisis'.

The lack of data on Aboriginal health indices and their trends is not always because the information has not been gathered. Often it appears that important data have been collected but have lain untouched, gathering dust in government offices and archives, never collated and analysed. In other cases, detailed but otherwise impersonal reports have been written, but have remained confidential *and thus forgotten* in the service that originated them. Critical public self-evaluation or self-assessment of services by services—essential in supporting or justifying courses of action taken across a culture-gap—has rarely occurred in the past.

In the last few years the rate of acquisition of Aboriginal health data has increased noticeably, but relatively little of this new information has been published by the services or departments directly involved with Aboriginal health. Most recent studies, and even the basic descriptive statistics on Aboriginal health, have emanated from universities, research institutes, or individuals within or outside the public service publishing their findings in medical or other scientific journals. Such independent or semi-independent inquiries and assessments, valuable as they are for their independence, have been severely handicapped where access to raw data has been denied or restricted for accredited researchers—an event which unhappily still occurs.

What fresh information does become available from time to time (freshly available, but often 'old hat' to government departments) may be seen as reflecting adversely on past and present policies and practices, or it may be used as political ammunition. At the same time, its 'freshness' precludes the responsible authority being able to demonstrate their

positive achievements, and leaves politicians and administrators alike vulnerable to ill-informed attack.

These may seem highly contentious statements with which to introduce a community health study. But health is an important political issue: if 'politics' were to be kept out of 'health', the latter could not be discussed openly at all. If health was not a political issue, very little would be done about it at the community level and 'public health' would not be an effective force.

Progress in Aboriginal health can only be *demonstrated* relative to a base-line. If there is no base-line, all new information will appear to reflect deficiencies in Aboriginal health policy. It could also be argued that, in the absence of guidance from the observation of trends, such an appearance may not be deceptive, and changes may not always be for the better.

Sydney, P.M.M.
April 1972

ACKNOWLEDGMENTS

The author is greatly indebted to a number of individuals and institutions for their assistance in pursuing this study. In particular, I would like to thank Professors R.H. Black and C.D. Rowley for their initial encouragement, repeated support and final patience over a period of nearly seven years; Professor F. Lancaster Jones for supplying data on Northern Territory Aboriginal childhood deaths not published in his own studies; Professor Walsh McDermott for supplying a draft publication on the Cornell University-Navajo Many Farms Project; Professor J.E. Cawte for permission to quote a long passage from one of his publications on transcultural psychiatry; the Commonwealth Bureau of Census and Statistics, through their offices in Canberra and the other capital cities, for assistance with Aboriginal mortality data; the Northern Territory Medical Service for data on Aboriginal births and infant deaths in the Territory; the New South Wales Registrar General's Department for raw data on part-Aboriginal mortality in New South Wales; the New South Wales Department of Health's head office and Tuberculosis Division office for access to departmental records relating to Aboriginal health surveys in New South Wales, including data on helminth surveys, anaemia and tuberculin surveys collected by Dr T.L. Dunn and Dr M. Andrew; the Bureau of Indian Health, United States Department of Health, Education and Welfare, for graciously making available unpublished data on American Indian mortality; and the New Zealand Department of Health for information on the compilation of Maori health statistics. I would like to thank Professor Rowley again for permission to extract data from his survey in rural New South Wales and the Eyre Peninsula of South Australia, and for the assistance he provided as Director of the Aborigines Project, together with that of Dr Fay Gale, to obtain data on Aboriginal mortality from two South Australian Aboriginal Reserves extracted from records in the South Australian Archives.

I also express my appreciation to Mrs L. Presdee for her care in

preparing the typescript, and to Professor R.K. Macpherson, Professor W.D. Borrie, Professor H. Burton and the editorial staff of the A.N.U. Press for their considerable help in seeing this study into print.

Professional colleagues within and outside the School of Public Health and Tropical Medicine, Aboriginal acquaintances, and many other persons have also contributed in a way which it is impossible to acknowledge individually. Their contributions have been ideas, opinions, stories, and perspectives absorbed unknowingly, digested and synthesised subconsciously, and resurrected in the writing. To those who have contributed in this way I would like to express my thanks—without apology, for they no doubt acquired their views in exactly the same way. It would seem that, particularly in this sense, no man is an island and few experiences are truly personal.

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INTRODUCTION

When the first white settlement in Australia was established in the Sydney area in 1788, it has been estimated that there were between 150,000 and 300,000 Aborigines inhabiting the continent. Records of first contact between settlers and explorers and Aboriginal groups not infrequently state that the Aborigines appeared in good health and free of disease; there is certainly no indication in these early records that Aboriginal populations were disease-ridden or were handicapped by widespread ill-health prior to contact.

As white settlement proceeded, however, the reports changed and the opposite picture was presented—that of an Aboriginal population riddled with disease and being decimated by it. It was believed by many that the diseases noted—mostly communicable infections—had been introduced by the settlers and flourished among the Aborigines because of racial susceptibility (i.e., an inherited lack of resistance). Others recognised that changes in Aboriginal conditions and behaviour consequent to settlement, and the partial adoption of white customs, were also likely explanations for what was occurring.

Today it is estimated that there are about 40,000 'full-blood'[1] Aborigines and about 80,000 identifiable part-Aborigines in Australia. Although the early reduction in numbers has been attributed in part to the harassment and killing of tribal Aborigines, and their dispossession from their tribal lands, there is no doubt that the major factor has been the effects of disease through mortality and reduced fertility—'disease' here including

[1] 'Full-blood' in the sense of 'of more than 50 per cent Aboriginal ancestry'.

psychological disturbances which have been thought to have contributed to the low fertility of full-blood populations.

In the last few decades, the number of Aborigines has increased rapidly, particularly among the part-Aboriginal component, and this appears to reflect an improvement in health (or at least a reduction in mortality and an increase in fertility). Nevertheless, the available data suggest the persistence of a wide gap in health status and expectations between Aborigines and white Australians, and 'Aboriginal health' remains a matter of major concern to governments and individuals interested in Aboriginal welfare. To what extent it is a matter of equal concern to the Aboriginal population is difficult to judge.

Most of the basic Aboriginal health and vital statistics available for the last decade closely parallel those for the white Australian population in earlier times. The gap appears to be about seventy years for Aborigines in northern Australia, and somewhat less among part-Aborigines not yet socially and economically assimilated in southern Australia. Present-day Aboriginal disease and mortality patterns would have been regarded as 'average' for Australia as a whole at the turn of the century, although in respect to certain communicable diseases amenable to control by immunisation such as diphtheria, whooping cough, and poliomyelitis, they would have been above average.

The aim of this study is to examine present Aboriginal health status and the factors that determine it. The picture that emerges will be largely a base-line from which to observe future progress, for unfortunately the data are insufficient or inadequate to construct a base-line for the past and examine subsequent trends up to the present. It might be both interesting and helpful to be able to look at Aboriginal health trends in terms of the apparent time-lag referred to above and see whether the gap is closing or widening. However, such an approach could only be used for death rates by age; death rates and disease rates by cause change not only with real changes in prevalence but also with changes in diagnostic skill, nomenclature, the discovery of 'new' disease entities, and medical fashions of the moment in respect to certification of death.

Deaths, as plain facts of occurrence which are easily recorded and not open to reinterpretation with the passage of time, are relied upon heavily in studies of the health of populations. The ages at which death occurs, together with the causes of death, provide even more valuable information, particularly for a study covering a point in time or a short period of years. It may well be asked what relevance mortality studies have to the health of the living—which is our primary concern—and to answer this

it is necessary to consider concepts and definitions of health and ways in which health can be measured. These are very difficult subjects to cover briefly, but must be touched on here because of their relevance to the cross-cultural evaluation of the health of Aborigines.

The white Australian population, and the highly developed technological societies in general, cannot be certain that their own health concepts and priorities are not leading them into ecological and economic peril. It seems to the writer quite impossible to continue with the view that health standards and goals which are good enough for us should be accepted by—or imposed upon—others whose life-style and attitudes are different from ours. The impact of death and disease, and of measures taken to alter or reduce this impact, must be critically examined from the viewpoint of the society affected—so far as this is possible—and not just from the viewpoint of the society which provides the money, manpower, or materials needed to bring about a change. This point is worth labouring a little for there are many people in the field of public health as well as outside it who either do not accept it, or lose sight of it when faced with a real problem in Aboriginal health. The outcome is further complicated by the fact that Aboriginal health problems cannot be divorced from the health, social, and economic problems of Australia as a whole.

1 CONCEPTS AND DEFINITIONS OF HEALTH

It would seem that an individual's concept of his own health, whether formulated or not, is based on a subjective feeling of well-being. He evaluates his own health relative to his past experiences, to how he thinks other people feel, to the norms for health in the society in which he lives, and to his ability to perform the roles he accepts. The presence of an illness or abnormality (apparent to others) will not affect his feeling of well-being unless he is aware of his abnormal state, or (and this amounts to the same thing) is frustrated in following a customary or newly-desired pattern of behaviour. Customary behaviour can be modified in the face of persistent physical, mental, or social handicaps to restore a feeling of well-being, so that it is not uncommon to find individuals with major health handicaps who feel in excellent health and would say so if asked. According to their personal concept of health, they would be quite right.

The subjectivity and relativity of this personal concept of health may make it appear irrelevant in the evaluation of the health of a population (or an individual), but this is not so; it motivates the individual to seek out medical advice, and thus conditions whether he will appear in the morbidity statistics of hospitals, clinics, and medical practices. Furthermore, his subjective feelings of abnormality become his medical symptoms, and in a medical examination these influence the areas in which he is most intensively investigated and the ultimate diagnosis attached to his state of ill-health (which also appears in various statistics).

The subjective and relative concepts of health and disease held by people very largely determine the range of data available for the classification of disease, and also determine priorities for research, medical

treatment, and public health action; that is, they underlie the supposedly objective evaluation of health by the trained observer. The physician who undertakes an 'objective' random or total survey of the health of a community can look for only those diseases he knows as entities (which he has been trained to accept). How often does he realise that these diseases, and the technology for detecting them, are ultimately defined and given status by the communities among which his teachers worked?

It is not surprising, therefore, that non-Western communities are nonplussed by many aspects of Western medicine, and highly selective (to the Western-trained observer) in the benefits they seek from it. It also seems likely that there are still many perfectly legitimate disease entities which are *common* in non-Western societies but remain outside the ken of the Western-trained physician because they do not occur on his check-list of disease entities—the International Classification of Diseases.

The foregoing suggests that it may be impossible to arrive at a valid evaluation of 'total health' for an individual or a population. But this is not just because standards of health must have components of subjectivity and relativity, culturally conditioned—the basic problem is that the field of 'health' has no clearly defined edges. The edges are not too indistinct when the field is restricted to 'physical health', but become more and more vague with the addition of 'mental health' and 'social health'. The well-known United Nations definition of health, incorporated into the Preamble to the Constitution of the World Health Organisation in 1946, reads 'Health is a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity'. This definition has been criticised for its use of words without self-evident meanings and for being unworkable as a basis for the measurement of health, although its value as a public relations slogan is admitted (Wylie, 1970:100).

It is evident that with such an open-ended definition it is not only impossible with current knowledge to rate accurately an individual's total health at a point in time (let alone over a period); it is also impossible to compare two or more individuals, or populations, with less than perfect health but suffering from different disabilities.

A definition which has more appeal is that of Herbert Spencer—'Health is the perfect adjustment of an organism to its environment' (quoted by Wylie, 1970:101), or, as modified by Wylie, 'the perfect *continuing* adjustment . . .' (1970:103). Again, however, 'perfect adjustment' defies satisfactory definition, and comparisons between individuals or groups are equally difficult.

Both types of definition, in spite of their commendable attempt to

embody a positive concept of health, ultimately rely on the ability of the professionally-trained observer to detect malfunction or maladjustment—that is, on his ability to detect disease. Wylie was not concerned with this, as he believes that the measurement of the absence of disease is a valid indication of the presence of health (1970:103). It is only possible to accept this view, however, where the measurements are carried out within a homogeneous society, by persons trained in the same society.

A different approach is that of Sullivan (1966:6), who says, 'Health, defined without reference to a specific situation or purpose of measurement, may be merely a verbal artifact'. The writer accepts this view by a competent authority with gratitude, since it is difficult to find any other justification in the literature for the methods used in this study—the well tried but not necessarily true evaluation of health in a piecemeal fashion through the study of multiple indices of health. Each index—such as the infant mortality rate or an age-specific disease rate—reflects only a facet of the overall health picture, but it is relatively easy to specify both the situation and the purpose of measurement.

Although these indices of health tend to compartmentalise health problems, they should not be permitted to compartmentalise health action—the solution to Aboriginal health problems will always remain as complex as the multiple interrelated factors which produce them, and their multiple, interrelated consequences. If the United Nations definition of health is accepted as the goal of health action, a simple answer to a health problem should be viewed with the utmost suspicion since in the long term it will almost certainly prove to be the wrong answer. There are all too many examples around the world of the catastrophic effects of the 'simple' answers given by medical science—attacks on specific diseases and the population explosions which follow, for instance.

Another possible objection to the evaluation of health by way of health indices—a quantitative approach—is that the quality of health may be overlooked, although this is an elusive concept to deal with when considering a population. For the individual Aboriginal who is found to be suffering from leprosy, for instance, and was probably unaware of his illness, the quality of his sense of well-being will be dramatically influenced by his diagnosis, isolation, or transfer to distant parts. Many people object to statistics on the grounds that they dehumanise what they are describing, and this is so. The quality of health may be safeguarded to some extent, however, by not forgetting that every contributor to a statistical figure is an individual not only as far as his disease is concerned but also with respect to treatment he must

undergo if the statistical picture is to be improved. The ability to translate individuals into figures would seem to require an accompanying ability to translate figures into individuals. In the field of health action, personal acquaintance with the target population is essential for those who make the decisions from statistical summaries.

The views expressed above are not radical, and are widely held, although they are rarely given much prominence in studies of the health of communities. The purpose of introducing the reader to them at this early stage, before looking at the statistical and other data on Aboriginal health, is to provide some degree of culturally-unconditioned perspective —particularly for those who may not have had occasion to consider 'health' in a cross-cultural setting.

There remains the practical problem of the measurement of health.

THE MEASUREMENT OF HEALTH

Quantitative measurements of the health of populations are necessary to health evaluation and health planning. In the first place they allow various aspects of the health of a community to be compared between populations and the trends followed over a period within one or more populations. In the second place, health indices not only determine priorities for health action, but the feed-back from continuous monitoring of the indices is used to evaluate the success of a program. The quantity and quality of data retrieved are also indicators of the efficiency of a health service, particularly at the point at which the health service is in direct contact with the community.

Despite considerable effort on the part of the World Health Organisation and others, no satisfactory single index of health has yet been devised. The traditional indices fall into several categories.

MORTALITY STATISTICS

These include death rates by age-group, death rates by cause of death and, derived from age-specific mortality, the expectation of life at various ages. The data used for the calculation of such indices come from information provided in death certificates, while the denominator for the calculation of rates—the population at risk—is based on population censuses, either national or local, for the mid-point of the period over which the events occurred. Deaths occurring in the first year of life are normally based on the number of live births occurring in the period considered rather than on the population of the relevant age-group.

Where the population at risk is not known with any accuracy, useful information may be obtained on the pattern of mortality by examining the proportions of deaths contributed by various causes or in various age-groups. Such figures must be interpreted with care when drawing comparisons since they do not indicate absolute risks of death within the categories chosen or risks relative to those for other populations.

Mortality statistics are much used as indices of health because they refer to events which are both final and incontrovertible, and they can pinpoint vulnerable age-groups with respect to serious, life-threatening diseases. In the bio-economic sense, they reveal a great deal about the wastage or conservation of human resources. Depending on the individual's state of health prior to death, his age, and the socio-economic system in which he lives, death may range in effect from loss of economic potential or production for the community through to being an economic benefit. The greater the individual's potential or actual contribution to society and his family, the greater the significance of his death, so that it is in childhood and early adult life that high mortality has caused the greatest concern—at least in Western societies where a great deal of the community's resources are invested in the growing child. There would seem to be more than humanitarian reasons why the same societies have invested enormous technical and financial resources in combating the health problems of infancy and childhood, with considerable success. Is it possible that in the more traditional Aboriginal communities, where only personal resources are invested in the growing child by his parents and relatives, that the death of a child is a personal but not so much a community loss when compared with the death of an adult?

In terms of health, mortality statistics merely describe the top of an iceberg—the minority of serious illnesses that can cause death. As Sullivan (1966:101) has pointed out in an analysis of the problems of developing an index of health:

> Indexes based on mortality . . . have traditionally been used as measures of the level of health. . . . The assumption is often made that changing mortality reflects changes in other aspects of health as well. Recently, however, crude and age-adjusted death rates for the U.S. population have shown little change after a long period of decline over the years 1900–1954 . . . Stability of the death rate would not imply no change in health status. It would merely emphasise a difficulty inherent in the use of mortality statistics as measures of health. They tell little about the living . . . .

However, the Aboriginal population has by no means passed through this 'long period of decline' in mortality, and their mortality statistics

still signify a great deal because of their variation from current Australian norms. The implication of these variations, in relation to causes and effects in the Aboriginal population, and in relation to the delivery of medical and health services to Aborigines, will be discussed later.

MORBIDITY STATISTICS

Statistics for the prevalence (number of cases) or incidence (rate of occurrence of new cases) of disease must also be related to the population at risk for their full significance to be appreciated. An illness is an infrequently terminal event, and because of this it is possible to survey a population on a total or a statistically random basis, count those who are free of each disease sought as well as those who are in ill-health, and derive from these data a valid picture of disease *prevalence*, and the absolute as well as relative importance of disease entities. The survey generates its own census of the population at risk, which is not the case in mortality studies. Studies of disease *incidence* require prolonged survey of a circumscribed population which can be re-examined at appropriate intervals, and because of this factor also generate their own census. There are relatively few diseases which, like a death, are almost invariably noted and recorded without the need for a physical survey.

Because many illnesses are not self-evident, or if self-evident do not invariably lead to treatment and their appearance in collatable records, surveying is essential to the analysis of prevalence and incidence. Self-selected groups of people in ill-health, such as those who attend at hospitals and clinics, may provide little information about the prevalence or incidence of diseases in the total community. Partly compensating for this, such figures tell us something about the behaviour of the community with respect to illness, and with respect to the services available to them.

In communities with a high appreciation of good health and of the available services very few serious or life-threatening diseases may escape the statistical net—provided that all avenues for treatment contribute to the statistical coverage. The corollary to this is that where health and medical services are limited in scope or acceptance, or where good health is not appreciated according to the values of those who categorise their illnesses, hospital and outpatient statistics tell us little more than the extent of services provided. That is, they may be a very poor indication of services needed by the community.

A problem more evident with morbidity than mortality statistics is that of comparability. Even where careful surveys are carried out in

different communities or different areas, by different surveyors, comparability of results requires that there be an overall standardisation of methods and scope. This degree of standardisation is almost impossible to achieve without collective training of those engaged in the survey task. In the case of the Australian Aborigines, health surveys have not only been usually restricted to within State boundaries, but have usually been carried out in smaller areas, on an *ad hoc* basis, with individualistic approaches by the surveyors. Health surveys of non-Aboriginal populations have been based on similar lines. The unfortunate result for the health researcher is that even where he has carried out a carefully designed health survey he has great difficulty in finding Australian data—Aboriginal or otherwise—with which to compare his findings and draw conclusions. It is usually not too difficult to find the necessary comparison data for overseas populations, but in most instances the comparison suffers from a complex of unknown or poorly-understood associated variables in the exotic population.

For certain selected communicable diseases, such as poliomyelitis, infantile diarrhoea, tuberculosis, and venereal disease, the various State Health Acts require that notifications of occurrence be supplied to local or other authorities, and it might be supposed that the data obtained in this way give a picture of the incidence of notifiable diseases. However, the supposition presupposes that, firstly, all cases of notifiable disease are reported to a doctor and secondly, that all doctors notify all cases reported to them. Neither presupposition is true, and there seems to be a considerable variation in the thoroughness of notification of different diseases—infantile diarrhoea and venereal disease (for different reasons) appearing to be particularly liable to under-notification.

With morbidity statistics, therefore, the conclusion is that the figures must be analysed with great care from data that are collected with great care. The comparison of data from different sources in different areas, and by different investigators, is especially hazardous. Morbidity statistics, to some extent, reflect the behaviour of the community and the behaviour of the medical profession as well as the behaviour of the diseases themselves; the three influences cannot be separated except by long-term surveys in depth.

OTHER HEALTH STATISTICS

Although not ordinarily considered measurements of health, many statistical indices available for populations (or potentially available) play an important part in the evaluation of health. Apart from helping in the

evaluation of health, many such indices are of course vital to health planning and the evaluation of health services.

It is appropriate at this point to consider the part that curative medicine plays in determining health status. Although preventive public health activities, environmental improvements, and many intentional or incidental by-products of a rise in the general standard of living operate to reduce the incidence—and therefore the prevalence—of a wide range of diseases, the disturbed equilibrium often encourages new diseases to replace them. Curative medicine, on the other hand, is entirely directed towards reducing the prevalence of existing disease although its primary target is the sick individual; it is only in the case of the communicable diseases that this has an important impact on incidence by removing sources of further infection from the community. Curative medicine, however, cannot claim to be entirely free of adverse effects on health, particularly, in the long view, on community health. The more powerful the weapons and techniques available to curative medicine, the greater the social, economic and therefore health repercussions of multiple cures. There exists today a serious conflict between the clinician's concern for the individual and the human ecologist's concern for the welfare of the community. The public health planner finds himself in the centre of this conflict, with sympathies for both viewpoints. It seems likely that economic realities—the staggering cost of sophisticated medical care predicted in the near future—and environmental realities—overpopulation accompanied by pollution and psychological disturbances—will take the public health planner into the ecologist's camp. If and when the population and health economic problems are solved, and some sort of equilibrium attained, it will be at the expense of many of our concepts and goals of health, but curative medicine will still be directed towards the maintenance of health via the restoration of well-being.

Bearing in mind some of the problems outlined, the health status of the Aboriginal community at a point in time or over a period, as measured by traditional health indices, will reflect the activities of both preventive and curative medicine; hopefully in partnership, although this partnership in Australia appears to be very weak compared with that obtaining in many developing and developed countries. There are some indices which refer to the activities of curative medicine—hospitalisation rates, recovery rates, days of illness, the cost of illness, the cost of medical care, and so on. In the sense that curative medicine is necessary for the maintenance of health—or the reduction of morbidity—such indices are also indices of health. It is apparent, however, that the optimum for many of

them is neither the highest nor the lowest possible value while disease in a community is an accepted fact of life. This applies to the Aboriginal population as much as to any other population, although few figures exist for the former. There is a need for a method of relating such figures to morbidity and mortality, rather than considering them separately. Without some method of weighting, indices of therapeutic activity are almost meaningless in themselves. The *per capita* expenditure on medical treatment for Aborigines may be informative as to the allocation of medical and financial resources, for instance, but will not indicate whether the medical (or health) services are appropriate, effective, well-organised, well-planned, or the opposite.

Another group of health statistics is that dealing with the medical and physical environment—the proportion of the population covered by various categories of service—general practitioner, specialist, nursing, hospital, maternal and infant welfare, dental, health insurance, pharmaceutical; and the proportion of the population having the use of various facilities important to health: adequate sanitation, water supply, housing, garbage disposal, electricity, refrigeration, communications—the list may be extended indefinitely. Although the availability of such services and facilities is no guarantee that they will be used in the most suitable manner, absence or inadequacy of one or more of them implies some form of restriction of the potential for optimum health. They are of obvious relevance to the field of health planning, but are largely controlled by economic factors: that is, they are just as much indices of economic development or community affluence.

Finally, important data relevant to health may be found in the demographic statistics for a population. Apart from the various mortality statistics, which are a major component of standard demographic data, there are health implications in population distribution and density, birth rates, fertility, family size, rates of natural increase, and age distribution. Within the concept of total health, many of these are excellent indicators of ecological stresses and breakdowns. A knowledge and understanding of them is as essential to health planning as it is to economic or social planning.

Before concluding this review of the ways in which health may be evaluated, there is a further method which deserves mention because it neatly summarises the real purpose behind all health indices—the measurement of health in terms of 'functional adequacy'. It is particularly relevant to a study of Aboriginal health because it could avoid many of the pitfalls created by cross-cultural value judgments.

THE MEASUREMENT OF HEALTH AS 'FUNCTIONAL ADEQUACY'

Sullivan (1966:4–16), in a most interesting attempt to define and measure health, has examined proposals by numerous writers. The definition he favours is a functional one, and the measurement one of 'functional adequacy'. Instead of measuring diseases, it measures the effects of disease on the ability of individuals in the population to fulfil a social role appropriate to their age and sex. Measurements of morbidity are replaced by measurements of days lost, at various levels of loss (i.e. 'complete', 'partial', 'nil') due to disabling diseases. It is not even necessary to pinpoint the medical diagnosis, although this would be required in determining 'causes' and in planning remedial actions. The effect on days lost of deaths at various ages can be added, and the net result is what Sullivan calls an 'effective life-years index' for the population considered which combines the consequences of morbidity and mortality.

Needless to say the calculations are not only complex in themselves, but must be based on detailed, complex definitions, and the problems of data-acquisition would be considerable. Sullivan does not go into how the index would be calculated, but he devotes some space to categorisation of levels of disability, and after weighing three types of evidence for disability—clinical, subjective, and behavioural—concludes that the behavioural evidence is most likely to produce valid data.

Many suitable data-acquisition methods are already in use in the United States and elsewhere, but covering limited areas of health evaluation in special fields. There are no international or national standards of 'functional adequacy', but the evidence presented by Sullivan suggests that the functional definition and measurement of health may emerge eventually as a most accurate means of assessing health standards and trends.

For the Australian Aborigines, or for Australia as a whole, such a method is not available at present, but should be borne in mind for future research as to its suitability. The writer is co-operating in a long-term socio-medical study of a part-Aboriginal community from which behavioural and 'days lost' data have been sought, and can attest to the difficulties in obtaining an unbiased and complete picture in this field. A great deal of information is needed for each community studied with respect to roles, behaviour patterns, and social norms before deviations due to ill-health can be identified with certainty. One of the greatest obstacles to the use of the method would appear to be the need for almost continuous monitoring of behaviour.

2 PROBLEMS IN EVALUATING ABORIGINAL HEALTH

We are not looking here at Aboriginal health problems *per se*, but at some of the difficulties which face the investigator in gathering health data for Aborigines, and some of the obstacles to achieving a valid analysis of the data obtained. Both may be viewed as handicaps to health investigation, and many of them are peculiar to the status of Aborigines in Australian society. In the sense that it is dangerous for a community for its health problems to be obscure or misinterpreted, these handicaps form part of the Aboriginal 'health problem'.

Furthermore, like the more obvious health problems such as high infant mortality or malnutrition, many of these handicaps to the evaluation process can be remedied or alleviated. Those which are more or less unavoidable, like the uncertainties in the delimitation of the part-Aboriginal population or the non-existence of a valid Aboriginal 'average' or stereotype, must be taken into account in any analysis of the Aboriginal population.

ABORIGINAL IDENTITY

In accordance with current custom in Aboriginal studies, the writer defines an Aboriginal as 'any person of Aboriginal descent who regards himself, or is regarded by the community in which he lives, as an Aboriginal'. This definition is social rather than biological or racial—it is not related (superficially) to the degree of Aboriginal ancestry. However, it is immediately apparent that the 'degree of Aboriginal ancestry' very much influences the choices open to a part-Aboriginal, particularly

a choice made by the 'community in which he lives'. The virtue of the social definition is that it allows a part-Aboriginal who is socially in a position to opt out of the Aboriginal population to exercise his option. This works both ways, of course, so that a part-Aboriginal who has 'passed' may re-identify himself as Aboriginal if he wishes to do so.

The problem arises from the fact that, no matter what sort of definition of an Aboriginal is used, it is impossible to avoid some untidiness at the borderline between 'Aboriginal' and 'white'. Thus there is no exact figure for the number of Aborigines, nor do trends in the number estimated from censuses necessarily reflect real changes to the degree suggested by the figures. From the viewpoint of health evaluation, the main handicap is that the statistician is unable to define a population base from which indices of health can be calculated. The lack of precision is not confined to the part-Aboriginal component—the open-endedness of any definition of full-blood Aborigines has the same effect, but to a lesser extent.

The problem has to be accepted as unavoidable. Even if some sort of infallible test was devised and applied to the total Australian population, the result would not be relevant to any of the objectives of the identification of Aborigines in the field of health or in other fields except that of population genetics. In any case, it can be shown that the precise identification of the Aboriginal population base is the least of the medical or health statistician's problems, as discussed below.

There is a social consequence of the open-ended definition of Aborigines which may not be so immediately apparent. This is the fixation of the part-Aboriginal stereotype—his characteristics, problems, socio-economic and health status—so that differences from white Australian norms are exaggerated. This comes about through the continuous loss to the white community of those part-Aborigines who, because they have the characteristics and attitudes acceptable to white Australians, are successfully assimilated. When discussing part-Aborigines, we are really discussing the remainder—identified by their differences from the white stereotype. There is thus a subtle circularity in the definition of part-Aborigines for there seems little doubt that in many cases where differences are attributed to part-Aboriginal ancestry, part-Aboriginal ancestry is established—for statistical purposes—because of the differences.

THE IDENTIFICATION OF ABORIGINES IN RECORDS OF EVENTS

The health of the indigenous minorities of New Zealand and the United States—the Maoris and the American Indians respectively—is accurately

documented in a wealth of annual and periodic statistical publications. Birth and death rates; deaths by age, sex, and cause; morbidity; hospitalisation; notifiable diseases; social security statistics; access to services and facilities; together with long-term trends in these indices, are all available for information, analysis, and planning purposes. For the Australian Aborigines, who in many respects occupy a similar place in the society of a relatively well-developed and affluent nation, such is not the case. The immediate cause of this relative statistical vacuum is obvious enough, but the underlying reason is more elusive.

The immediate cause is that Aborigines are not identified as such in State or national records of birth, death, marriage, hospitalisation, infectious disease notification, pension entitlement, Social Welfare assistance and so on. This is not strictly true in that records are kept—or have been kept—of the occurrence of births and deaths for Aborigines who are wards of a State Aboriginal welfare department, or resident at government settlements and mission settlements. Such groups cannot be equated with the Aboriginal population as a whole. The part-Aboriginal population not living in government or mission settlements is completely undocumented in any official returns which would permit the calculation of health and vital statistics. With wards of a welfare department, or residents of settlements and missions, the published data (that is, data generally available to the public and to research workers) are extremely limited, and deaths or illnesses cannot be linked to the relevant medical documentation except to a limited extent through personal research in local records.

This, of course, is a tremendous handicap in Aboriginal health research and planning. The absence of data on the occurrences to the Aboriginal population is the reason why the imprecision of population estimates is a relatively minor handicap. The occurrence of both factors together is the reason why it is necessary to mount special surveys to evaluate the extent of Aboriginal health problems when the same evaluation of white Australian health problems is often available automatically from central records. This is not to imply that general Australian health indices are well documented—they are not, in many areas, nearly as detailed or covering as broad a picture as those already referred to as available for Maoris and American Indians.

What the writer considers to be the generally poor quality of health data retrieval in this country is not the subject of this study, but it is one of the underlying reasons for the statistical void in the matter of Aboriginal health. So long as the void persists, however, there is little scope

for more than a 'muddle through' policy in health and social planning—the series of *ad hoc*, pragmatic decisions and changes in policy which have so bedevilled the Aboriginal population since 1788.

There would appear to be only two feasible solutions to the problem. One is to set up multiple, co-ordinated, *continuous* data-collecting projects implemented through an army of community health workers in close personal contact with the target population. The other is to introduce the identification of Aborigines in the documentation of events—those events which the various indices of health are concerned with. While the first solution has great appeal, I wish to limit the present discussion to the second.

There may be many who would claim that the recording of Aboriginal identity on relevant documents would be an equivalent or greater handicap for Aborigines than the lack of information which is the present handicap. This may be so, but the Aboriginal viewpoint on this matter is unknown and untested, and its adverse effects purely speculative. In the United States of America and New Zealand, where the inclusion of 'race' in data relating to births, deaths, and illnesses is an established practice of long standing, there are no reports of adverse reactions by their indigenous minorities (N.Z. and U.S. Departments of Health, personal communications). There is no need, of course, for the information on race to appear on the certificate which a person may have to show to others, so long as it appears on the notification and is abstracted as a variable for tabulating purposes only. In the United States, the information also appears on the actual certificate (U.S. Department of Health, 1963). One of the correspondents contacted by the writer commented that there was no apparent sensitivity to answering questions on race in an official document, although the reaction socially might be quite different.

The introduction of such a practice in Australia, now, may have untoward effects and would certainly generate political repercussions. These must be balanced against the untoward effects of avoidable ignorance, and the political repercussions and social consequences of 'muddling through' in the field of Aboriginal health and social welfare.

The present situation, then, is that no overall health and vital statistics are available for the Aboriginal population—neither for full-blood Aborigines nor for part-Aborigines. The data that are available are not generally of high quality or broad coverage, and are available for limited administrative or geographical groupings of Aborigines, mostly institutionalised full-bloods.[1] In Australia, any special risks associated with

[1] The writer apologises for the repeated use of the word 'full-blood' in place of 'of total or

occupational, economic, or residential status can be inferred with considerable accuracy from information provided as a matter of course in most notifications or social records. To infer Aboriginal identity, and determine the associated special risks, is exceptionally difficult. Some of these difficulties will be discussed further when dealing with Aboriginal mortality data which, apart from data from institutionalised Aboriginal communities, can be acquired only by inference. Unfortunately there is no choice but to tolerate the probability of bias in the results, although every effort has been made to minimise this effect.

THE CLASSIFICATION OF EVENTS

Even in the most remote Aboriginal communities, the fact of a birth or death is likely to be recorded, and serious or grossly incapacitating illnesses are unlikely to occur unnoticed. However, two aspects of the data on such events may be questionable as to accuracy: the age of the person affected, and the medical diagnosis.

Inaccuracies in the recorded age of persons dying, or suffering from a noteworthy illness, may influence the distribution of such events by age-group when the data are tabulated. Fortunately for the health statistician this effect is minimised in the Aboriginal population because the chief impact of death and disease is upon the younger age-groups, particularly the children. In the young Aboriginal population, either a record of birth exists or the age may be more accurately assessed by indirect evidence. The estimation of the age of children from growth parameters—height and weight or the 'general impression' generated by these parameters—may have occurred in the past where lack of records and difficulties in communication precluded a more objective assessment; in view of the well-documented evidence for widespread growth retardation in Aboriginal children, this method is not only invalid but medically dangerous where it leads to a growth-retarded child of 7, for instance, being accepted as a healthy child of 4. The extent to which Aboriginal children's ages have been underestimated in death and disease records because of this misjudgment is unknown. Where the possibility of the error is generally recognised, it is unlikely to have much effect on the statistical picture, but attention has been drawn to growth-retardation in the last few years only (Jose *et al.*, 1969).

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more than half Aboriginal ancestry' but can find no suitable alternative. In this study, the term 'full-blood' is intended to convey a particular geographic, economic, and cultural situation rather than a biological one.

On the other hand, inaccuracies in medical diagnosis are likely to continue to be a major source of statistical misrepresentation of the real situation. There are several underlying reasons. In the first place, Aborigines in remote areas may not have access to skilled medical opinion and diagnostic facilities for their illnesses before they either recover or die. Secondly, Aborigines may not make use of such facilities even when they are available. The reconstruction of an accurate diagnosis after the event is difficult where the patient has recovered and impossible in the case of death from illness without an autopsy. Autopsies may be forgone for a variety of reasons, including unwillingness of the relatives to agree to this procedure. Crotty and Webb (1960), reporting a survey of Aboriginal deaths in the northern part of the Northern Territory in the latter 1950s, stated that in 179 out of 354 reported deaths no accurate diagnosis was possible. Of the fairly certain diagnoses, 108 out of 175 were based on autopsy findings.

A third reason for inconsistencies in medical diagnosis is local medical custom, so that in one State or area within a State local medical opinion as to the underlying cause of a death or illness may differ from that in another area or State. There is evidence for this being a factor in the differences in patterns of causes of death between the Northern Territory, northern Western Australia, and north Queensland full-blood populations, in data made available by the Bureau of Census and Statistics. Certain diseases of childhood, such as malnutrition, figure prominently in one State but not in another when there is no apparent explanation for the difference other than medical custom. The way in which medical custom is likely to operate here is as follows: when the physician completes the medical details on the death notification he is expected to arrange the events leading to death in a logical sequence: 'bronchopneumonia' due to 'operation' due to 'strangulated hernia', the last-mentioned diagnosis being regarded as the underlying cause of death—which is coded as *the* cause of death and appears in the death statistics. There is additional space on the death notification, however, for contributory causes—that is, illnesses or abnormalities which, although not considered directly responsible for death, are believed to have accelerated it or added to the effects of the 'primary' cause. It is largely a matter of personal opinion (of the certifying doctor) whether diseases such as malnutrition, worm infestation, parental neglect, anaemia, or any other chronic illness or factor, are regarded as underlying or contributory causes in cases of death from such acute diseases as gastroenteritis or pneumonia. Where there are multiple 'contributory causes', a doctor who wishes to attribute the

death to one of them as the 'primary' cause can, of course, choose only one of them. It is the writer's contention that this choice is very much influenced by local custom—by a tradition of identification of 'primary' causes established for a particular region or perhaps a particular State.

The most apparent effect of such inconsistencies is upon the comparability of data from different localities, regions or States. A more subtle consequence is upon the priorities established for dealing with health problems—in one area statistics may signify a 'gastroenteritis problem' and in another area a 'malnutrition problem' when in fact the basic disease patterns are identical.

There is no evidence for there being any official policy of playing down certain causes of death or illness, but there is a possibility that current custom is influenced by past policy and administrative convenience in what was then the frontier of white settlement.

To sum up, then, the acquisition of reliable health and mortality data for Aborigines, on an Australia-wide basis, poses some special problems: the identification of the population at risk; the identification of those affected by events of medical or health interest; and variability in the categorisation of events. These factors inevitably produce inaccuracies and bias (as well as voids in knowledge) in the evaluation of Aboriginal health. At least some of them are simply remedied.

THE EFFECTS OF ABORIGINAL HETEROGENEITY

One outcome of a presentation of Aboriginal health statistics is the formulation of an Aboriginal health stereotype—either for a population or a community. This is intentional, since the object of such a presentation is to reduce a mass of detail and variability to a form that can be handled for analysis and discussion. Built into the concept of an 'average' or a stereotype is the concept that it is a valid representation of actuality, however condensed the raw data may be in the process.

While it is well recognised that the 'average' person or community is largely a mythical representation, most of us probably feel that we can appreciate the reality underlying the average, and make allowances for the effects of condensation in producing it. The underlying statistical assumption is that the population (or sub-group) is relatively homogeneous for the variables which are being considered; when the population is not homogeneous, the usual procedure is to further subdivide it by age, sex, residence, occupation, etc.—until each group is internally homogeneous. What I wish to suggest here is that the 'average Aboriginal' or the

'average Aboriginal community' is more of a myth than most population averages, and in particular is more mythical than the 'average Australian'.

Almost any white Australian, if he wishes, can uproot himself and his family, move across the nation, re-establish himself in a job and similar home environment to that he has left, and fit himself into the local socio-economic system. His diet, living habits, attitudes, behaviour, and socio-economic status will change in a superficial way only. Because most white Australians are interchangeable in this sense, the concept of the 'average Australian' has a certain validity.

If general mobility is any test of homogeneity, then the 'average' Aboriginal Australian, not being interchangeable to this degree, has less validity. There are the tribally-oriented Aborigines on the northern reserves and missions, the stock-men and their families born on cattle stations, the rural-urban fringe-dwellers in all States, the metropolitan slum-dwellers, and the economically and often socially assimilated part-Aborigines of the larger cities and towns. Whether full-blood or part-Aboriginal, the individual is restricted in his ability to merge in with communities with a different background.

Added to this (and partly responsible for it) is the fact that the Aboriginal population consists of a large number of small communities, each isolated from the others by distance or natural obstacles, each with its own climatic, topographic, social, economic, or administrative peculiarities, and each with its own group history.

When these two factors are taken together—heterogeneity of communities and small community size—it can be seen that the amalgamation of discrete populations necessary to reduce the effect of chance (i.e. to increase statistical significance) also reduces the validity of the average or stereotype that results. It would appear particularly likely that the combination of full-blood and part-Aboriginal populations would produce a picture of an entirely non-existent stereotype and the two groups have been separated in this study partly for this reason.

This conflict between what might be called statistical accuracy on the one hand and representational accuracy on the other might be partly resolved in an Aboriginal study by looking at the averages produced for a specified community observed over a number of years—five-year average death or disease rates, for instance. Unfortunately this is not often possible because the necessary data are rarely available for an unbroken series of years. This again is a criticism of the quality of Aboriginal data.

SELECTION AS A FACTOR IN ABORIGINAL HEALTH STATISTICS

Although Aborigines tend to live in small discrete communities, data relevant to health are usually available only for a narrow range of such communities—the more closely supervised government settlements, mission settlements, and the larger cattle stations. Elsewhere there may be no one to keep the records, or it may be impossible to separate out the records relating to Aborigines in a mixed community. The only exceptions to this rule occur where special community-based health surveys have been undertaken—usually by governments or universities; occasionally by private individuals or commercial firms.

The available data are therefore biased towards that component of the Aboriginal population which, by preference or necessity, is in a dependent situation. There is an obvious underrepresentation of the relatively large fringe-dwelling and metropolitan groups, with their special environmental, social, and economic features, but greater independence and self-sufficiency when compared with the residents of the supervised communities. Generally, the supervised communities have better access to both medical and health services, and pay little or nothing for them.

All these factors are important in determining health status and its consequences, so that it is unsafe to generalise health indices from the easily accessible data to the total Aboriginal population. Studies of the part-Aboriginal community are few, and this has created a large and very important gap in knowledge of Aboriginal health.

The bias introduced by dependence on the populations of supervised communities for health and related statistics varies with the type of community, its geographical location, and the parameters being considered. In the southern areas of Australia, where part-Aborigines predominate, the settlements and reserves are peopled by the descendants of former inhabitants of the same communities, but not all their descendants. The discrepancy between the expected growth of the population (based on births minus deaths) and the actual growth rate shows that there has been a continual skimming-off of surplus population over a long period—in some cases since as far back as the records go. It may be presumed that those who have left the settlements and reserves were more economically or socially viable, or more independent in spirit, and that those who remained were the opposite. Added to those who remained are the outsiders who, for one reason or another, have been unable to maintain their independence. Thus the southern reserves, in latter years, have tended to be a refuge for the socially, psychologically, or economi-

cally handicapped—even though the inhabitants may not see themselves in this light. There are numerous exceptions—families with a good stable income, a car and household improvements they have installed at their own cost; families who would appear to be quite able to leave the settlement and do as well elsewhere if they wished, but preferring to remain perhaps because of suitable local employment or family ties.

One of the results of this skimming-off process, and re-entry based on failure to cope outside the settlement, may be the observed absence of young adults and the high proportion of children in New South Wales Aboriginal settlements (Long, 1970:87; Moodie, unpublished). Apart from the social and psychological effects of this distribution, the high ratio of dependants to income-producers is likely to exert a direct effect on nutrition, the domestic environment, and access to medical attention.

In the north of Australia, the institutionalised full-blood population appears to be a less-selected sample of the total. This is particularly so in the Northern Territory, where the population of the government and mission settlements, together with the semi-institutionalised cattle station population, account for something like four-fifths of the total Territory Aboriginal population. Not only does the sample approximate the total in numbers, but in each community the age, sex, and family structure is fairly uniform (Moodie, unpublished). The greatest observable difference lies between the Aborigines on cattle stations and the remainder —the former show an excess of young adult males, and a relative absence of children and the aged because young men leave other communities to work in the cattle industry. The *per capita* income may be higher on cattle stations, but the higher proportion of single men will prevent this being reflected to the same extent among the children and the aged. Personal observations in the Northern Territory suggest that much of the extra *per capita* income on cattle stations is spent on personal clothing and effects by those that earn the money.

Another factor differentiating the health ecology of northern and southern institutionalised Aboriginal communities is that in the north, medical or para-medical (nursing, first-aid, and dispensary) services are available free in nearly all full-blood communities, and there are no associated transport costs such as bus or taxi fares to the local hospital, clinic, or pharmacy as is usual in the southern reserves. The costs of hospitalisation for a full-blood Aboriginal in the north, including ambulance or air transport, are nil or nominal. In the south, the part-Aboriginal without a pension is expected to pay his hospital and ambulance bills, although the matter is not generally pursued if he is unable or refuses to

do so. It seems certain that inability to pay for services diminishes the enthusiasm of both the service and the recipient to the extent that southern part-Aborigines living within or on the fringe of towns and cities get less medical and hospital attention than they require.[2] In the northern communities—certainly in the larger settlements and missions—the impression is that the Aboriginal inhabitants can obtain as much medical advice and treatment as they wish—sometimes perhaps more than they wish!—by attending the daily clinics.[3]

The important point about data available for part-Aboriginal communities in southern Australia is that they come from a smaller sample of the total, and one that tends to be locally atypical in some socio-economic parameters, family and age-structure, and more affected by economic hardship in relation to access to medical and related services. The point has already been made that the defined part-Aboriginal 'total' population of a region or State is already a selected proportion of the real total.

[2] From personal observation and inquiry in a New South Wales coastal town, 1970-1.
[3] From personal observation throughout the Northern Territory, 1968.

3 DEMOGRAPHIC FACTORS IN ABORIGINAL HEALTH

The importance of the human component in the human environment, as reflected by various demographic indices, requires a brief consideration of demographic factors in Aboriginal health—particularly where Aboriginal demographic patterns differ widely from Australian norms. These demographic factors are numbers, distribution, density, family size, age-structure, fertility, and mobility. Mortality is considered separately, along with cause of death, in the next chapter.

No demographic analysis is proposed, but rather a consideration of some interactions between demography, disease patterns, and access to health services.

NUMBERS

There are no national figures available to this study for Aboriginal numbers estimated from the social definition of Aboriginality, and none will be available until the results of the 1971 Census are published. Census figures, including those for the 1966 Census, were based on a biological definition—the proportion of Aboriginal ancestry.[1] Before 1966, census questions on race were literally unanswerable for the majority of part-Aborigines—those whose parents were themselves part-Aboriginal—because the only categories open to them were 'full-blood Aboriginal' and 'European'. The category 'half-caste' was restricted by the directions in the census schedule to offspring of one Aboriginal and one European

[1] The 1966 Census Schedule question on 'Race' was: 'State each person's race ... state whether Aboriginal, Chinese, Indian, Japanese, etc., as the case may be. If more than one race, give particulars, for example, ½ European-½ Aboriginal, ¾ Aboriginal-¼ Chinese ...'.

Table 1: *Aboriginal population by State, 1966*

	Qld	N.T.	W.A.	S.A.	Vic.	N.S.W.	Total
Aborigines*	12,000‡	20,120	9,905	3,128	—	130	45,283
Part-Aborigines†	29,700‡	4,000	11,985	4,632	3,500	23,000	76,817
Total	41,700‡	24,120	21,890	7,760	3,500	23,130	122,100

* Persons of more than 50 per cent Aboriginal ancestry.
† Persons of 50 per cent or less Aboriginal ancestry.
‡ Figures for Queensland exclude Torres Strait Islanders who, by reason of ethnic and geographic affiliations with Papua, are not included in this study.

parent. The necessary rationalisation of this anomaly by or on behalf of the majority of part-Aborigines made all three categories open-ended.

Although the 1966 Census schedule removed this anomaly, it still posed difficulties for part-Aborigines of complex origins and for those who did not know their exact racial admixtures in terms of fractions. At least partly as a result of the changed schedule in 1966, the enumerated part-Aborigines and full-blood populations were so different in numbers from those predicted from the 1961 Census that the Acting Commonwealth Statistician stated:

> the revised instructions resulted in a greater number of persons being classified as 'full-blood' at the 1966 Census than at previous Censuses. However, evidence suggests that the number of full-blood Aborigines may well be overstated, particularly in New South Wales and Victoria, and the number of half-bloods understated owing to some half-bloods answering 'Aboriginal' and therefore being classified as 'full-blood'. There is therefore some doubt as to the accuracy of the separate figures . . . and for this reason it is considered desirable not to publish separate figures for these two groups (C.B.C.S., 1967)

It should be noted that the figures given were for 'full-blood' (more than 50 per cent Aboriginal ancestry) and 'half-blood' (50 per cent Aboriginal ancestry) combined, and did not include part-Aborigines of lesser degrees of Aboriginal ancestry. The addition of the latter might be expected to produce figures which would be a close approximation to a socially-defined enumeration. Such figures have not been generally released by the Bureau of Census, but are included in a population estimate for 1966 prepared by the Department of Territories based on departmental records, local knowledge, and some guesswork, as evidenced by the frequency of numbers ending in two or three zeros (Commonwealth Department of Territories, 1967). In spite of the reservation about the accuracy of these figures, it is considered that they are a more valid representation of the numbers of Aborigines identifiable on social grounds

than any others available, including those calculated from census data. They are reproduced as Table 1.

The total Aboriginal population from Table 1, a little over 120,000, amounts to close to 1 per cent of the total Australian population.[2] This is an important fact in relation to the place in Australian society, economics, and health and medical services occupied by the Aborigines. The study will show that in many areas relevant to health the Aborigines contribute far in excess of 1 per cent of the 'problems', and appear to need more than 1 per cent of the health and medical resources available.

The proportion of Aborigines varies according to the definition of Aborigines, according to region or State, and according to time. From the figures in Table 1 and the total State populations in 1966, the full-blood, part-Aboriginal and both groups are shown as a percentage of the State total populations in Table 2. Since health and medical services are a State responsibility, these figures give a better indication of the potential impact of Aboriginal health problems on State budgets and other resources.

Table 2: *Aborigines as a percentage of State populations, 1966*

	Full-bloods %	Part-Aborigines %	Total %
Northern Territory	35·7	7·1	42·8
Western Australia	1·2	1·4	2·6
Queensland	0·7	1·8	2·5
South Australia	0·3	0·4	0·7
New South Wales	0·003	0·05	0·05
Victoria	—	0·01	0·01
Australia	0·4	0·7	1·1

Published estimates for the full-blood population suggest that they became a minority in the total Australian population by 1830, due to a marked decline in their numbers; the fall to present levels (0·4 per cent) has occurred mainly as a result of rapid growth of the white population, with a further gradual decline in the Aboriginal population through to the 1940s and 1950s, when the decline in numbers halted. For the future, Jones predicts that if present levels of fertility and mortality are maintained, the Aboriginal population will have doubled by 1986 (Jones, 1970a:46).

[2] The American Indians (excluding Alaskans) number approximately 500,000, or 0·3 per cent of the total population of the United States. At the 1961 Census in New Zealand, Maoris numbered 167,000, or just under 7 per cent of the total.

It seems likely that the proportion of Aborigines in Australia will have doubled from 1 per cent to 2 per cent by the end of the century, with rapid growth of the full-blood as well as the part-Aboriginal component. The implications are serious: if Aboriginal health status remains the same, the health 'problems' will have doubled in volume, in cost to the community, and in the cost of remedial action; if the health 'problems' are solved without accompanying solutions to social and economic problems, their population increase will be further accelerated and their quality of life threatened in other ways, again with repercussions upon their overall health status. The case for urgent, carefully planned *co-ordinated* action in health, social, and economic problem-solving appears overwhelming, not only for humanitarian reasons but for reasons of cost-benefit to the total community of Australia thirty years hence.

GEOGRAPHIC DISTRIBUTION AND HEALTH

Tropical Australia is singularly free of the classical tropical scourges—malaria, cholera, sleeping sickness, schistosomiasis, smallpox, yellow fever, plague—and has only a few diseases that are determined by climate, such as hookworm, trachoma, and the few insect-borne viruses and tick-borne rickettsias that are a relatively insignificant contribution to Aboriginal ill-health.

The relative absence of vector-borne diseases and zoonoses in Australia, when compared with other continents, is partly due to Australia's past physical isolation and the absence of suitable vectors, and partly due to present quarantine measures. The sparse, nomadic Aboriginal population of preceding millennia must also have discouraged the evolution of endemic or epidemic communicable diseases.

The result is that no matter where Aborigines live today, their domestic, social, and economic environment, and their behaviour within this environment, have more bearing on their risks of ill-health than their geographic location *per se*.

The distribution of Aborigines does affect their access to health and medical services, however. The distribution of full-blood and part-Aborigines illustrated by Long (1970:2-3) shows that the majority of full-bloods live north of a line which curves irregularly south and west from Cooktown on the north Queensland coast to the Great Australian Bight, and thence north-west to North West Cape in Western Australia. It is no coincidence that this line corresponds almost exactly with one drawn by Taylor (1947:4) to divide what he called 'Empty Australia' from

'Economic Australia'—'Empty Australia' was either too hot, too arid, too infertile or too remote to have encouraged white settlers in sufficient numbers to have displaced the Aborigines. Similarly, more intensive settlement in 'Economic Australia' has both displaced the full-blood Aborigines and produced the part-Aboriginal population.

The part-Aboriginal population thus shares the administration, facilities, goods and services of the white population in a way that the full-blood Aborigines for geographic reasons and dependent historical reasons cannot. For health and medical services they are theoretically better placed provided that access to those services is not restricted by other factors associated with their part-Aboriginal identity. If, as has already been mentioned, their access is restricted, they may be less well placed than the Aborigines in North Australia.

A final point is that Aborigines, being largely a rural and rural-urban population, share the health handicaps of this situation with the rural minority of white Australians, and not the metropolitan majority. It is well documented in Australian vital statistics that rural Australians have higher rates for infant and maternal mortality and deaths at any age from certain communicable diseases than their metropolitan compatriots. What is not clear is the extent to which these rural death rates are raised by the relatively small percentage of part-Aborigines included in the figures on which rates are based. Whatever this effect might be (and it is likely to be considerable in the case of infant and particularly second-year mortality —Moodie, 1969:183), Aboriginal health statistics are perhaps more validly compared with rural than general Australian health statistics, because the added effect of the rural-metropolitan differences in death and disease rates in the non-Aboriginal population are avoided. Unfortunately, separate rural and metropolitan figures are rarely featured in official statistical publications, nor is it possible to calculate them from data for single years of age or single causes of death or disease.

ABORIGINAL POPULATION DENSITY AND COMMUNITY SIZE

There are two aspects of population density—general and local—only one of which is particularly relevant to Aboriginal health.

The figure for the number of Aborigines per square mile in Australia is approximately 0·04, a practically meaningless figure because the Aborigines are not spread thinly across the continent as they once were, but clustered thinly in discrete localities.

Apart from the occasional isolated individual or family, and the very rare nomadic family groups in Central Australia or the remote north, Aborigines live in communities ranging in size from perhaps a dozen persons to around 2,000. Metropolitan Aborigines, as in Sydney, may number 10,000 or more but they are not communities in the same sense, being distributed over a number of widely scattered suburbs (Beasley, 1970:137).

Few isolated Aboriginal communities in the north are sufficiently large to warrant a resident doctor, and a large number are too small to warrant a resident community nurse. The same may be said for most part-Aboriginal settlements in temperate Australia. Both these statements should be qualified in that it is the current shortage of suitable personnel, Australian attitudes to the qualifications necessary for such work, and economic considerations that are the determining factors—not the Aboriginal health situation.

Small community size has some beneficial effects on health in that it limits the spread of epidemic disease throughout the population and reduces the possibility of maintenance of some communicable diseases within the population—those diseases with a short course followed by a period of immunity die out when the community runs out of susceptible persons. However, epidemics when they do occur may be more devastating in that all age-groups may be affected at once, with social and economic disruption as a result.[3]

The local density of Aboriginal communities—the number of people per acre excluding waste land and waterways, etc.—is difficult to estimate without survey data. From personal experience in the Northern Territory, the writer estimates that the figure may reach or exceed 100 per acre in some settlements and missions, in single-storey dwellings. This may be compared with the Sydney Municipality of Waverley (the most densely populated) for which the overall figure is approximately 29 per acre (N.S.W. Bureau of Census and Statistics, personal communication, 1970), including parts with a much higher density in high-rise apartments. The implication here is that the typically small Aboriginal dwellings may be very closely juxtaposed indeed by Australian urban standards, with conditions favouring the diseases of poor hygiene and overcrowding, particularly the communicable diseases.

A different implication of high density in Aboriginal housing is that it is physically easy to carry out household visiting during surveys, or in

[3] See Adels *et al.*, 1962, reporting an epidemic in Cape York with 100 per cent incidence of measles in most of the west coast missions in 1944.

community health practice, once the appropriate personnel reach the location of the community. The writer has had personal experience of this, and believes that most people with similar opportunities would agree. There are few Aboriginal communities where a clinic building is necessary for the same reasons as in suburban or urban Australia—as a central point close to public transport. The clinic-orientation of Australian public health agencies in the past has been unfavourable to the improvement of Aboriginal health at the family or domestic level. It is pleasing to note that there is a recent trend towards more home visiting and less clinic work—a trend which brings the benefits of a health service to the people rather than the other way around.

Other aspects of density of importance to health are the number of persons per dwelling, and the number of persons per room—factors which have been considered by Rowley (1967:10) under the name of 'accommodation pressure'. The number of persons per dwelling is directly related to family size (including grandparents and other relatives, and permanent 'visitors') and is of particular relevance to the physical facilities present—cooking, food storage, washing, bathing, excreta disposal, sleeping—all of which may be grossly overloaded in very large households irrespective of the number of rooms. The average Australian domestic refrigerator and septic tank, for instance, are designed for an average household of around five persons. Even if these facilities are present in an Aboriginal household of fifteen, there will be problems of food spoilage and possible food poisoning on the one hand, and raw septic tank effluent contaminating the environment with pathogenic bacteria and worm eggs on the other. Inadequate sleeping accommodation results in sitting and dining areas, and bathrooms when present, being adapted as bedrooms and storage areas, with further deterioration in domestic hygiene.

Compared with the average persons per dwelling for Australia as a whole in the 1961 census (3·55), some recent figures for Aboriginal communities are: rural New South Wales—7·04 (Rowley, 1967:9); Walgett, New South Wales—8·9 (Edwards, 1970:1008); Sydney metropolitan—7·03 (Beasley, 1970:140); Northern Territory—5·2 (Moodie, unpublished)[4]; New South Wales south coast 6·6 (Hausfeld and Moodie, 1967:44). Most medical surveys of Aborigines mention the number of persons in the area but very few mention the number of households, so that a wider range of data on household size is not available.

[4] From a random sample of households in 19 localities.

The average number of occupants per household may not be as significant as the range, because it is in the upper part of the range that the problem of overloaded household facilities becomes acute. In a study of New South Wales South Coast Aborigines, about one house in seven contained ten persons or more, the highest number being seventeen (Hausfeld and Moodie, 1967:11). To be able to cope with the household's needs at current working-class standards for Australia, these dwellings would require two refrigerators, two bathrooms, two toilets, at least five bedrooms, and at least twice the household income. Aboriginal housing, then, poses problems not only with regard to the number of houses required, but also with regard to design, size and the load on domestic facilities. Such problems need to be solved in the face of a generally low *per capita* income for the occupants.

The number of persons per room in Aboriginal communities has also been studied by Rowley and others, in the face of some difficulties in comparing the results for Aborigines with those for Australia because of the frequency of overflow of Aboriginal occupants from bedrooms to other rooms. Overcrowded sleeping space is generally believed to add to the risks of contracting communicable disease, particularly respiratory and contact infections. It is difficult to be sure whether the primary factor is overcrowding itself or some of the other factors which accompany overcrowding—low incomes, poor appreciation of hygiene, and other defects in domestic facilities. School dormitories and army barracks are often overcrowded in terms of 'persons per room', but are not hotbeds of communicable disease where other aspects of communal hygiene are controlled or supervised.

Apart from effects on physical health, overcrowding influences mental and social health, but this influence is not necessarily adverse except in attributes expected of white Australian society—quiet neighbours, adequate homework space for children, and opportunity for privacy within the home. Whereas overcrowding might be seen as a handicap to assimilation, it does not follow that overcrowding (according to Australian norms) is adversely affecting Aboriginal mental or social well-being; the converse is as likely.

FAMILY STRUCTURE

Differences from Australian norms in family structure among part-Aborigines have drawn considerable attention—the matrifocal family, the absent or deserting husband, the *de facto* marriages, the influence of

'granny', the wide network of 'cousins' and other relatives. It is difficult to see any direct effects on physical health, but indirect effects can be postulated: economic hardship, stress disorders, and perhaps the persistence of 'old wives' tales' in the cause and treatment of physical disease are likely to be associated with absent husbands and the influence of 'granny'. On the other hand, 'granny's' conservatism and her greater experience in child rearing and coping with illnesses among her grandchildren may operate to advantage for family health and well-being, notwithstanding possible misconceptions about health and disease. The economic handicap due to an absent husband seems likely to be balanced by a wider network of family help and advice—a store of human and financial resources to be drawn upon when needed—as has been suggested by Lickiss (1971: 224).

For social and mental well-being, the consequences of different structure and dynamics in Aboriginal households will be particularly stressful for part-Aborigines on the verge of full acculturation. In this situation, remaining differences will be a barrier to full acceptance by the white community, and in terms of the functional definition of health, members of such households may be unable to fulfil their appropriate social roles in the wider community through lack of support within their families—as well as through lack of experience, lack of a suitable model, or physical disability.

FAMILY SIZE

The reference here is to the size of the nuclear family, rather than the numbers in the household. The immediate cause of large families is high fertility (whether intentional or otherwise) and low infant and childhood mortality. The immediate results as far as health is concerned will depend on the social and economic setting, the physical and mental health of the parents—particularly the mother—the nutritional environment, the spacing of children, and the final size that the family reaches. All these factors may vary widely even within a small community. Those who are most likely to suffer ill-effects from excessively large nuclear families (leaving the level of 'excessive' to be determined in a particular case by the factors mentioned above) are the mother on the one hand, and the middle and later children on the other.

A large number of children is inevitably associated with early commencement of childbearing and a short interval between pregnancies. In a nutritionally poor environment, the mother may not only become physically, nutritionally, and mentally debilitated, but increases her

chances of death or disability from one of the complications of childbirth and the puerperium. The later children may not only be malnourished at birth because of maternal malnutrition during pregnancy (from deficient body stores of iron, calcium, vitamin A, other minerals and nutrients; Trowell and Jelliffe, 1958:14), but receive another nutritional setback when their access to breast-milk and extra attention is denied them within twelve months or so upon the arrival of another infant. In the intervening period, malnutrition may result from inadequate breast-milk, or breast or cow's milk deficient in specific nutrients—all of which can in part be attributed to effects of a rapidly expanding family.

Theoretically, the first few and the very last child born to a large family in nutritionally poor circumstances should have a nutritional advantage over the remainder. In nutritional studies of Aboriginal populations information on family size and the place in birth-order of malnourished children have not been reported upon, although the relevant data should be easy to obtain.

Some recent figures available for family size, expressed as either the total live births (TLB) or total surviving children (TSC) to women who have had children (aged 40 and over except where indicated) are as follows:

Australia (non-Aboriginal) (TLB)	2·5	(Jones, 1970b:25)
Victoria (part-Aborigines) (TLB)	6·5	(Barwick, 1963 quoted by Jones, 1970b:25)
N.S.W. (part-Aborigines) (TLB)	6·4[5]	
Sydney (part-Aborigines) (TLB)	4·7	(all ages—mean unstated) (Beasley, 1970:178)
N.S.W. South Coast (TSC)	5·6	(mean age 36·6 years) (Hausfeld and Moodie, 1967:116)
N.T. (full-blood) (TSC)	3·3	(Moodie, unpublished)[6]

Of greater significance to health and health services (as well as social services) than the average number of children per family is the proportion of larger-than-average families in the part-Aboriginal population—that is, the proportion of families who are likely to face some sort of problem because of large or rapidly expanding numbers of children. Data from

[5] Social Science Research Council, Aborigines Project. Data tabulated by the author, quoted with permission of the Director, C.D. Rowley.
[6] From data obtained by random sampling.

rural New South Wales Aboriginal communities compared with the equivalent for Australia, for women with children, are as follows:

	Rural N.S.W. Aboriginal	Australia
Women with 5 or more children (age-group 20-29)	1 in 4	1 in 22
Women with 10 or more children (age group 30-39)	1 in 12	1 in 87

The economic handicap of a very large family may be mitigated to some extent by two factors: contribution to household income by older children leaving school early; and child endowment payments (which may be a large component of total income for families already on a low income). Since medical and hospital benefit contributions at the family rate are not influenced by family size, large Aboriginal families who belong to one or other benefit organisation might appear to be at no great disadvantage in meeting the cost of medical and hospital care. However, this overlooks the fact that rebates do not completely cover costs, particularly the costs of doctor's prescriptions which will now cost the family $1.00 a time. The subsidising of poor families with respect to medical and hospital insurance, recently introduced, is based upon levels of total family income. A family with one child with an income of $45.00 per week can obtain free medical and hospital insurance, but a family with ten children and an income of $55.00 per week cannot. The differences in *per capita* family income—from $15.00 to $4.60—are not taken into account, making somewhat a mockery of the subsidy with respect to large families in poor circumstances. This anomaly is easily removed by making the subsidies relate to family *per capita* income rather than family total income.

There are, of course, many other costs that are not covered by subsidy or insurance, such as the cost of fares to and from the doctor and the pharmacy, for hospital visiting, and when country patients are referred to city specialists or out-patient clinics, and all of these are a proportionately greater burden for the large family on a small income. The point is simply that a high proportion of Aboriginal families are so far outside the Australian norms in the number of dependent children that partly subsidised health and medical services designed for Australian norms are still effectively beyond reach. Because the health status of Aboriginal children is considerably lower than the Australian norms in most respects,

the effects of larger family size are compounded by the greater need for health and medical services. The mechanisms for creating several vicious circles are apparent.

AGE-STRUCTURE

The age-structure of a population, as illustrated by a population pyramid, is largely determined by its birth rate and, to a lesser extent, its age-specific mortality rates. The Aboriginal population's age-structure has been shown from census and other data to reflect the high birth rate and reduced expectation of life, with a net effect of rapid population increase (Jones 1963, 1970a, 1970b). As would be expected, the population pyramid reflects at the community level what has been discussed above at the family level—a high proportion of dependent children to bread-winners.

It is not necessary to devote much space to a discussion of the age-structure in relation to the health of Aborigines, since the important factors have been covered in the discussion of family size, at the level where the economic and other consequences of the broad-based population pyramid exert their main effects in the health field.

The implications are that even with full employment, the part-Aboriginal population could not be self-supporting at the same standards of usage of health services as the white population, although this is offset to some extent by the reduced requirements for care of the aged.

The pronounced difference in age-structure between part-Aborigines and white Australians can thus be seen as an important factor encouraging dependence of the former on the latter—and therefore a factor in Aboriginal mental health also. The cause of the problem is high fertility; the solution should be sought in control of excessive fertility—preferably, of course, by encouraging internal social controls in the Aboriginal community.

FERTILITY

High fertility in the Aboriginal population can be seen as underlying many of the demographic features which distinguish Aborigines from other Australians—the youthfulness of the population, the large families and overcrowded households, the high densities of population within Aboriginal settlements. It is an important factor also in that it contributes to social and economic difficulties, is a handicap to assimilation, and alarms the white population.

Jones (1970a:22) has stated that 'the level of Aboriginal fertility in amost all parts of Australia is already high, and where it is relatively low is on the increase'.

Some of the health consequences of 'excessive' fertility have already been discussed under different headings. Unless there is a major social and economic revolution in Aboriginal society, these consequences are not only likely to remain for the foreseeable future; if Jones's predictions are correct they will get worse.

There is considerable justification for regarding high fertility as a 'disease' of the community in its own right, particularly if high fertility is uncontrolled (i.e. involuntary). In traditional societies, including Aboriginal society where high fertility has not been countered by high infant mortality (that is, an infant mortality of around 300-500 per 1,000 live births), the societies themselves have often controlled it by evolving rules governing sexual behaviour or incorporating practices such as infanticide into its accepted norms of behaviour. Developed Western societies and many non-Western societies (India, Japan, for example) have relied on contraception, abortion, and sterilisation to achieve the same ends, with varying success.

It is not clear whether high fertility in many Aboriginal women is voluntary or involuntary. A series of fifty part-Aboriginal mothers questioned by the writer in 1967 as to what they thought was an 'ideal' number of children gave answers which, when averaged, yielded a high 'ideal' of 4·8, but their 'mean number of children living' was 5·6 and the 'mean number of live births' in the group was 6·1. In this small sample, the indications were that their high fertility was partly voluntary and partly involuntary.

Voluntary high fertility may be seen as a form of 'social insurance' initially against the effects of high infant and childhood mortality; it may persist not only as a 'custom' but—in a welfare-supported community—as a source of income through child endowment payments. In Rowley's survey of rural New South Wales Aborigines in 1965, the average weekly income from child endowment of 110 mothers aged 20-39 was a little over $5.00—a not inconsiderable sum for a poor family.[7]

For Australian health and medical services responsible for Aboriginal health the implications are clear: as the social, economic, and health impacts of high fertility are made greater by control of mortality, family planning facilities and techniques must be promoted as well as offered as

[7] Calculated by the writer from Rowley's original data.

a counterbalance, together with social and economic incentives to limit family size. The alternatives would seem to be either drastic population control when ecologically limiting factors can no longer be staved off by health and welfare agencies, or continued social and economic chaos for an increasing number of Aboriginal families. The inherent instability of the present pattern of high fertility, relatively high infant and childhood mortality, and rapid population increase in the face of social and economic depression, was referred to by the writer some years ago as 'high output failure' (Moodie, 1969). This term was borrowed from clinical medicine, where it refers to heart failure (muscle exhaustion) due to or accompanying an abnormally rapid turnover in the blood circulation. The ecological analogy may not be entirely appropriate, but was intended to convey the concept of 'failure' of the Aboriginal communities from exhaustion of their resources due to abnormally high demands upon these resources, and occurring in spite of (and because of) the apparent biological 'success' indicated by a high fertility.

Two points merit comment with respect to Aboriginal fertility. Firstly, there is no suggestion intended that Aboriginal women are inherently more fertile than others—that is, physiologically more capable of becoming pregnant or bearing children. Secondly, there is no suggestion intended that Aboriginal women are resistant to the idea of family planning—where advice and techniques are available to them, both full-blood and part-Aboriginal women make use of both oral and intrauterine contraceptives.[8]

MOBILITY

The idea that part-Aborigines 'go walkabout' is ridiculous to anyone acquainted with part-Aboriginal communities and their problems, but the idea still persists—particularly among some employers of part-Aboriginal labour—as if it was a racially-inherited characteristic. The idea that full-blood Aborigines 'go walkabout' may have a certain validity, so long as it is not equated with an urge to wander aimlessly. It would appear to be quite rare for an Aboriginal to leave a locality without a definite reason and a definite objective: that is, his departure is based on a decision, not a whim or an 'instinct'. The type of mobility under discussion here is spatial mobility involving a shift of residence, or mobility associated with occupation.

[8] Based on personal and colleagues' observations in the Northern Territory and rural New South Wales.

Although it has been suggested that the sort of free-ranging spatial mobility open to Australian whites is largely ruled out for Aborigines even between Aboriginal communities, because of social and cultural factors, many writers have referred to two other forms of mobility which have health repercussions: frequent changes of residence within a locality, and the drift of Aborigines to the cities.

Frequent changes of residence—'house-hopping'—within a locality, a suburb or a city, appears to result from economic and domestic crises and landlord-tenant confrontations in metropolitan Sydney (Lickiss, 1971b: 205). Another reason given by Lickiss is as an evasive tactic when faced with legal, health or welfare department action. Similar factors have been observed to operate in a New South Wales rural-urban area, although eviction for failure to meet health or child welfare requirements was a rare event. Less frequent moves may be attributed to difficulties in obtaining long-term leases, changes in local employment opportunities, or prolonged visits to relatives. Needless to say, 'house-hopping' is most likely in those households with severe economic, marital, or other domestic problems.

From talking to a number of part-Aboriginal mothers, the writer has formed the opinion that the most important direct consequence to health of frequent moving arises from lack of continuity in health and medical care for established chronic illness, and disruption of an established appropriate pattern of behaviour for new illnesses; that is, failure to establish contact with new doctors, new hospitals, new maternal and child-health nurses, or new immunisation services. This applies only where a move has taken place between towns or districts, although families that move out of a town into the surrounding countryside may find it difficult to maintain contact with town-based health and medical services previously utilised.

The most mobile group are those who follow seasonal work, taking their children along with them, shifting from farm to farm, and usually occupying temporary accommodation of the worst kind. Upon questioning mothers of such families about their children's previous illnesses, one hears of their children's recurrent admissions to hospitals scattered over a wide area, typically 'very sick' with pneumonia or gastroenteritis, and a complete absence of any medical follow-up (Moodie, unpublished).

Risks to physical health associated with the drift to the cities of rural part-Aboriginal families in southern Australia are more likely to be the indirect result of the move—the overcrowding in relatively expensive slum accommodation, a smaller proportion of income available for food

and medical attention—but these effects may be counterbalanced by more direct access to some health services and medical care, together with better employment prospects. The writer is not aware of any evidence to support the view that families are any worse off in health in Redfern than they would have been if they had remained in a depressed rural environment.

For Aborigines in north Australia—full-blood Aborigines for the most part—'house-hopping' is minimal, primarily because Aboriginal housing is already set aside for exclusive Aboriginal use on settlements, missions, and cattle stations, and is fully utilised. Mobility between Aboriginal communities appears to be restricted in the Northern Territory to communities within the same general area, except for the town visiting pattern. Of 1,570 Aborigines of all ages from a Territory-wide random sample in 1968 (Moodie, unpublished), only 8·7 per cent had never travelled outside their locality of birth, whereas 79·5 per cent had visited or lived in a town (such as Darwin, Alice Springs), and another 7·7 per cent had visited or lived elsewhere, except in a town. For adult males taken from the same sample, 19 per cent had worked in a town, 30 per cent had worked in the pastoral or mining industry but never in a town, and a further 37 per cent had worked in other rural occupations, mostly in their own locality, but never in a town. The implication of these figures is that half the household heads at least had moved their residence in association with employment (not necessarily accompanied by their families, if they were married at the time), but that over 90 per cent of the total population sampled had travelled to other localities for one reason or another, mostly as temporary visitors, and returned. The health consequences of this relatively low degree of residential mobility are not clear; but in the rural setting of the Northern Territory there is unlikely to be any added restriction of access to health and medical services because of it.

There is evidence, in figures for increasing urban populations of Northern Territory Aborigines, of a drift to the towns—but whether this is a permanent move by individuals or families or a net effect which includes many who subsequently return to rural areas cannot be ascertained from data available to the writer. There is little doubt that the urbanisation process must accelerate with the rapidly expanding populations in many peripheral communities, if the latter are not to be overcrowded to bursting point or rapidly expanded in number or size. There may be serious implications for health in the future, particularly with the threatened development of large urban fringe communities of full-blood

Aborigines in northern towns and cities. In this situation, unlike that at the settlements in the rural hinterland, Aborigines will have to compete for health and medical services with the more affluent white community as in southern Australia.

Among the various demographic characteristics of Aborigines which have been touched on above, high fertility is suggested as outranking all others in its ultimate impact on present and future health status. It appears to operate directly and indirectly at the population, community, and household levels to place Aborigines at a health disadvantage in relation to other Australians, and to generate various stresses internal to the Aboriginal community. Fertility has been shown by Jones (1970a:22 *et seq.*) to be higher among part-Aborigines than among full-blood Aborigines, and its impact, irrespective of its degree, is likely to be higher in the part-Aboriginal population because of its closer association with the white population.

Thus far in this study it has been possible to discuss part-Aborigines and full-blood Aborigines together or in parallel, at a general level of discussion. In turning to a consideration of specific indices of health in the next chapters, part-Aborigines and full-blood Aborigines are discussed separately. Separate treatment has been necessary not only because of the differences in general and health ecology between the two groups, but also because the accessible data come from separate sources, or deal with different aspects of health. Separate treatment is not intended to imply marked differences in health status, disease pattern, or health priorities. Where such differences can be shown to exist, they will be commented upon at the appropriate point. The different ecological settings, however, most definitely suggest that different approaches are indicated to remedy a problem situation—in the health field as much as in the field of employment, education, or social integration—even though the basic problem may be identical in a particular field of inquiry.

4 ABORIGINAL MORTALITY AND CAUSE OF DEATH

From the viewpoint of a health evaluation, mortality statistics may be divided into two types—those which are primarily concerned with death as an event in itself, with its effects on population numbers and trends, the age-structure of the population, and economic aspects of human ecology; and those which are concerned with determining the causes of death and the relative and absolute risks of death from specific causes in different age-groups. The first group, usually considered as lying more within the province of the demographer, is particularly important to this study because they may be combined with incomplete cause-of-death data to derive estimated cause-specific death rates. The second group supplies important clues to the ecological and behavioural factors underlying a population's health status. Detailed cause-of-death statistics such as age-sex specific death rates by single causes point to where major health problems lie (in the medical sense) although they do not necessarily indicate solutions. This is only to say that it is never the *disease* which is the ultimate health problem, but the environmental and behavioural circumstances which allow the disease to flourish. Thus cause-of-death statistics should provide clues to further investigation rather than a basis for action.

There are some diseases responsible for a high mortality which can be controlled—even eradicated—with minimal regard for the ecological circumstances of a population. This applies particularly to those diseases for which effective immunisation has been developed (diphtheria, tetanus, smallpox, poliomyelitis) and to diseases spread by insect vectors or from animal reservoirs (malaria, plague, rabies). These diseases do not figure

prominently, if at all, in Aboriginal death and disease patterns, and their failure to thrive may be attributed to effective health action and quarantine measures. The mortality from such diseases is low or nil because the incidence is low or nil.

Where medical technology does not provide a powerful preventive weapon, deaths from such diseases do not reflect directly the incidence in the community. The death rate from gastroenteritis, for instance, is influenced not only by the incidence of gastroenteritis in the community, but also by the speed with which appropriate treatment is sought and the efficiency with which the treatment is administered. Where a high death rate from gastroenteritis is seen as a technical problem, there is a temptation to attack it with technological weapons—hospitals, aerial evacuation, sophisticated fluid-replacement techniques and sophisticated drugs, because this is the quickest effective means of lowering the death rate. In fairness to health departments it must be stated that such activities are fostered or insisted upon by public and political pressures for quick results from applied technology. In terms of community health, however, the prevention of death from recurrent acute infections such as gastroenteritis is entirely symptomatic, however necessary it might be on humanitarian grounds. The real cause lies back in the community, not in the hospital bed, and it is in the community that the real solution must be found. Unfortunately it seems that relentless pressures in Australian society to save individual lives at all costs divert nearly all health and medical resources into the curative field. That this diversion of resources eventually may cause many more illnesses and deaths than it prevents (in the Aboriginal but not necessarily in the white population) tends to be ignored once the death rate appears to be under control.

A recent case in point is the agitation for improvements to the Alice Springs hospital in Central Australia in response to a sharp rise in the number of deaths of Aboriginal children in the area. The agitation appears to have come from within the medical and nursing professions and from outside them, as reported in the press. The incongruity of the solution proposed (upgrading facilities for curative technology) is understandable only if the death rate is seen as the problem. The death rate may be a technological and a political problem, but it is not the basic health or human problem.

It is very important, therefore, that cause-of-death statistics as well as fact-of-death statistics are looked upon as indices of the behaviour of people and not merely indices of the behaviour of diseases. It may be difficult to maintain this perspective in the face of a mass of figures in

which lists of mortal diseases play a prominent part; it is hoped that comparisons in death and disease rates between different populations may help to keep the figures in this study people-oriented. Most of the diseases of Aborigines are ubiquitous in terms of world health, so that there is little else but human ecology that can account for variations between populations.

SOURCES OF DATA ON ABORIGINAL MORTALITY

Mortality data for full-blood Aborigines come from two main sources—the number of deaths recorded for institutionalised or semi-institutionalised Aboriginal communities, and the causes of death as listed in Bureau of Census and Statistics records prior to 1967.[1] To the latter may be added a few special surveys undertaken by health departments, usually for limited areas or selected communities.

With the possible exception of the Northern Territory and Western Australia, there appears to be poor correspondence between the number of Aboriginal deaths reported and the lists of Aboriginal deaths by age, sex, and cause compiled by the Bureau of Census and Statistics. In the Northern Territory, records of Aboriginal numbers, births, and deaths were maintained by the Welfare Branch of the Northern Territory Administration, and for this and other reasons the population at risk, the numbers dying, and the causes of death are more easily associated.[2] For reasons which are not clear it has proved exceptionally difficult for the writer to obtain full access to such data. The only Welfare Branch data available to this study are those which have appeared in published reports or in other studies based on Welfare Branch data.

[1] Before the 1967 Referendum on the Australian Constitution, it was required that full-blood Aborigines not be numbered among the Australian population. Apart from omitting Aborigines from census figures, this requirement meant that Aboriginal births and deaths should not appear in the numerator of Australian vital statistics. To satisfy this requirement, the attempt was made to identify full-blood Aboriginal death notifications so that they could be individually subtracted from the total. Because Aborigines are not distinguished as such in death notifications, there were obvious handicaps to achieving total identification and subtraction. Nevertheless, lists of Aboriginal deaths derived in this manner were the only 'official' data for causes of death by age and sex on a State or Territory-wide basis. The figures were never published, partly because there was no constitutional requirement to do so, and partly, one believes, because of strong and justifiable doubts as to their accuracy and completeness. Since the 1967 Referendum, the need for separate identification of Aborigines in death records has disappeared along with the need to subtract their numbers.

[2] Other reasons include the fact that Aborigines and non-Aborigines are more easily distinguished by name and residence because of the relatively small non-Aboriginal population of the Territory and the physical segregation of the two groups.

For part-Aborigines, the number of deaths are available only for supervised communities, a limited number of which are covered from this aspect in various departmental Annual Reports. Cause-of-death data for New South Wales part-Aborigines have been gathered by the writer from personal research in the New South Wales Registers of Death for the period 1950 to 1964. Some additional figures are available from other surveys in various States.

Limitations imposed by the quantity, quality, or completeness of mortality and census data for full-blood and part-Aborigines usually preclude the calculation of death rates directly from the data: extrapolation and other manipulations have been necessary in nearly every case. Rates quoted are thus estimates, and an effort has been made to see that such estimates do not overstate the case (i.e. exaggerate death rates).

COMPARATIVE STATISTICS

Figures for Aborigines have been compared with equivalent figures for Australia (excluding full-blood Aborigines) in most cases, and also with those for Maoris and United States Indians—representing two other indigenous minorities—and other populations around the world showing a range of values for health indices better and worse than those for Aborigines. Among the latter, South African 'coloured' and 'asiatic' populations figure for the reason that, like the Maoris and United States Indians, they are well-documented statistically (in the United Nations' *Demographic Year Books*).

It is relatively easy to show Aboriginal health indices in an unfavourable light by comparing selected local or regional Aboriginal figures such as those for Central Australia with figures for the total indigenous population of another country such as Thailand or New Zealand. If the object is to rank Aboriginal statistical indices on a world-view basis, such comparisons are, of course, unfair in that sub-populations of other countries will have a very much lower ranking than the national average. Those with the lowest ranking of all are unlikely to appear in world statistical summaries because their poor health status is accompanied by poor or non-existent data retrieval. Furthermore, health indices based on the experience of a small community of Aborigines are subject to a greater degree of variation above and below a long-term mean for two reasons—the operation of chance (a greater standard error in statistical terms), and the irregular impact of epidemics, seasonal and other factors on small isolated communities.

Comparisons with overseas populations are thus offered as a perspective in Aboriginal health rather than as a means of ranking. Comparisons between Aboriginal populations will suggest ecological differences, as will comparisons with Australian averages, but in many cases the extent of the difference between two or more rates cannot be quantified accurately because of chance and other uncontrolled variations in a small population.

5 DEMOGRAPHIC AND MORTALITY PATTERNS IN FULL-BLOOD POPULATIONS

MORTALITY IN THE NORTHERN TERRITORY

CRUDE DEATH RATE

The crude death rate is not so much an index of mortality as it is an index of longevity and population increase. According to current world ecological concepts, a low crude death rate is not an ideal objective. A population of stable numbers, with no deaths occurring under the age of 75, would show a crude death rate of around 13 per 1,000 population. The addition of a 2 per cent infant mortality would raise this rate very slightly, to around 13·5. Higher rates may be the outcome of a reduced expectation of life or a birth rate too low to replace those dying. Lower rates imply a rising expectation of life and a high birth rate. A high crude death rate with a high birth rate implies a very much reduced expectation of life.

It is impossible to select a figure as representing the 'ideal' crude death rate without first specifying ideal birth rates and expectations of life. Table 3 therefore signifies more than differences in mortality, and is not necessarily a valid representation of Aboriginal health status on a world basis.

Except for selected institutionalised or specially-surveyed Aboriginal communities, crude death rates for full-blood Aborigines are not available for other States. The crude death rate quoted, together with separately-established estimates of age-distribution and the distribution of deaths by age and cause, may be used to estimate age- and cause-specific death rates for Northern Territory Aborigines. Rates may be estimated for

Table 3: *Crude death rates, Northern Territory Aborigines and others, c. 1959*

Population	Source of data	Crude death rate per 1,000	Year
N.T. Aborigines ('top end')	1	16·7	1957
		19·7	1958
N.T. Aborigines (total)	2	17·1	1957
		15·6	1958
		17·8	1959
		15·4	1960
	3	15·6	1958-60
Australia (excl. Aborigines)	4	8·8	1956-60
Northern Territory (excl. Aborigines)	4	5·3	1956-60
Europe	5	11	1956-60
Tropical Africa	5	23	1956-60
South-east Asia	5	21	1956-60
Central America	5	15	1956-60
Oceania	5	9	1956-60
New Zealand Maori	5	8·5	1959
U.S. Indian	6	9·0	1959-61
American Samoa	5	7·4	1955-9
Portuguese Timor	5	17·5	1955-9
South Africa (asiatic)*	5	8·1	1959
South Africa (coloured)*	5	15·2	1959
India	5	19·2	1958-9
Philippines	5	7·1	1960

* The terms 'asiatic' and 'coloured', as employed in the *U.N. Demographic Year* books, refer to Asian and part-'Bantu' respectively. No figures are given for the 'Bantu' population.

Sources:
1 Crotty and Webb, 1960 (deduced from figures supplied by the Northern Territory Welfare Branch).
2 Jones (1963).
3 Calculated from population and deaths given in Jones, 1963: 67, 91, 92.
4 Commonwealth Bureau of Census and Statistics, *Demography, 1963.*
5 United Nations, *Demographic Yearbook*, 1961.
6 United States Department of Health, Education and Welfare, Public Health Service, *Indian Health Highlights*, 1964, ed. p. 18.

other States only by assuming equivalent crude and age-specific death rates, and such an assumption would appear to be a reasonable one only in the cases of northern West Australia and northern Queensland. Because the Northern Territory provides the most comprehensive data available for full-blood Aborigines, it has been considered separately in this section.

AGE-SPECIFIC MORTALITY RATES

Table 4 gives the age-specific mortality rates for Northern Territory Aborigines, for Australia (excluding full-blood Aborigines), for New

Zealand Maori, and for South African 'asiatic' and 'coloured' populations, *c.* 1959. The mortality rates for Northern Territory Aborigines are based on data collected by Jones, partly re-organised into different age-groups corresponding to those used by Crotty and Webb in their 1960 study of cause of death. The mortality rates for the second year of life have been separated out in the 1-4 age-group because this is a year of exceptionally high risk for Aboriginal children. The sexes have been combined.

It should be noted that the under-1 mortality rate is not the same as the infant mortality rate, being based on the mid-year population rather than the number of live births.

Table 4: *Age-specific mortality rates for Northern Territory Aborigines and others, c. 1959, deaths per 1,000 population by age-group, both sexes*

Age-group	N.T.[1] Aborigines 1958–60	Australia[2] 1959	Maoris[3,4] 1958	U.S. Indians[5] 1959	South Africa 1958[6] Asiatic	Coloured
Under 1	142·0	22·0	52·9	56·6	70·6	155·2
1-	64·7	2·0	5·8	—	—	—
2-	12·5	1·2	2·2	—	—	—
3-	5·8	0·9	2·6	—	—	—
4-	4·8	0·7	1·4	—	—	—
1-4	21·6	1·2	3·0	3·8	8·1	23·4
5-9	2·3	0·5	1·2	1·2	1·5	1·9
10-19	2·6	0·6	1·3	1·6	1·3	1·7
20-29	4·5	1·1	3·0	4·5	2·2	4·2
30-39	4·8	1·7	4·7	6·6	3·4	6·0
40-59	13·5	6·6	15·8	9·9	12·4	14·8
60 and over	61·5	51·5	65·7	50·0	80·6	76·8

Sources:
[1] Calculated from data given by Jones (1963) together with additional data communicated personally by Jones.
[2] Calculated from data published in *Demography, 1959.*
[3,4] Calculated from data published in *Report of the Vital Statistics of New Zealand for the Year 1961* and in *Population Census 1961, Vol 8, Maori Population and Dwellings.*
[5] Calculated from original data supplied by the United States Division of Indian Health, from *Indian Health Highlights*, 1964 ed. and from *United States Census of Population 1960, Nonwhite Population by Race.* Rates synthesised for 24 Federal Reservation States.
[6] Calculated from data published in *United Nations Demographic Yearbook 1961.*

In Table 4, the American Indian rates may be slightly underestimated, as it was necessary to calculate the rates from the number of deaths in the twenty-four Federal Reservation States of the United States of America in 1959 divided by the age-group populations in 1960—the latter values determined by applying the published age-distribution percentages of a 25 per cent sample of the Indian population to the total given for the

twenty-four Federal Reservation States. It was not possible to calculate rates for single years of age from one to four for American Indians and the two South African minorities.

The figures show that the Northern Territory Aborigines in 1959 experienced a particularly high mortality in the first and second years of life, and also had a relatively high mortality in the third, fourth, and fifth years—and at all other age groups—when compared with Australia. The Aboriginal mortality rate in the first year was over six times the Australian rate, nearly three times the Maori and Indian rates, and twice the rates for the South African 'asiatic' population in the example given.

In the case of the second-year mortality rate, the Aboriginal figure was nearly half that for the first year. It was over thirty times the equivalent rate for Australia, and about twelve times the Maori rate. Although data were not available from which to compute this rate for the American Indian and South African populations, the high 1-4 rate for the South African 'coloured' population suggests that this population also experiences a high mortality in the second year of life.

At the time of writing, the figures quoted above are nearly twelve years old, and it may well be asked what the trends have been since. The writer has seen figures indicating a steep and progressive rise in under-5 mortality from 1957 through to 1966 (after which there was a fall). There is a possibility that the rise may be partly accounted for by an increase in the completeness of registration of deaths, but it seems unlikely that this could conceal a real fall or even a steady level in this rate. The trends in Aboriginal mortality must remain unnecessarily obscure until such time as the relevant authorities collate and publish all the data that is being collected—if necessary with comments on probable biases and errors.

NORTHERN TERRITORY INFANT MORTALITY

It is apparent from the figures that have been derived so far that death is a major hazard to infants and young children in the Northern Territory Aboriginal population, and that death occurring after the age of 5 years and before the age of 60 years is not an exceptional hazard, either in terms of the proportion of such people who die each year or in terms of the total numbers who die in this age-group.

Thus the defects in the collection and availability of data for the deaths of Aborigines over the age of 5 years are not so serious as they might be otherwise. In contrast, the deaths occurring to young children are seen to warrant as much detailed attention as can be given them. Changes in the pattern of events occurring to the very young would seem to hold

the greatest portents of major changes in demographic, economic, and perhaps social status in the near and distant future.

Statistics concerning the death of infants can be based on the numbers of births and infant deaths without any knowledge of the age-sex structure of the population. In this section an attempt has been made to extend the principle behind the calculation of infant mortality into the second year of life. The relevant data is much more easily obtainable than in the case of age-specific mortality, and the statistics that are produced are just as comparable between populations—with a few minor reservations.

There are two possible methods of calculation of second-year mortality rates based on live births: firstly, as a rate per 1,000 live births in the previous year; and secondly, as a rate per 1,000 estimated survivors from the previous year—live births minus infant deaths. With the second method, the rate will approximate an age-specific rate based on a population census.

In spite of the apparent advantages of the second method, it has seemed preferable to calculate a rate based on the previous year's live births, and to tabulate this beside the infant mortality rate for the same cohort in the previous year. Since the denominator (population base) for both infant and second-year mortality rates will be the same live births, the rates can be added together to give the rate for the first two years of life, per 1,000 live births. The rates calculated as above for the second year will tend to be considerably lower than the age-specific mortality rate for the same age-group because the denominator for the latter will have been reduced to survivors only—in the case of the Northern Territory Aborigines the survivors of a high infant mortality. Similarly, the infant mortality rate will be lower than the under-1 year age-specific rate, based on the survivors from early infancy when the majority of infant deaths occur. Provided that the numbers of births and infant deaths in each year remain reasonably steady—or increase or decrease with a steady trend—the infant and second-year mortality rates based on live births will give a more accurate picture of the risks of death faced by a new-born baby than the rates based on mid-year populations.

Table 5 shows the Northern Territory Aboriginal infant mortality rates for two periods—1958-60 and 1965-8.

There is evidence that the infant mortality rates shown in Table 5 for 1958-60 were overstated by the under-registration of live births in the figures available to Jones up to 1962. Figures sighted by the writer showing births occurring in 1958-60 but registered later (between 1962 and 1965) suggest that only 80 per cent had been registered by 1961. If there were

Table 5: *Infant mortality rates, Northern Territory Aborigines*

Year	Live births	Infant deaths	Infant mortality per 1,000 live births
1958	481	71	148
1959	527	58	110
1960	521	90	173
1958-60[1]			143
1965	743	107	144
1966	825	123	149
1967	860	87	101
1968	927	75	81
1965-68[2]			117

Sources:
[1] Jones (1963): 76, 91, 92.
[2] Northern Territory Medical Service, personal communication, 1968.

no late-registered infant deaths for the same period, the infant mortality rate in 1958-60 may be estimated to have been about 112 per 1,000 live births instead of 143 as shown. It is likely that the unregistered births discovered up to five years after the event would more often relate to surviving infants than to those who had subsequently died in infancy or early childhood, so that the inclusion of late registrations would understate the infant mortality rate at 112 per 1,000. It is doubtful if the exact figure can ever be known. Increased accuracy of future estimations can be expected with earlier and more complete registration, but until such time as birth and infant death registration is immediate as well as complete the trends in Aboriginal infant mortality will not be accurately reflected in published figures.

Table 6 shows the infant and second-year mortality rates for Northern Territory Aborigines by various groupings in 1965-7, and equivalent rates for other populations.

The figures shown in Tables 5 and 6 need little additional comment. The rates for the first two years of life, when based on the live births, show the same order of differences from Australian and Maori rates as was noted with those based on mid-year populations in these years of age. The figures suggest that, for the total Aboriginal population of the Northern Territory, 13 per cent of live-born babies had died before reaching one year, and another 4 per cent died in their second year, in 1965-7. Seventeen per cent, or one baby in six, failed to survive to the age of 2 years.

Table 6: *Infant and second-year mortality rates for Northern Territory Aborigines, by various groupings, and others*

	Mortality rates per 1,000 live births			
	Years	Infant	2nd year*	Under 2 years
Total full-blood population	1965-7[1]	130	40	170
Northern Division	1965-7[1]	110	30	140
Southern Division	1965-7[1]	168	47	215
Total missions	1965-7[1]	97	22	119
Total settlements	1965-7[1]	161	41	202
Total pastoral properties and others	1965-7[1]	144	63	207
Australia (excluding full-blood Aborigines)	1958-60[2]	21	2	23
New Zealand Maori	1958-60[3]	51	6	57
American Indian	1958-60[4]	47	†	†

* Calculated as deaths per 1,000 live births in the previous year(s).
† Not calculable from available data.

Sources:
[1] Northern Territory Medical Service (personal communication, 1968).
[2] Australia, *Demography 1958, 1960, 1961.*
[3] New Zealand, *Report of the Vital Statistics of New Zealand for the year(s) 1958, 1959, 1960, 1961.*
[4] United States Department of Health, Education and Welfare, *Indicators; Indian Poverty and Indian Health* (1964).

In the affluent developed societies, the combined effects of a high standard of living and medical technology have brought about a reduction of the infant mortality to just under 2 per cent. The risk of death in the second year, for the 98 per cent who survive the first, is negligible—around 0·2 per cent in Australia. For this reason, the Northern Territory Aboriginal second-year death rate of 4 per cent of live births signifies a greater short-fall in child care than the infant mortality rate of 13 per cent. It immediately suggests a nutritional problem, for similar patterns of high second-year death rates occur in developing countries because of—or contributed to by—nutritional deficiencies in the weaning period. As will be shown in the next section, malnutrition does not figure prominently in the causes of death in this age-group, but bearing in mind the remarks made earlier about medical custom in death certification this does not rule out malnutrition as a major underlying cause in Aboriginal childhood mortality.

Table 6 shows the variations in infant and second-year mortality between some geographic and administrative groupings of Northern Territory Aborigines. Rates were higher in Central Australia than in the northern division of the Northern Territory, and higher in government settlements and on cattle stations than at mission settlements.

CAUSE OF DEATH IN THE NORTHERN TERRITORY ABORIGINAL POPULATION

The first evidence to be examined is that contained in Crotty and Webb's study published in 1960. This was an analysis of 354 deaths registered between July 1956 and March 1959, excluding a few intervening months. The data were drawn from the population of the northern part of the Northern Territory. No population base for this study is quoted or has been obtainable from sources other than Crotty and Webb's estimate of about 10,400, from which to calculate rates.

The authors were satisfied as to the accuracy of certification in only half of the 354 deaths they examined. In 108 cases the diagnosis was established at autopsy, and in another 67 cases the cause was reasonably certain without autopsy. The distribution of causes—actual or as certified—is unknown for the remaining 179 cases, but in a study of mortality patterns these cannot, of course, be excluded without giving a very biased picture. Including them here has had the result that the commonest cause of death at all ages is 'undiagnosed', so that the proportion dying from specified causes is the minimum. If these proportions are used—via estimated age-specific death rates—as a basis for estimating cause-specific rates, the rates again are minimum and therefore not properly comparable with rates from other populations.

Crotty and Webb believed that deaths were under-registered to an unknown extent. They also experienced the problem caused by selection of cases leading to hospitalisation in Darwin. They stated:

> The selection of the material favours the series being loaded with aborigines living around Darwin—young but not new-born children, and young adults especially males—and with easily diagnosed diseases in those that do not come to autopsy. (Crotty and Webb, 1960:492)

The authors do not tabulate their data by sex. However, with the sexes combined it is possible to compare the age-group proportions with the same age-group proportions in the deaths listed by Jones (1963) for the whole Territory in 1958-60. There is not a very close agreement in these two sets of proportions, particularly in the under-5 age-group where Crotty and Webb's data appear to be deficient. This makes it likely that cause-specific death rates by age calculated by distributing Crotty and Webb's causes of death within age-groups among Jones's death rates for the same age-groups (for the whole Territory) would be a poor reflection of cause-specific rates in the northern division of the Northern Territory. A better estimate is likely if Crotty and Webb's

northern population of 10,400 is divided into age-groups according to Jones's proportions for the whole Territory, and using the resultant figures as the denominators for Crotty and Webb's deaths by cause. The reasoning here is admittedly difficult to follow; it may be rephrased in saying that it has appeared preferable to work from Jones's age-distribution for the population of the Territory rather than from his age-specific death rates for the Territory, on the assumption that the former are more likely to agree with the figures for the northern division taken alone.

On this basis, and using as the numerator Crotty and Webb's data averaged for one year (i.e. deaths × 12 ÷ 26, as the data were collected over 26 months) the results are an estimate of the age-specific death rates for this northern division Aboriginal population.[1] These results are shown in Table 7.

Table 7: *Age-specific death rates for N.T. Aborigines and Maoris, per 1,000, c. 1959*

Age-group	Northern N.T. Aboriginal	Maori	Australia
Under 12 months	92	53	22
12 months and under 24	42	6	2
2-4	8	3	1
5-9	3	0·7	0·4
10-19	2	1	0·6
20-29	5	2·5	1
30-39	12	5	2
40-59	18	16	7
60 and over	60	66	51
All ages (crude death rate)	16	8·5	8·8

These rates are a little lower in the younger age-groups than those given by Jones for the whole Territory, and it would be reasonable to assume that they were minimum rates for the under-5 population from which Crotty and Webb drew their data. The numbers in the age-groups from age 5 to age 29 are so small that an attempt to further subdivide them by causes would be completely unrealistic. Omitting these age-groups, Table 8 gives the proportions and estimated rates by leading causes of death for the northern division Aboriginal population, together with equivalent proportions and rates for Maoris and Australia.

[1] That one has to go to such lengths to compute death rates reflects the poor quality of basic data available, even for the Northern Territory. For other States the situation is even worse, so that the attempt at estimation by devious means has seemed justified in this case. It is only the fortunate coincidence in time of Jones's and Crotty and Webb's studies in the Northern Territory that has made any sort of estimate of age- and cause-specific death rates possible.

Taking into account the certainty that if the undiagnosed deaths were distributed according to their actual cause, at least some of them would be added to the leading causes, the rates for specific causes are therefore absolute minima in every sense, but it can be seen that even with this indirect method of calculating rates from probably biased data there are some interesting conclusions to be drawn.

The rates for gastroenteritis and pneumonia/acute respiratory infection are very high, both absolutely and relative to Australia and Maoris. The minimum gastroenteritis death rate in the second year of life is suggested to have been over 150 times higher in the Aboriginal group than it is in Australia, and over thirty times the Maori rate.

The pulmonary tuberculosis death rate from the age of 30 onwards was very high by Australian standards, and the death rate from cardiovascular disease in the age-group 60 and over relatively low. However, in the case of comparatively low rates, particularly in this age-group where nearly 70 per cent of deaths were undiagnosed as to cause, the fact that the Aboriginal figure was an absolute minimum means that the latter observation is of no particular significance. If half the undiagnosed deaths were allotted to the category of cardiovascular disease, the rates would be comparable with those for Maoris and Australia. The same applies to the deaths from malignant neoplasms in the 40-59 age-group. Interestingly, this rate was very much higher in the Maori population than in Australia. The high rate for malignant disease in Aborigines in the 30-39 age-group is not of great significance as it arises from only three deaths.

As expected, the figures confirm both by proportion and by estimated age- and cause-specific rate that the high infant and childhood mortality is mostly due to bowel and respiratory infections (as 'primary causes').

Crotty and Webb, having access to clinical and autopsy data not tabulated in their study, have made some useful comments in connection with specific diseases which may be summarised as follows:

Tuberculosis. Did not appear in the list of causes of death until after the age of 20, and after this age caused almost one-fifth of the deaths. Its absence as a cause of death under 20 may be connected with extensive B.C.G. immunisation campaigns since 1951, although glandular (lymph node) tuberculosis is common in Aboriginal children. Most of the tuberculosis in the series was a pulmonary infection.

Malnutrition and anaemia with diarrhoea or pneumonia. This group caused 44 out of 64 diagnosed deaths under 5 years. The common clinical picture was of a child between 6 and 30 months, often underweight, severely

Table 8: *Common causes of death for selected age-groups, both sexes, by proportion and estimated rates, for northern Aborigines, Northern Territory, with comparative proportions and rates for Maoris and Australia, c. 1959 (rates per 100,000)*

Age-group	Cause of death	Northern N.T. Aborigines[1]			Maoris[2]		Australia[3]	
		Deaths	%	Estimated* mortality rate	%	mortality rate	%	mortality rate
Under 12 months	All causes	71	100	9,200	100·0	5,290	100·0	2,200
	Undiagnosed	35	49	4,500	0·0	—	0·3	7
	Gastroenteritis†	12	17	1,500	10·1	534	2·3	51
	Acute respiratory infection‡	11	15	1,400	8·2	434	8·2	180
12 months and under 24 months	All causes	29	100	4,200	100·0	580	100·0	200
	Undiagnosed	10	34	1,400	0·0	—	0·7	11
	Gastroenteritis†	13	45	1,900	10·8	63	6·2	12
2-4	All causes	16	100	800	100·0	320	100·0	91
	Undiagnosed	7	44	350	0·0	—	0·9	1
	Pneumonia	3	19	150	12·9	41	9·3	8
	Gastroenteritis†	2	12	100	4·8	15	4·2	4
	Age-groups 5-29 omitted							

30–39	All causes	36	100	1,200	100·0	470	100·0	170
	Undiagnosed	15	42	500	0·0	—	0·3	5
	Malignant neoplasms	3	8	100	7·7	36	18·6	32
	Pulmonary tuberculosis	1	3	50	5·7	27	1·2	2
40–59	All causes	67	100	1,800	100·0	1,580	100·0	660
	Undiagnosed	34	51	900	0·0	—	0·2	1
	All respiratory diseases	10	15	270	2·9	46	4·9	32
	Pulmonary tuberculosis	6	9	160	5·8	92	1·1	7
	Malignant neoplasms	3	4	70	15·7	248	20·9	138
60 and over	All causes	103	100	6,000	100·0	6,570	100·0	5,110
	Undiagnosed	70	68	4,080	0·0	—	0·1	5
	Pulmonary tuberculosis	6	6	360	5·5	361	0·5	25
	All respiratory diseases	16	15	900	17·5	1,150	8·0	409
	Disorder circulatory system	9	9	540	44·8	2,943	45·9	2,346

* Rates have been rounded.
† Gastroenteritis and dysentery (bacillary and unspecified) combined.
‡ Pneumonia and influenza.

Sources:
[1] See text and Table 7.
[2] See Table 7, and New Zealand, *Medical Statistics of New Zealand 1959*.
[3] See Table 7.

Notes:
The commonest causes of death as listed relate to the commonest causes of *Aboriginal* deaths.
Age groups between 5 and 29 have been omitted because of very small numbers in the Aboriginal data.

anaemic, pot-bellied, a large fatty liver, with discoloured lifeless skin and hair, presenting with dysentery or pneumonia. In these children, malnutrition and/or anaemia is usually mentioned as a secondary (contributing) cause of death, and does not appear in the tabulations according to primary cause.

Malignant disease. The authors surmise that the true incidence may be higher than it appears due to elderly sufferers dying undiagnosed because they are afraid to leave their tribal territory and possibly die in hospital. One would agree that this is most likely.

Circulatory disease. The authors state that severe arterio-sclerosis is not uncommonly found at autopsy.

Chronic bronchitis and emphysema. Although only responsible for less than a quarter of deaths over age 60 (as a primary cause), these conditions are commonly present in elderly Aborigines coming to autopsy.

Accidents and violence. These conditions caused one-tenth of all deaths with established causes. (They are not listed among the common causes at various ages in Table 8 because they are scattered through all age groups.) Ten out of seventeen of these deaths were the result of brawls and fights.

Finally the authors comment that there were three major epidemics during the period of the study—an influenza epidemic in late 1956 and early 1957 (probably Type A); influenza in July-August 1957 (Asian); and an undiagnosed epidemic towards the end of 1958 characterised by diarrhoea in children and a respiratory tract infection in adults. The authors indicate in a diagram that the epidemics corresponded with peaks in the numbers of total and infant deaths.

The estimated rates for leading causes of death in Crotty and Webb's series, when compared with those for Maoris, American Indians, South African 'coloured' and 'asiatics' and Australia, show that, not counting the 'undiagnosed' group, only the South African 'coloured' population had generally higher rates.[2] The chief exception was in deaths due to homicide and manslaughter, in which the Aboriginal population topped the scale at 45 deaths per 100,000, the next highest figure being 14 per 100,000 for American Indians and South African coloureds. A very high figure for deaths due to accidents in the Indian population was due very largely to deaths from motor vehicle accidents—an insignificant hazard in the Northern Territory environment.

The other body of data on Northern Territory Aboriginal deaths

[2] The comparative figures are not included in this study.

consists of figures compiled with the assistance of the Commonwealth Bureau of Census and Statistics, and is derived from the individual coded deaths of 700 full-blood Aborigines during the years 1964 and 1965.

Figures obtained from the Welfare Branch indicate that they registered 463 Aboriginal deaths in the Northern Territory in 1964, but the Bureau of Census and Statistics data amount to only 364 deaths in 1964, so the latter data appear to account for only four-fifths of the total. To what extent the other 100 deaths have been selectively excluded it is impossible to say, but again it throws some doubt on the validity of detailed rates computed from the data.

Population figures by age and sex are available only for 1961 and 1966, and having been derived in different ways (because of differences in the census schedules) it would be difficult to interpolate figures for 1964 and 1965. Furthermore, a projection forward from 1961 is handicapped by evidence (not included in this study) for an unusually high childhood mortality in 1962. In the absence of detailed data on deaths from 1961 onwards, by single years of age, the availability of data on the numbers of births does not allow the accurate estimation of age-group numbers in 1964 and 1965, particularly for the important childhood age-groups.

With the 1964-5 death data, no attempt has been made to derive age- and cause-specific death rates. Instead, the data have been examined in two other ways—as leading causes of death for all ages by number, proportion and an estimated all-ages rate; and as leading causes of death by age-group by number and proportion only. Accepting that Aboriginal death rates are high at all ages, the analysis as outlined directs attention towards those diagnostic groupings which are of particular importance. As has been suggested already, the figures do not indicate what action may be required except in broad terms, but they are a useful base-line from which to observe trends and the effects of health and welfare activities.

Table 9 lists the leading causes of death, all ages, for 1964-5. The rates quoted have had to be based on an estimated crude death rate of 21 per 1,000. The crude death rate for 1964 has been calculated at 22·5, and the writer has no data to calculate it for 1965. It is possible that the figure of 21 per 1,000 is too high, but a reduction to 19, for instance, would have relatively little effect on the rates given (for instance, it would reduce a rate of 370 to 340, but would not materially alter a rate of 70 per 100,000).

Table 10 compares the same figures with those for Maoris, American Indians, and Australia. With the exception of deaths from heart disease, and vascular lesions of the central nervous system ('strokes'), the Abori-

ginal rates were higher than the highest in any of the populations listed. With deaths from accidents, the Aboriginal rate was exceeded only by that for American Indians. With malignant neoplasms, the Aboriginal rate was exceeded by that for Australia only.

Table 9: *Leading causes of death, Northern Territory Aborigines, 1964 and 1965, all ages*

Cause	International Classification of Diseases Code Groups	Number of deaths (2 years)	% of total deaths	Estimated mortality rate per 100,000 per annum
All causes	(001-E999)	700	100·0	2,100*
Pneumonia and influenza	(480-493)	125	17·9	370
Senility and ill-defined or unknown	(794,795)	121	17·3	360
Certain diseases of early infancy	(760-776)	76	10·9	230
Gastroenteritis and dysentery	(045-048, 571)	75	10·7	220
Heart disease	(400-443)	42	6·0	130
Accidents	(E800-E962)	36	5·1	110
Malignant neoplasms	(140-205)	28	4·0	80
Anaemias and malnutrition	(280-287, 290-293)	25	3·6	70
Tuberculosis	(001-008)	25	3·6	70
Vascular lesions of C.N.S.	(330-334)	11	1·6	30

* Estimated mortality rates based on estimated crude death rate of 21 per 1,000 for 1964 and 1965. The crude death rate calculated for 1964 was 22·5. A figure for 1965 cannot be calculated from data available to the writer.

Source: Author's research assisted by the Commonwealth Bureau of Census and Statistics.

In the 1964-5 series, deaths due to 'senility, and ill-defined and unknown causes' still amounted to 17 per cent of total deaths, and although this was considerably less than the percentage in the 1956-9 series, when it was 50 per cent, it still implies that many of the rates given for Aborigines were minimal.

The age-distribution of the Aboriginal deaths, by age-group, is shown in Table 11, together with percentages for the same age-groups for Maoris, American Indians, and Australia for comparison. These figures again indicate the comparatively high proportion of deaths in the Aboriginal population among children under the age of 5 years.

The median age at death (the age at which half the deaths occur at younger ages, and half at older ages) was approximately 25 years for Northern Territory Aborigines, 40 years for Maoris, 45 years for American Indians and 71 years for Australia. In other words, a Northern Territory

Table 10: *Leading causes of death, all ages, Northern Territory Aborigines 1964 and 1965, compared with Maoris 1964, United States Indians 1961-3, and Australia 1963, by percentage of total deaths and rates per 100,000*

Cause	Aborigines[1]		Maoris[2]		U.S. Indian[3]		Australia[4]	
	%	Rate	%	Rate	%	Rate	%	Rate
All causes	100	2,100	100	620	100	900	100	870
Pneumonia and influenza	18	370	9	55	9	77	3	29
Senility and ill-defined, etc.	17	360	0·5	0·5	?	?	0·7	6
Diseases of infancy	11	230	8	50	8	68	3	25
Gastroenteritis and dysentery	11	220	2	14	3	27	0·4	4
Heart disease	6	130	24	151	16	142	37	322
Accidents	5	110	10	63	17	156	6	49
Malignant neoplasms	4	80	12	72	8	70	16	135
Anaemia and malnutrition	4	70	0·3	0·2	?	?	0·3	2
Tuberculosis (all forms)	4	70	2	15	3	25	0·5	4
Vascular lesions of C.N.S.	2	30	6	37	6	50	13	115

Sources:
[1] See Table 9.
[2] New Zealand, *Medical Statistics Report: Part 1—Mortality and Demographic data 1964.*
[3] United States, *Indian Health Highlights*, 1964 ed.
[4] Australia, *Demography 1963*, Bulletin No. 81.

Table 11: *Percentage of deaths by age-group, Northern Territory Aborigines 1964-5, Maoris 1964, American Indians 1960, and Australia 1963*

Age-group	Aborigines[1]		Maoris[2]	U.S. Indians[3]	Australia[4]
	Number	%	%	%*	%
0-1	220	31·4	20·5	22·0	4·8
1-2	63	9·0	2·8	—	0·4
2-4	28	4·0	2·4	—	0·5
1-4	91	13·0	5·2	5·3	0·9
5-9	10	1·4	2·0	1·6	0·5
10-19	16	2·3	3·6	3·3	1·2
20-29	30	4·3	4·5	6·2	1·7
30-39	37	5·3	5·2	7·1	2·6
40-49	49	7·0	10·1	8·9	5·8
50-59	42	6·0	15·0	9·3	11·6
60 and over	201	28·7	34·0	36·3	70·9
All ages number	700†		1,168	4,111	94,894

* For 24 Federal Reservation States (excludes Alaska and non-Reservation States).
† Total includes four deaths at unknown age.
Sources:
[1] As for Table 10.
[2] As for Table 10.
[3] United States, Division of Indian Health, personal communication.
[4] As for Table 10.

Aboriginal baby at birth had once chance in two of dying before the age of 25 if present death rates remained unaltered, whereas an average Australian baby has once chance in two of dying before the age of seventy-one.

Alternatively, this may be expressed as an Aboriginal baby at birth having a 50 per cent chance of living beyond the age of 25, while for a white Australian there is a 50 per cent chance of living beyond 71. The comparison is not strictly valid in that it does not take into account effects of different birth rates and age structure, and different rates of change in health status, but the premises on which it is based are the same as those used in the calculation of expectation of life at birth from detailed age-specific mortality figures.

Table 12 shows the leading causes of death in the first, second, and third-to-fifth years, and in the total 0-4 age-group from the 1964-5 data expressed as numbers and percentages within age-groups. No attempt has been made here to estimate rates—for reasons already explained—but the proportions may be compared with those in Table 8, which also includes estimated rates.

Table 12 shows that in the first year of life pneumonia and gastro-enteritis are the most common single causes of death, each contributing just over a fifth of all deaths.[3] Immaturity (i.e. prematurity of infants) was responsible for another one-eighth of infant deaths. The grouped cause 'Congenital Malformations' contributed just under 4 per cent. The largest grouped cause in this age-group was 'Certain Diseases of Death in Early Infancy' (a group which has recently been retitled 'Certain Causes of Perinatal Morbidity and Mortality'). The latter group, which includes 'Immaturity', was held responsible for a third of deaths under one year of age.

In the second year, already noted to have an exceptionally high mortality, the commonest causes of death were gastroenteritis (a third of all deaths), then pneumonia and malnutrition/anaemia, each responsible for about one-fifth of the deaths.

In the 2-4 age-group, the relatively fewer deaths limited the listing of

[3] 'Single causes' refers to individual disease entities, or small groups of diseases that have a similar aetiology and clinical effect. 'Grouped diseases' refers to the broadest groupings in the International Classification of Diseases: there are seventeen broad groups, of which some are loosely based on 'cause' (e.g. Infectious and Parasitic Diseases, Congenital Malformations, Accidents by External Cause), some are based on the bodily system affected (e.g. Mental Disease, Cardiovascular Disease, Diseases of the Digestive System), and some refer to selected groups in the population (e.g. Diseases of Pregnancy, Childbirth and the Puerperium, Certain Causes of Perinatal Morbidity and Mortality).

Table 12: *Leading causes of death in infancy and childhood, Northern Territory Aborigines, 1964-5*

Age-group	Cause	Cause of death code group	Number of deaths	% of deaths in age group
Under 12 months	All causes	(001-E999)	220	100·0
	Single causes:			
	Pneumonia	(490-493)	50	22·7
	Gastroenteritis (4 weeks and over) and bacillary dysentery	(045, 571)	49	22·3
	Immaturity unqualified	(776)	27	12·3
	Ill-defined diseases of infancy	(773)	12	5·5
	Nutritional maladjustment	(772)	7	3·2
	Grouped causes:			
	Diseases peculiar to infancy	(760-776)	76	34·6
	Congenital malformations	(750-759)	8	3·6
	Infectious and parasitic diseases	(001-138)	8	3·6
12 months and under 24 months	All causes	(001-E999)	63	100·0
	Gastroenteritis and bacillary dysentery	(045, 571)	22	34·9
	Pneumonia	(490-493)	12	19·0
	Malnutrition and anaemia	(286, 293)	13	20·6
2-4	All causes	(001-E999)	28	100·0
	Pneumonia	(490-493)	7	25·0
	Gastroenteritis and bacillary dysentery	(045, 571)	3	10·7
Total 0-4	All causes	(001-E999)	311	100·0
	Single causes:			
	Gastroenteritis and bacillary dysentery	(045, 571)	74	23·8
	Pneumonia	(490-493)	69	22·2
	Immaturity unqualified	(776)	27	8·7
	Malnutrition and anaemia	(286, 293)	20	6·4
	Grouped causes:			
	Diseases peculiar to infancy	(760-776)	76	24·4
	Infectious and parasitic diseases	(001-138)	18	5·8

Source: Author's research assisted by Commonwealth Bureau of Census and Statistics.

leading causes to two—pneumonia (a quarter of the deaths) and gastroenteritis (a tenth).

Within the whole 0-4 age-group, gastroenteritis and 'Certain Diseases of Early Infancy' shared the lead as causes (24 per cent and 22 per cent respectively).

It can be estimated from the figures that those who survive their fifth birthday have a better than 50 per cent chance of surviving their fiftieth. There is another index of health which can be derived from

mortality data—the Proportional Mortality Ratio at Age 50 and Over—which merits consideration, but it is not a familiar health index to most people and a preliminary discussion of its derivation and significance is necessary.

PROPORTIONAL MORTALITY RATIO—AGE 50 AND OVER

In 1957, Swaroop and Uemura published an account of their investigation into the various rates and ratios which could be derived from commonly collected data on population and mortality. Their intention was to test these rates and ratios mathematically to see which had the highest discriminatory power to distinguish between and rank populations with various levels of 'health, including demographic conditions'.

Selecting two groups of countries which had, on *a priori* grounds, the best and the worst health respectively, the authors tested the discriminatory powers of the crude death rate, the infant mortality rate, the expectation of life at age 1, and the proportion of deaths under age 5. They also tested all the possible combinations of these indices. They then tested the proportional mortality ratios of deaths under 1, under 5, and so on by five-year intervals to age 70, and several other indices including the mean age of death and the expectation of life at birth.

The basic assumption made for the purposes of this analysis was that countries such as New Zealand, Australia, Canada, England and Wales, the United States of America, and the Scandinavian nations were at the top of the health scale, and that countries such as Ceylon, Colombia, Egypt, India, Thailand, the Philippines, and Peru were at the bottom. It is possible to argue against this assumption on some grounds (for instance, is it 'healthy' to have a large and increasing number of aged-and-infirm in a population?) but in general it would seem that the health and demographic conditions of the 'healthy' populations are the goals of the 'unhealthy' ones.

A most interesting result of this analysis was the relatively poor performance of the traditional health indices, particularly the crude death rate. It was found that the proportional mortality under age 50 was by far the most sensitive to differences between the two groups of populations, and far exceeded in sensitivity any of the other indices, either singly or in any combination. Because of its sensitivity it should, in theory, be ideal for ranking populations according to how nearly they approach the 'ideal' Western standards of health.

The figure derived for the proportional mortality under 50 varies inversely with health status—countries with higher values have poorer

health. The proportional mortality at age 50 and over (hereafter abbreviated as PMR(50+)) varies directly with health status and has the same discriminatory power, and was therefore suggested by Swaroop and Uemura to be the most appropriate index of 'health, including demographic conditions'.

Apart from its sensitivity, a great advantage of this index is the ease with which it can be determined—the only data required are the deaths occurring in a population divided according to whether they occurred under age 50 or at 50 and over. The authors claimed that the index is not affected by more than a few units as a result of mis-statement of age in the vicinity of 50, of under-registration of deaths in general, or of the relatively greater under-registration of infant deaths by up to 20 per cent.

The value of this index to the present study lies in its ability to rank Aboriginal populations in the absence of accurate census or age-specific mortality data. The main reservations as to its validity arise from the likelihood that many Aboriginal communities from which the data might come are already selected to some extent by age, economic status or other determining factors—particularly in the part-Aboriginal population. However, this is not a serious handicap provided the type of community providing the data is specified.

Figure 1 shows the PMR(50+) for Northern Territory Aborigines in 1958-60 (based on Jones, 1963), compared with other selected populations around this time. It indicates that in general terms Northern Territory Aborigines ranked with the underdeveloped countries, as would be expected. It should be noted that there is a considerable jump in the PMR(50+) from Fiji to the developed populations as represented by Australia and New Zealand. Swaroop and Uemura's original study contains a table in which this gap is filled by the range of values for the populations of eastern Europe, southern Europe, Japan, Argentina, and Uruguay. This suggests that the ranking of a population's PMR(50+) follows its ranking according to economic, social, and educational factors, and is not merely a function of race, health facilities, and the physical environment. The implications as far as the Aborigines are concerned are that movement up the scale of values is unlikely to occur through attention to physical health alone.

The Northern Territory Aboriginal PMR(50+) from Jones's data *c.* 1958-60 was 35·4. For the 700 deaths recorded for the years 1964-5 it was 34·7. Most populations studied by Swaroop and Uemura (except India) showed a fairly steady rise in the PMR(50+), particularly over the ten years from 1945 to 1955 (beyond which data was not available to

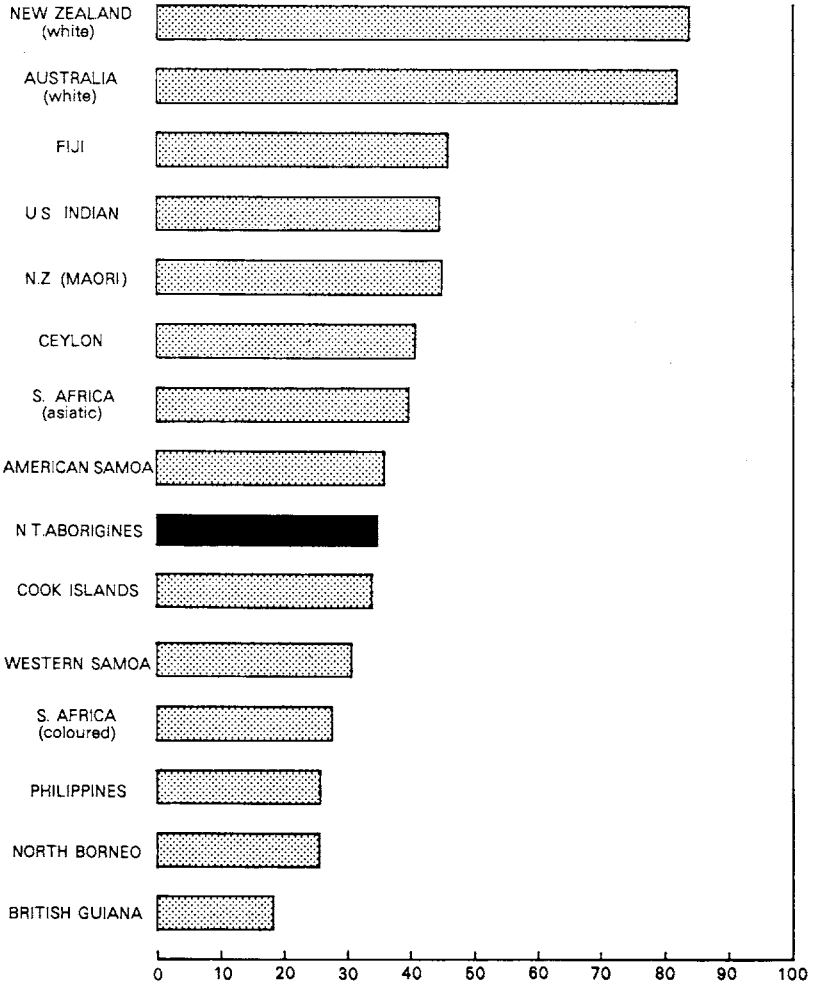

Fig. 1. Proportional mortality ratios age 50 and over

them). It is possible that no such rise is occurring in the Northern Territory Aboriginal population—the figures suggest a slight fall—implying that the gap between Aborigines and whites in 'health, including demographic conditions' may be widening.

The patterns of mortality in the Northern Territory Aboriginal population have been described in some detail, the intention being to construct a mortality model with which other Aboriginal populations may be compared. Before looking at the rather more meagre data for Western Australia and Queensland full-blood Aborigines, it has seemed desirable ro review the Northern Territory data in conjunction with some demographic observations which have not been presented so far (or which may be found in other publications), and in doing so round out the health ecology of this population to a greater extent than is possible with mortality indices alone. The more remote implications of the Aboriginal mortality data will be discussed with the morbidity and other relevant information after the latter has been presented.

AN ECOLOGICAL REVIEW OF NORTHERN TERRITORY DEMOGRAPHIC AND MORTALITY PATTERNS

The ecological criteria by which a human society may be evaluated are basically those by which any biological population may be evaluated, whether bacteria, fish, a forest, or flock of birds. Is it adapted successfully to its environment and to changes in its environment? Is it competing or associating successfully with other life forms occupying the same area? Is it making the most economical use of its internal and external resources? For a human society, the additional question may be asked—does it have control over its destiny?

For the Aboriginal population of Australia prior to 1788 it is probable that the answer to all these questions was 'yes'. With the advent of white settlement, the answer to the last question immediately became 'no', and this altered the answers to the other questions. For the Aboriginal population in 1971 it would seem that the answer is a qualified 'yes' in some instances only.

From the demographic and mortality patterns of the Northern Territory Aborigines, the following is of ecological importance:

(i) Northern Territory full-blood Aborigines make up about 45 per cent of the total full-blood population of Australia, and number about 21,000. The Northern Territory has the smallest proportion of part-Aborigines to total Aborigines for all States; about 20 per cent. Full-blood Aborigines contribute about 36 per cent of the total population of the Territory—the highest figure for any State.

(ii) The total numbers appear to have declined slightly from the earliest estimates in the 1920s through to the 1940s, but are now increasing again.

(iii) Northern Territory Aborigines occupy a variety of physical environments from the arid semi-desert southern part to the tropical coastline of the north. In between there is a belt of savannah woodland-type cattle country, almost entirely taken over by the pastoral industry. They also occupy a variety of what may be termed administrative environments, from the welfare-oriented missions and settlements, through semi-paternalistic cattle stations to the independence of reserves and fringe-camps. A little over a quarter of the total live in each of the environments classed as settlement, mission, and cattle station, and the balance in towns, on reserves, or elsewhere. Less than 2 per cent were classified as town dwellers in 1958; this had risen to 13 per cent by 1966.

(iv) The Aboriginal population is clustered in small aggregates, many of these being in very isolated situations.

(v) The population pyramid is not particularly broad-based—the population is 'older', for instance, than the Maori, American Indian, or most South-east Asian populations. The proportion under the age of 15, in 1961, was 39 per cent, as against 49 per cent for Maoris, 42 per cent for American Indians, 46 per cent for the Philippines, and 33 per cent for Australia. The Aboriginal figure had risen to only 40 per cent by 1966.

(vi) The relatively 'old' population is associated with a relatively low fertility rate on world and other Aboriginal standards. According to Jones's estimate, the average number of births experienced by an Aboriginal woman throughout the childbearing period was 4·25 in 1958-60, and between 5·16 and 5·66 in 1961 (the latter being a re-estimate from improved data). This fertility rate is still enough to produce a fairly rapid population increase, and a rapid fall in the mean age of the population, unless countered by a high mortality in childhood. Fertility may be highest in the northern missions, and in some Central Australian communities; Jones recorded a fertility ratio of 5·76 at Bathurst Island Mission during the period 1957-60.

(vii) The 1957-64 crude birth rate for Territory Aborigines appeared to lie near 34 per 1,000 population per annum, and 39 in 1967. These are not particularly high figures by world standards, although nearly twice as high as the average for Australia. Maori and American Indian rates were 49 and 39 per 1,000 respectively around 1960. Although the evidence available to the writer is limited, there is good reason to believe that the birth rate on missions is generally much higher—around 50 per 1,000, and about average on settlements, and appears to be quite low in the population outside these two administrative settings.

(viii) Largely due to higher birth rates and lower child death rates,

the proportion of the Aboriginal population on missions and settlements (and particularly the missions) appears to be increasing steadily, and their populations becoming younger. Northern Territory Report figures show that in 1955, 44 per cent of the mission population and 35 per cent of the settlement population were 'children' (? under 16). By 1967, these proportions had risen to 51 per cent and 46 per cent respectively. It appears that the population increase of Northern Territory Aborigines will arise primarily from the missions and settlements, and not from the remainder.

(ix) Age-specific and infant mortality rates for Northern Territory Aborigines are very high by world standards. Mortality in the second year of life is particularly high. In some southern communities in some years it has been up to 70 per cent *higher* than the infant mortality rate for the same communities when based on live births. The limited data available shows that the same communities had exceptionally high birth rates. It is believed that the traditional society of the southern area had a low birth rate and rate of natural increase because their nomadic way of life meant that more than one infant in arms was an insuperable handicap. This low 'birth rate' was achieved, probably, by a combination of deliberate or accidental birth control (via sub-incision of the male urethra), abortion, and infanticide, plus the effects on fertility of prolonged breast feeding. In their new circumstances, the ex-desert nomads appear to have neither the means, the experience nor the will to control their birth rate,[4] nor to handle the child-rearing problems which result.

(x) From limited data, it appears that the mission communities in general have the lowest infant and second-year mortality rates; they also have the highest birth rates. An increasing preponderance of children in the missions is inevitable. Unfortunately, because of their locations, the majority of the missions do not appear to be particularly well-placed to provide gainful employment in the 'real' economy. Thus a fall in the employable proportion of the mission populations will increase their dependence on the mission for the necessities of daily living—the genesis of a dangerous vicious circle, receiving added impetus through the good intentions and humanitarian concern of the missionaries struggling to improve living conditions.

(xi) The main factor which is exerting some check on Aboriginal population explosion in the Northern Territory is the very high infant

[4] In 1968, at least one of the government settlements in Central Australia was experiencing a demand for the insertion of intra-uterine loops from the relatively more sophisticated Aboriginal women in that particular setting.

and childhood mortality. The main causes of infant death have been shown to be the common 'diseases of poverty'—gastroenteritis, pneumonia, and malnutrition. Such disease can be reduced in two ways—by encouraging a marked rise in the standards of living, hygiene and nutrition; and by providing close medical and nursing support in 'captive' communities. In the writer's view, the former can solve the problem, but the latter can only postpone it for perhaps a generation.

(xii) The psychological aspects of Aboriginal ecology are not directly evident from the data which has been discussed, but the attitudes described by Elkin as 'intelligent parasitism' and by Christophers as 'institutional neurosis' can be sensed in the biologically successful but materially and psycho-socially dependent institutionalised communities in the Northern Territory. The prolonged and in a sense 'hereditary' impact on community enterprise and initiative must be a most potent factor in either preventing Aborigines from becoming a viable component of Australian society on the one hand, or preventing them from returning to a state of ecological balance with their physical and biological environment (as enjoyed by their ancestors) on the other hand.

(xiii) As for medical or health services available to the Northern Territory Aborigines, there is little to be learned from the data presented so far except that the services have not been particularly successful in reducing infant and childhood mortality and the birth rate. Nor do these services appear to have achieved the degree of close medical contact necessary to achieve an accurate and detailed picture of the causes of death, for example.

DEMOGRAPHIC AND MORTALITY PATTERNS IN WESTERN AUSTRALIA

Depending on the source of data, the Western Australia full-blood Aboriginal population in 1961 was 10,121,[5] about 8,825[6] or 8,121.[7] In 1966, the Department of Territories' estimate (see Table 1) was 9,905. The 1966 Census figures do not distinguish full-blood from half-Aborigines.

The only figures for the age and sex distribution of this full-blood population are those published by Jones (1970b:11, 21) for the 1961

[5] *Commonwealth Year Book*—Census population of 8,121 plus 2,000 estimated as being 'out of contact'.

[6] Interpolated into Western Australian Department of Native Welfare figures published in the Annual Reports of the Commissioner of Native Welfare.

[7] Census of 1961.

Census. The population pyramid for Western Australian full-bloods is noticeably narrower at the base and has a broader upper section when compared with that for Northern Territory Aborigines for 1961. It has an overhanging male and female component in the 60-69 age-groups—most likely the result of a mis-statement of age in connection with entitlement to age pensions or other benefits; the same feature can be seen in the American Indian age-sex pyramid.

The Western Australian full-blood Aboriginal population in 1961 was thus 'older' than its equivalent in the Northern Territory. On Jones's evidence, the proportion under the age of 15 was 33 per cent, almost identical with that for Australia (32 per cent). At first sight, this suggests that the Western Australian fertility and birth rates had been low (or else the infant and childhood Aboriginal mortality had been exceptionally high) and that the Western Australian full-blood population had not entered a phase of rapid population growth.

The Annual Reports of the Commissioner of Native Welfare give the Aboriginal populations for the various years, divided into 'children' and 'adults'. It is presumed that 'children' are defined as those under 16; the proportion under 16 should amount to about 1 per cent more than the proportion under 15 (obtainable from census data).

The Native Welfare Department's figures for the ten years from 1957 to 1966 indicate that the proportion of 'children' had increased from 24·9 per cent in 1957 to 31·4 per cent in 1966. The very low figure in 1957 (equivalent to an under-15 proportion of about 24 per cent) suggests that the population had been through a prolonged period of gradual decline. This is not apparent in the *Year Book* estimates for the Western Australian Aboriginal population, but the inaccuracies in the estimated numbers including Aborigines 'out of contact' are evidenced by the abrupt change from 20,338 in 1947 to 10,195 in 1954.

The Western Australian 'other bloods' (i.e. part-Aboriginal) proportions under 15 in 1957 and 1966 were 53 per cent and 54 per cent respectively—quite a different picture, indicating a very high birth rate and an exploding population.

The only direct evidence for birth, death, and natural increase rates for Western Australian full-blood Aborigines was published with the *Annual Report of the Commissioner of Native Welfare* for 1961 (pp. 60-2) in which births and deaths for the combined full-blood and mixed-race populations of three Native Welfare Districts were given, together with the adult and child population numbers for full-bloods and part-Aborigines separately (for two districts) and together (for the third

district). It is not possible to derive separate birth and death rates from these data, but the figures are of some interest (Table 13).

Table 13: *Proportion of children in three Western Australian districts, 1961*

	Full-blood		Mixed-race		Total Aborigines	
	Total pop.	% children	Total pop.	% children	Total births	Total deaths
Southern District	235	57	2,566	59	97	23
Central District	39	33	2,757	56	33	23
Northern Division—						
W. Kimberley Subdistrict	(Total population 2,011)				95	37

From these figures the following rates may be derived:

	Rates per 1,000 (total Aborigines)		
	Crude birth rate	Crude death rate	Natural increase rate
Southern District	34·6	8·2	28·4
Central District	12·0	8·3	3·7
W. Kimberley Subdistrict	47·2	18·3	28·9

In the Southern and Central Districts (the far south-west of Western Australia and the area around Perth respectively) full-bloods make up a minor proportion of the total, and nothing can be deduced for full-bloods from rates for the combined population. The high proportion of children in the small full-blood Aboriginal population of the Southern District may be attributable to children from other areas in schools and hostels in this District.

In the West Kimberley Subdistrict, part-Aborigines are a minority, so that the figures are more relevant to the full-blood population. Most of the northern missions are situated in this area, and in this and other respects it corresponds to Arnhem Land and the north coast of the Northern Territory. The high crude birth rate for this area probably reflects a moderately high birth rate in the full-blood population, added to by a higher birth rate in the part-Aboriginal minority.

The 1961 *Annual Report* lists the adult and child populations of thirty missions in Western Australia, but does not separate full-blood and mixed-race components except for the case of six missions stated to have wholly full-blood populations (W.A., *Annual Report*, 1961:52-8). Many of the mission establishments are hostels for children rather than the residence

of complete families as in the Northern Territory. The total population of the thirty missions was 3,233 persons, of whom 64 per cent were children. If fourteen missions where the residents were wholly or almost wholly children are excluded, the remaining sixteen missions had a population of 2,212, of which 48·1 per cent were children. Taking only the six missions with entirely full-blood populations, the total was 1,205 of whom 44·7 per cent were children. Of these six missions, all except Kulumburu (on the northern Kimberley coast) were in the drier desert and semi-desert areas of Western Australia. The populations of these missions, and the proportion of children, for 1961 and 1966, are given in Table 14.

Table 14: *Population and proportion under age 16 at six selected Western Australian missions 1961 and 1966**

Mission	1961[1] Population	% under 16	1966[2] Population	% under 16
Balgo Hills	181	50·3	248	57·7
Cundeelee	156	40·4	182	42·9
Jigalong	141	46·8	208	43·3
Kulumburu	220	32·3	203	32·0
Warburton Ranges	427	46·8	377	47·5
Wiluna	80	60·0	68	57·4
Total	1,205	44·7	1,286	46·2
Total W.A. full-blood population of stated age[3]	7,618	% under 15 33·2		

* The six missions selected were those recorded as having only full-blood residents. Others were recorded as having a mixture of full-bloods and mixed-race Aborigines, the actual proportions not being stated.

Sources:
[1,2] Western Australia: *Annual Report of the Commissioner of Native Welfare, 1961* and *1966*.
[3] Jones (1967), Table 3.

The figures show that the proportion of children was much higher on the missions than in the total census-enumerated full-blood Aboriginal population of 1961, except at Kulumburu. It is not possible to be sure that the high proportion of children in the mission population described was due to a higher fertility and birth rate. The proportion is highest in those missions where the community was formerly desert-dwelling (and probably had a very low proportion of children because of the exigencies of the environment).

It is possible to compute a crude birth and death rate for Western Australian full-blood Aborigines only by the most indirect means from the available data. The estimate derived is based on so many assumptions that one is hesitant to use the figures for further extrapolations in the computation of cause-of-death rates, etc., but unfortunately there is no alternative. The following calculations are based on the assumptions that data on full-blood deaths for 1961-4 compiled with the assistance of the Bureau of Census and Statistics are complete, and relate to the same full-blood population as estimated by the Department of Native Welfare, and that an end-of-year population interpolated as being half way between the two adjacent mid-year populations is reasonably close to the true value. The computation of a birth rate then depends on the assumption that births = natural increase plus deaths (derived from: natural increase = births minus deaths).

Table 15: *Estimated births, Western Australian full-blood Aborigines, 1960-5*

Year	Estimated mid-year population[1]	Interpolated end-of-year population	Natural increase 1961-4*	Deaths 1961-4[2]	∴ Births 1961-4†
1960	8,800	8,812	—	—	—
1961	(8,825)‡	8,840	38	201	239
1962	8,854	8,975	121	176	297
1963	9,205	9,405	200	179	379
1964	9,411	9,658	253	169	422
1965	9,905	—	—	—	—
Total 1961-4	36,295	—	612	725	1,337
Average 1961-4	9,074	—	153	181	323
Crude death rate 1961-4			19·9 per 1,000 per annum		
Natural increase rate 1961-4			15·6 per 1,000 per annum		
Crude birth rate 1961-4			35·6 per 1,000 per annum		

* Natural increase = end-year population minus end-year population of previous year.
† Births = natural increase + deaths.
‡ Interpolated value—no population figure available for 1961.

Sources:
[1] Western Australia, *Annual Reports of the Commissioner of Native Welfare*, 1960-1965.
[2] Author's research assisted by Commonwealth Bureau of Census and Statistics.

These rates correspond fairly closely to those which have been determined for the Northern Territory. The relatively high Western Australian Aboriginal crude death rate of 19·9 implies that the data for full-blood deaths were probably fairly complete (or at least did not underestimate the number of deaths). Northern Territory equivalent

rates calculated by the writer from data privately supplied for the period 1961–4 were: crude death rate—18·2; crude birth rate—31·6; natural increase rate—13·4.

If the birth, death, and natural increase rates for Western Australian full-blood Aborigines are nearly the same as those for the Northern Territory, but the population is 'older', there are two likely explanations: the birth rate rose later in Western Australia than in the Northern Territory, or the infant and childhood mortality in Western Australia fell earlier or has been lower in past decades, during a period of low fertility.

Some ecological differences for Western Australian full-blood Aborigines, when compared with the Northern Territory, are that the institutionalised component is smaller—about one-fifth—and there are no government settlements in the sense that they exist in the Northern Territory. The full-blood population is only about 1 per cent of the State total. About 44 per cent inhabit the drier desert and semi-desert areas of the State, as against about 40 per cent for the Northern Territory. The higher proportion of part-Aborigines to total (about 55 per cent in Western Australia as against 20 per cent in the Northern Territory) suggests a greater degree of Aboriginal-white miscegenation in the past, and therefore a greater degree and duration of social contact with the white population in Western Australia as a whole.

The population increase in Western Australian full-blood Aborigines appears to have had a later take-off than in the Northern Territory, but on the evidence of the birth and death rates computed above may keep pace with and possibly exceed that in the Northern Territory, and cannot be attributed to institutionalisation as has been suggested for the Northern Territory. Can it be attributed to 'a greater degree of social contact'?

WESTERN AUSTRALIAN MORTALITY, 1961–4

The following analysis is based almost entirely on data for 723 coded deaths over the years 1961 to 1964 inclusive, obtained by the writer with the assistance of the Bureau of Census and Statistics. As has been suggested above, their probable completeness is attested to by the high crude death rate derived when they are divided by the full-blood population estimates.

Table 16 shows the number of deaths by age-group, together with percentages of total deaths and some estimated age-group mortality rates for the period 1961–4. The proportions may be compared with those shown in Table 11 for Northern Territory Aborigines and others.

The most striking difference is between the proportions of deaths under

age 5 to total deaths. The Western Australian figure (20·3 per cent) is less than half that for Northern Territory Aborigines (about 44 per cent in both 1958-60 and 1964-5) and is lower than that for Maoris (25·7 per cent in 1964) and American Indians (27·3 per cent in 1960). As a result, the proportions dying at age 60 and over (58·2 per cent) is higher than in any of the other populations mentioned. The PMR(50+) for Western Australian full-blood Aborigines is 66·4—a high figure by world standards.

Table 16: *Percentage of deaths and estimated rates by age-group, Western Australian full-blood Aborigines, 1961-4*

Age-group	Number of deaths[1]	Percentage of total deaths	Estimated rate per 1,000 in age-group per annum (rounded)[2]
0-	101	14·0	—
1-	35	4·8	—
2-4	13	1·8	—
0-4	149	20·6	33
5-39	48	6·6	2
40-59	104	14·4	15
60 and over	419	58·0	80
Age unknown	3	0·4	—
All ages	723	100·0	20

Sources:
[1] Author's research assisted by Commonwealth Bureau of Census and Statistics.
[2] Average deaths per annum = number of deaths ÷ 4—see 1. Age-group population = total mid-period population (9,070) estimated from Department of Native Welfare figures published in *Annual Reports of the Commissioner of Native Welfare* for years 1961 to 1964, divided by age-group percentages obtained from Jones (1970), Table 3, for the 1961 Census enumeration of 7,618 Aborigines where age was stated.

The estimated 0-4 age-group mortality rate (33 per 1,000) is also relatively low compared with the Northern Territory figure (47·5 in 1958-60, around 52 in 1964-5). There are no data for the numbers of Western Australian full-blood Aboriginal children by single years of age, so it is not possible to calculate age-specific rates for children aged under 1, 1, and 2-4 years. The percentages of deaths falling in the second year of life (5 per cent of all deaths) is considerably lower than in the Northern Territory, but still higher than in the Maori or American Indian populations.

The only method by which the Western Australian full-blood Aboriginal infant mortality rate may be estimated is to divide the number of

infant deaths (100) by the estimate of births in the same period given in Table 15 (1,337), giving an infant mortality rate for the period of 75 per 1,000 live births. A second-year mortality based on the previous year's live births can be estimated for 1962, 1963, and 1964 only. The data disclose 23 such deaths from a cohort of 915 estimated births—a rate of 25 per 1,000 births (Northern Territory 1965-7 = 40, from Table 6). The deaths under age 2 thus amounted to 100 per 1,000 live births, a considerably lower figure than that for the Northern Territory, which was 170 for a slightly later time period. The Western Australian rate was still about twice that for Maoris, and five times that for Australia.

The median age at death can be calculated from Table 16 to lie somewhere in the over-60 age-group, compared with about 25 years for the Northern Territory, 40 years for Maoris, 45 years for American Indians and 71 years for Australia (calculated from Table 11). As already mentioned, this figure can be taken as suggesting that a full-blood Aboriginal in Western Australia has a better than 50 per cent chance at birth of reaching the age of 60 years, given unchanging mortality patterns.

Here, then, is one explanation for the 'older' Western Australian full-blood Aboriginal population—a lower mortality in infancy and childhood (over a long period, with low fertility). Why this should be so deserves further research—the basic reason is not apparent from the cause-of-death pattern, although this supplies some clues.

CAUSE OF DEATH, 1961-4

Table 17 shows the leading causes of death (all ages) by number of deaths, percentages of total deaths, and estimated rates, for full-blood Western Australian Aborigines during the period 1961-4. The figures may be compared with those in Tables 9 and 10 for Northern Territory Aborigines, Maoris, U.S. Indians, and Australia.

While the 'senility and ill-defined' group is still large (21 per cent), there remains a major re-ordering in the ranking of other common causes when compared with the Northern Territory figures. 'Heart disease' and 'vascular lesions of the central nervous system', both diseases of later life, have moved up in proportion and rate, reflecting the older Western Australian population structure. 'Pneumonia and influenza', diseases which kill in both infancy and old age, were slightly less common in the Western Australian population, but gastroenteritis, a disease which kills infants and young children almost exclusively of any other age-group, had a lower percentage and rate (8 per cent, 150 per 100,000) than in the Northern Territory (11 per cent, 220 per 100,000).

Table 17: *Leading causes of death, all ages, Western Australian Aborigines 1961–4, by percentage of total deaths and estimated rates*

Cause	Cause of Death Code	Number*	% of total deaths	Estimated rates per 100,000†
Senility and ill-defined causes	(794, 795)	155	21·4	430
Pneumonia and influenza	(480–493)	117	16·2	320
Heart disease	(400–443)	110	15·2	300
Vascular lesions of C.N.S.	(330–334)	75	10·4	210
Gastroenteritis and dysentery	(045, 571)	55	7·6	150
Malignant neoplasms	(140–205)	37	5·1	100
Diseases of infancy	(760–776)	28	3·9	80
Accidents	(E800–E965)	25	3·5	70
Anaemia and malnutrition	(280–287, 290–293)	12	1·7	30
Tuberculosis (all forms)	(001–008)	2	0·3	6
All causes	(001–E999)	723	100·0	2,000

* In these data there was difficulty in assigning some deaths to a particular category of cause, giving rise to minor reservations with respect to the proportional status of the categories listed in the table.
† Estimated rates based on estimated crude death rate of 20 per 1,000, derived by dividing the average annual deaths (723/4 = 181) by the average population for 1961–4 (9,070). A higher crude death rate of 22·3 per 1,000 results if the average annual deaths are divided by the 1961 Census population of full-bloods, which was 8,121, but this value has been discarded because it is more likely that the deaths were drawn from the Native Welfare Department's base population, and in any case the census figures were available for one year only—1961.

Source: Author's research, assisted by Commonwealth Bureau of Census and Statistics.

Two groups of chronic diseases—'anaemia and malnutrition' and 'tuberculosis' which might be considered to reflect, among other things, the standards of health services available to communities and the use made of them—were at lower rates in Western Australia. In Western Australia and the Northern Territory respectively the figures for anaemia and malnutrition were 1·7 per cent, rate 30 per 100,000 and 3·6 per cent, rate 70 per 100,000. Tuberculosis caused only 0·3 per cent of deaths in Western Australia (rate 6 per 100,000) as against 3·6 per cent (rate 70 per 100,000) in the Northern Territory.

The mortality picture, then, is one of more deaths in Western Australia from the diseases of older ages than in the Northern Territory, and fewer deaths from infections of all sorts. Nevertheless, deaths from gastroenteritis and dysentery are still very common when the proportions and rates are compared with those for Maoris, American Indians, and Australia as a whole.

Table 18 shows the leading causes of Western Australian full-blood deaths in three age-groups under 5, together with estimated rates for the whole 0–4 group.

Table 18: *Leading causes of death in infancy and childhood, Western Australian full-blood Aborigines, 1961-4*

Age group	Cause	Cause of Death Code	Number of deaths	Percentage of deaths in age group	Estimated mortality rate per 100,000 in age group
Under 1 year	All causes	(001-E999)	101	100·0	—
	Gastroenteritis and dysentery	(045, 571)	31	30·7	—
	Pneumonia and influenza	(480-493)	21	20·8	—
	Immaturity unqualified	(776)	16	15·8	—
	Diseases of infancy	(760-776)	28	27·7	—
1 year and under 2 years	All causes	(001-E999)	35	100·0	—
	Pneumonia and influenza	(480-493)	15	42·9	—
	Gastroenteritis and dysentery	(045, 571)	12	34·3	—
	Anaemia and malnutrition	(280-287, 290-293)	3	8·6	—
2-4	All causes	(001-E999)	13	100·0	—
	Gastroenteritis and dysentery	(045, 571)	4	30·8	—
Total 0-4	All causes	(001-E999)	149	100·0	3,300
	Gastroenteritis and dysentery	(045, 571)	47	31·5	1,040
	Pneumonia and influenza	(480-493)	38	25·5	840
	Diseases of infancy	(770-776)	28	18·8	620
	Immaturity unqualified	(776)	16	10·7	350
	Unknown and ill-defined causes	(795)	12	8·1	265
	Anaemia and malnutrition	(280-287, 290-293)	6	4·0	130

Source: As for Table 17.

'Gastroenteritis and dysentery' is seen to be the commonest cause in the under 1 year and 2-4 age-groups, and the second commonest cause after 'pneumonia and influenza' in the 1 year, under 2 years age-group. In all these groups it caused around a third of all deaths. If the proportion in the under 1 age-group is coupled with the estimated infant mortality rate, 24 out of each 1,000 babies born die from this cause before reaching one year of age. 'Pneumonia and influenza' is the second commonest cause of death of infants (21 per cent) but is responsible for only about 15 deaths per 1,000 live births. The equivalent figures for Western

Australian full-blood infants, Northern Territory full-blood infants (1964–5), Maori infants, and Australian infants are as follows:

Table 19: *Percentage of infant deaths from intestinal and respiratory infection, Western Australian full-blood Aborigines and others*

Population	Dysentery and gastroenteritis		Pneumonia and influenza	
	% of infant deaths	Rate per 1,000 live births	% of infant deaths	Rate per 1,000 live births
Western Australian full-bloods	31	23	21	15
Northern Territory full-bloods	22	26	23	28
Maoris	10	5	8	4
Australia	2	0·5	8	1·6

From Table 19 it appears that Western Australian and Northern Territory full-blood infants (under 1 year) run very nearly the same high risk of death from dysentery and gastroenteritis, while the Western Australian full-blood infants face about half the risk of death from pneumonia and influenza. In both cases, the risk of death from these conditions is very much higher than in the Maori or Australian infant group.

Detailed comparisons cannot be drawn with certainty when dealing with small populations, with populations examined over asynchronous periods, and where the accuracy of data and the methods by which they have been compiled is unknown or open to doubt. However, there remains strong evidence to suggest that between the Northern Territory and Western Australian full-blood populations there are real differences in age-structure, in general and infant mortality patterns and causes of death, but that the overall birth (and fertility) rates may be very similar. The apparent difference in ecology between institutional and non-institutional Northern Territory Aborigines does not appear to be so marked in Western Australia. Health problems, as evidenced by mortality, are broadly similar in the two populations although differing in degree in certain age-groups and from certain causes. Gastroenteritis and acute respiratory infections in infancy, and a rising birth rate, and the balance between these, are seen to be the major determining factors for the future structure and ecology of the full-blood populations of both States. The trend towards a younger, largely institutionalised population appears to

be more advanced in the Northern Territory. In both groups, the highest birth rates seem to occur in mission populations. In the Northern Territory there is some evidence that these populations are not expanding with exceptional rapidity because the infant and child mortality are also very high. The same might be true for Western Australia but there is no evidence.

Indicators such as the Proportional Mortality Ratio, the median age at death, and the estimated infant mortality rate, together with the age structure of the population and the relatively restrained birth rate, suggest that the Western Australian full-blood population is ecologically more successful than its counterpart in the Northern Territory—that is to say, the Western Australian full-blood population has adapted itself to the change from traditional life with greater efficiency and economy of human effort. Why this should be so is not clear, but it is very tempting to suggest that one reason might be the larger proportion of Western Australian full-bloods who live outside any sort of institutional atmosphere. It must be admitted, however, that part of the picture is consistent with a population which has aged because of a steady decline in numbers, in births and fertility, until perhaps the last ten years or so, and that the patterns might have been very different twenty years ago. If the Western Australian full-bloods have entered a phase of population expansion, this must have occurred very recently—probably not before the late 1950s.

DEMOGRAPHIC AND MORTALITY PATTERNS IN QUEENSLAND

No account is taken here of the Torres Strait Islanders. Although it could be argued that they are as 'aboriginal' to Queensland as the mainland Aborigines, they are distinguishable on ethnic, residential, and administrative grounds,[8] and it has seemed preferable for these reasons to exclude them from this study.

Population figures for Queensland Aborigines given by the Department of Native Affairs since 1961 do not distinguish between Aborigines and Torres Strait Islanders. For 1961, there is a choice of population figures: the *Commonwealth Year Book* and Jones (1970: Table 3), both give a figure of 8,686 full-bloods, while the 1961 *Annual Report of the Director of Native Affairs* for Queensland gives a figure of 11,445, of whom 10,325 were classed as 'controlled' and 1,120 as 'non-controlled'. The 1966 estimate (see Table 1) was 12,000.

[8] Torres Strait Islanders possess a degree of self-government in accordance with the Torres Strait Islanders' Act, 1939-46.

According to Jones's estimates, 44 per cent of Queensland Aborigines were full-blood at the 1961 Census, while according to the Department of Native Affairs the figure was 41 per cent. The Department of Territories' estimate for 1966 was 29 per cent, but the denominator here included a large number of part-Aborigines who would not have been identified in censuses. According to the Department of Territories' figures, full-blood Aborigines contributed about 0·7 per cent of the total population of the State of Queensland in 1966.

The age-sex population pyramid based on Jones's analysis of 1961 Census data has almost the same base as that for the Northern Territory, but tapers a little more evenly towards the older age groups. Its shape is consistent with a population having had a relatively high birth rate for a considerable period, and a relatively high infant and childhood mortality. It is very similar to pyramids for Maoris and part-Aboriginal populations, although not quite so broad at the base. The proportion under age 15 was 40·4 per cent in 1961—a higher proportion than that in the Northern Territory or Western Australia.

There are no data available to the writer from which birth, death, or infant mortality rates may be calculated for the total full-blood population of Queensland. The Department of Native Affairs does not publish births and deaths except for the populations of missions and settlements, and does not publish any figures for infant deaths.

Data for full-blood deaths excluded from Queensland's vital statistics in 1962, 1963, and 1964, which were made available to the writer, are already grouped by age (infant deaths are included in the 0–4 age-group), and in any case appear to be underestimated—as is discussed below.

Jose and his colleagues (Jose *et al.*, 1969:74) estimated the crude birth and death rates, and the infant mortality rate, for nine Queensland mission and government settlements during the period 1962–6, as follows:

Crude birth rate	41·7 per 1,000 population per annum
Crude death rate	15·0 per 1,000 population per annum
Infant mortality rate	112 per 1,000 live births.

The population on which these figures are based contains an unrecorded proportion of part-Aborigines. If the figures are based on the populations of six settlements around the Gulf of Carpentaria, which Jose *et al.* (1969:72) states to be 'inhabited mainly by full-blood Aborigines representing the descendants of particular tribal groups', the following figures are derived:

Crude birth rate	38·0 per 1,000 population per annum
Crude death rate	13·1 per 1,000 population per annum
Infant mortality rate	86 per 1,000 live births
Natural increase rate	25 per 1,000 population per annum.

These figures cannot be compared with those for the Northern Territory and Western Australia because they are based on a small proportion of the Queensland full-blood population selected by residence on a mission or settlement.

The *Annual Reports of the Director of Native Affairs*, from 1951 through to 1960, contain figures for the full-blood and mixed-race Aboriginal populations separately for Queensland missions and government settlements, together with the combined births and deaths.

In 1951, 52 per cent of the Queensland full-blood population resided on missions and settlements, but only 46·5 per cent did so in 1960. During the same period the institutionalised Northern Territory full-blood population proportion increased from 51 per cent to 60 per cent. It can be shown that the apparent decrease in Queensland was not due to a reduction in numbers in the institutionalised component, and therefore can only have been due to a particularly rapid increase in the non-institutionalised component. Was this due to a net migration out of the institutionalised communities, or to a population explosion among the non-institutionalised communities? The answer to this question is important to the health ecology of Queensland full-bloods, including the planning of health and medical services for them.

The population, birth, and death figures for eleven missions and three settlements given in successive annual reports from 1951 to 1960 were extracted and tabulated, and five-year-average crude birth and death rates calculated for 1951-5 and 1956-60. The missions were divided into two groups—seven with entirely, or almost entirely, full-blood populations, and four with a mixed-race majority. The three settlements also had a mixed-race majority. The results are presented in Table 20.

This table shows that in the three groups the crude birth rate rose between 1951-5 and 1956-60. For the seven full-blood missions it rose from the relatively low value of 22·2 per 1,000 to 28·9 per 1,000. The crude death rate also rose slightly from 13·7 to 14·4 per 1,000. The average natural increase rate therefore rose from 8·5 to 14·4 between the two periods. In the other groups, the proportion of part-Aborigines is reflected by higher birth rates.

If the natural increase rates of 8·5 per 1,000 (for four years) and 14·4

Table 20: *Percentage full-bloods, crude birth and death rates for three groups of Queensland institutions for Aborigines, 1951-5 and 1956-60*

	1951-5			1956-60		
	% Full-blood	Crude birth rate	Crude death rate	% Full-blood	Crude birth rate	Crude death rate
7 Missions*	95·5	22·2	13·7	95·1	28·9	14·4
4 Missions†	40·5	42·2	13·4	38·2	45·2	10·3
3 Settlements‡	37·7	45·9	18·4	38·8	49·6	18·4

* Lockhart R., Edward R., Mitchell R., Arakun, Weipa, Mornington Island, Doomadgee.
† Yarrabah, Mapoon, Monamona, Hope Vale.
‡ Palm Island, Cherbourg, Woorabinda.
Source: Queensland, *Annual Report of the Director of Native Affairs*, 1951-60.

per 1,000 for five years are applied to the 1951 full-blood mission population (2,441) the expected increase is to 2,712 in 1960. The observed increase was to 2,718—very close to the expected. Although the mathematical argument is crude, it suggests that the missions with an almost entirely full-blood population remained self-contained during the period, and did not contribute to the non-institutionalised full-blood population by migration.

In the composite communities, however, the picture seems to be different as far as the full-bloods were concerned. The annual reports, for the years 1953 to 1959 only, gave the births and deaths at Cherbourg Aboriginal Settlement for full-blood and mixed-race Aborigines separately, and it is therefore possible to calculate separate crude birth and death rates for this seven-year period. The results are as in Table 21.

Table 21: *Births, deaths, and natural increase, Cherbourg, 1953-9*

	Full-blood	Mixed-race
Average population	150·6	915·4
Percentage of total	14 per cent	86 per cent
Average births (per annum)	18·3	47·7
Average deaths (per annum)	12·4	15·3

From these figures the following rates may be derived:

Crude birth rate	121·5	52·1
Crude death rate	82·5	16·7
Natural increase rate	39·0	35·4

While the mixed-race rates are quite average for similar communities elsewhere, the birth and death rates for full-bloods are extraordinarily high. During this six-year period the full-blood population actually decreased from 164 to 158, whereas it should have increased to about 200. The birth rate can only be accounted for by a very high proportion of very fertile women in the community at any one time—as if women were migrating to Cherbourg to have babies, and subsequently leaving. Similarly, the death rate can only be accounted for by either an extraordinarily high infant mortality rate or a very high proportion of aged adults.

The figures presented, taken overall, suggest a population explosion among full-blood Aborigines which was arising primarily from those not resident at missions and settlements, although contributed to by the relatively few full-bloods in composite settlements such as Cherbourg. This was the picture which it is suggested applied up to 1961. What has happened in the ten years since then cannot be ascertained from available data.

CAUSE OF DEATH, 1962-4

The available data are of limited quantity and quality. The principal sources are, firstly, a list of 240 full-blood deaths subtracted from Queensland vital statistics during 1962-4, and secondly, a list of 133 infant and 32 childhood deaths prepared by Jose (Jose *et al.*, 1969:74) for the combined population of eight Aboriginal settlements (an unstated number of which had composite full-blood and mixed-race populations) for 1962-6.

The number of deaths from the first source amount to only about half to two-thirds of those which would be expected from estimated crude death rates,[9] implying marked under-identification of full-blood deaths during the period.

Table 22 gives the number and percentages of total deaths by age-groups, and compares the percentages with those for the Northern Territory and Western Australia. The grouped data have precluded a separation of the 0-4 age-group into infants, one-year-olds and others. The proportion in the 0-4 age-group appears exceptionally low at 16 per cent, being lower, for instance, than the equivalents for the Maoris (26 per cent) and the American Indians (27 per cent).[10] This implies that the Queensland data is biased by the omission of many deaths in infancy

[9] The deaths for the period would yield a crude death rate of just over 9 per 1,000 population per annum.

[10] Maori and American Indian figures derived from Table 11.

and early childhood. The apparent similarity in age-distribution of deaths between Queensland and Western Australia—including similarities in the PMR(50+) and the median age at death—cannot be accepted therefore.

Table 22: *Deaths by broad age-groups, full-blood Aborigines, Queensland 1962-4 compared with Northern Territory and Western Australia*

Age-group	Queensland 1962-4[1] Number of deaths	Queensland 1962-4[1] % of total deaths	Northern Territory 1964-5[2] % of total deaths	Western Australia 1961-4[3] % of total deaths
0-4	38	15·8	44·4	20·6
5-39	21	8·8	13·3	6·6
40-59	40	16·7	13·0	14·4
60 and over	141	58·8	28·7	58·0
All ages	240	100·0	99·4*	99·6†
Proportional mortality ratio age 50 and over		67·1	34·7	66·4
Median age at death		60+	25	60+

* Age at death unknown = 0·6 per cent.
† Age at death unknown = 0·4 per cent.
Sources:
[1] Author's research, assisted by Commonwealth Bureau of Census and Statistics.
[2] See Table 11.
[3] See Table 16.

Table 23 lists the leading causes of death, all ages, by number and percentages of total, from the same data source. The same doubts as to the validity of the age-distribution apply to the distribution by cause and the ranking of leading causes. The high proportion of deaths attributed to 'senility and ill-defined' causes is also noteworthy.

In general, then, no firm conclusions can be drawn from the available data. There remains a general impression that Queensland full-blood Aborigines experience a pattern of mortality intermediate between those for the Northern Territory and Western Australia, and that this would be revealed if the full-blood deaths which seem to be missing from the data were included.

Queensland, at the 1961 Census, had the youngest full-blood population of the three States or Territories considered so far—44 per cent under age 15. The majority live on or near the coast, particularly the north coast, Cape York Peninsula, and the Gulf of Carpentaria, and relatively few inhabit the drier south-west quarter of the State. In the settlements and many of the missions, and probably in the pastoral areas, full-blood

Table 23: *Leading causes of death, all ages, full-blood Aborigines, Queensland 1962-4 compared with Northern Territory and Western Australia*

Cause	Cause of Death Code	Queensland 1962-4 Number of deaths	Queensland 1962-4 % of total deaths	Northern Territory 1964-5 % of total deaths	Western Australia 1961-4 % of total deaths
All causes	(001-E999)	242	100·0	100·0	100·0
Senility and ill-defined	(794, 795)	73	30·2	17·3	21·4
Heart disease	(400-443)	44	18·2	6·0	15·2
Pneumonia and influenza	(480-493)	21	8·7	17·9	16·2
Vascular lesions of C.N.S.	(330-334)	15	6·2	1·6	10·4
Malignant neoplasms	(140-205)	13	5·4	4·0	5·1
Gastroenteritis and dysentery	(045, 571)	11	4·5	10·7	7·6
Accidents	(E800-E965)	8	3·3	5·1	3·5
Diseases of infancy	(760-776)	5	2·1	10·9	3·9
Anaemia and malnutrition	(280-287) (290-293)	3	1·2	3·6	1·7
Tuberculosis (all forms)	(001-008)	1	0·4	3·6	0·3

Sources: As for Table 22.

and mixed-race Aborigines appear to be more intermixed residentially than in Western Australia and the Northern Territory, and it may be that the process of detribalisation—or of acculturation towards mixed-race social attitudes and behaviour—is more advanced in the Queensland non-institutionalised full-bloods than among their counterparts in the Northern Territory or in Western Australia. This is a possible explanation for a high birth-rate in non-institutionalised full-bloods. The quality of the argument suffers along with the quality of the data.

Jose's figures for eight settlements, on the other hand, are probably complete but of limited relevance to the total Aboriginal population of Queensland. In addition, more than half the deaths he examined appear to have come from the part-Aboriginal population. The leading causes of death in infants on the eight settlements, the percentage of total infant deaths, and death rates per 1,000 live births calculated from Jose's data, are shown in Table 24.

The pattern is similar to that for the Northern Territory and Western Australia, with gastroenteritis, pneumonia, and prematurity contributing to the same degree, relatively and absolutely, in the three States.

Table 24: *Leading causes of infant death, eight Queensland full-blood settlements, 1969*

Cause	Percentage infant deaths	Rate per 1,000 live births
Gastroenteritis	25·6	28·6
Pneumonia	20·3	22·7
Prematurity	12·0	13·4
Unknown	11·3	12·6
Birth trauma	6·8	7·6
Congenital malformation	6·8	7·6

Jose *et al.* (1970:74) state that 'diagnoses were often recorded by non-medical staff and only rarely confirmed at autopsy'—a comment that would apply equally well in the Northern Territory or Western Australia in the relevant periods.

DEMOGRAPHIC AND MORTALITY PATTERNS IN SOUTH AUSTRALIA AND NEW SOUTH WALES

SOUTH AUSTRALIA

The number of full-blood Aborigines in South Australia has not been determined with any certainty except at the 1961 Census when the number enumerated was 2,147. The South Australian Aborigines Protection Board did not attempt to give estimates year by year, and did not publish figures for total full-blood Aborigines in the State. From 1953 to 1960 the annual reports of the Aborigines Protection Board merely stated that there were 'slightly more than 5,000 in approximately equal proportions of full-bloods and mixed-bloods. On account of their nomadic habits it has proved difficult to obtain accurate statistics relating to Aborigines'. (South Australia, *Report*, 1953:3). In 1960 the estimate was increased to 'approximately 6,000', and in 1963 'approximately 2,000 Aborigines and 4,000 persons of Aboriginal blood'. The estimated totals rose to 6,100 in 1965 and 6,200 in 1966. The 1966 estimate by the Department of Territories (Table 1) was 3,128 Aborigines and 4,632 part-Aborigines, total 7,760. The 1966 Census total was 5,505 persons with 50 per cent or more Aboriginal ancestry. With this wide variety of estimates, and the relatively small population and small number of births and deaths, calculation of birth and death rates would be a waste of effort (and in any case, no birth or death data are published in the reports).

The age-sex distribution of South Australian full-bloods, from Jones's tabulation of full-blood census data, most resembles that for the Northern

Territory. Of South Australian full-bloods, 39·9 per cent were aged under 15 in 1961—very nearly the same proportion as in the Northern Territory.

The only data for age and cause of death available to the writer are for 83 deaths excluded from South Australian vital statistics over a four-year period from mid-1961 to mid-1965. These data are tabulated in Tables 25 and 26. The figures in Table 25 indicate a high mortality in childhood—particularly in the first year: the percentage of total deaths occurring in the first year (27 per cent) is higher than in any other group examined so far except that in the Northern Territory. The PMR(50+) is 50 and the median age at death a little over 50—both values noticeably higher than those for the Northern Territory but lower than those for Western Australia and Queensland.

Table 25: *Deaths by age-group, South Australian full-bloods, 1 July 1961-30 June 1964*

Age-group	Number of deaths	Percentage of total deaths
0-	18	22
1-4	12	14
0-4	30	36
5-39	7	8
40-59	16	19
60 and over	29	35
All ages	83*	98
Proportional mortality ratio age 50 and over	= 50	
Median age of death	= 50+	

* Includes one death where age not stated.

Source: Author's research, assisted by Commonwealth Bureau of Census and Statistics.

Table 26 lists the leading causes of death, all ages. There is a relatively small proportion of ill-defined causes (10 per cent). Heart disease is at the top of the list (19 per cent), but gastroenteritis accounts for nearly as many deaths (17 per cent) and contributes a higher proportion than in any other State.

A high proportion of deaths due to accident (10 per cent) results almost entirely from the recording of six out of eight such deaths as the effects of 'heat and insolation'—evidently a risk of the desert environment of South Australian full-bloods.

The numbers are so small that it is pointless to examine the cause of death in this population in further detail. The limited amount of data

Table 26: *Leading causes of death, all ages, South Australian full-blood Aborigines, July 1961 to June 1964*

Cause	Cause of Death Code	Number of deaths	% of total deaths
All causes	(001-E999)	83	100
Heart disease	(400-443)	16	19
Gastroenteritis and dysentery	(045, 571)	14	17
Pneumonia and influenza	(480-493)	11	13
Accidents	(E800-E965)	8	10
Senility and ill-defined	(780-795)	8	10
Diseases of infancy	(760-776)	6	7
Malignant neoplasms	(140-205)	5	6
Anaemia and malnutrition	(280-287) (290-293)	2	2
Vascular lesions of C.N.S.	(330-334)	1	1
Tuberculosis (all forms)	(001-008)	nil	—

Sources: As for Table 16.

suggests that the demographic and ecological situation of South Australian full-bloods is rather more similar to that of the Northern Territory (not unexpectedly), than that of Western Australia or Queensland.

NEW SOUTH WALES

The full-blood population of New South Wales is small. The Census of 1961 identified only 1,488 persons as 'full-blood'. Among deaths registered in New South Wales from January 1960 to June 1965,

Table 27: *Deaths by age-group, New South Wales full-blood Aborigines, 1 January 1960-30 June 1965*

Age-group	Number of deaths	% of total deaths
0-	20	26
1-4	6	8
0-4	26	34
5-39	10	13
40-59	10	13
60 and over	30	40
All ages	76	100
Proportional mortality ratio age 50 and over	= 49	
Median age at death	= 48	

Source: Authors research, assisted by Commonwealth Bureau of Census and Statistics.

a total of 76 were identified as full-blood Aborigines. The distribution by age-group and cause of death, as far as the limited data are able to show, was similar to that for South Australian full-bloods:

Table 28: *Leading causes of death, all ages, New South Wales full-blood Aborigines, 1 January 1960–30 June 1965*

Cause	Cause of Death Code	Number of deaths	% of total deaths
All causes	(001-E999)	76	100
Heart disease	(400–443)	21	28
Pneumonia and influenza	(480–493)	10	13
Gastroenteritis and dysentery	(045–048, 571)	9	12
Malignant neoplasms	(140–205)	6	8
Accidents	(E800–E962)	5	7

Among the remainder, malnutrition and alcoholism received three mentions each (but not as primary causes), and there were two suicides—one of the latter the result of self-immolation with kerosene by a middle-aged woman in 1965, of interest because it is a most unusual mode of suicide in Australia.[11] There was one death from pulmonary tuberculosis recorded.

MORTALITY AND RELATED STATISTICS: SOME CONCLUSIONS

The data from all States and the Northern Territory for full-blood Aboriginal mortality and vital statistics, but in particular those from Queensland, South Australia, and New South Wales, must be viewed with some scepticism as to their completeness and accuracy. The Bureau of Census and Statistics has been understandably concerned at the possible misuse of figures derived from questionable data, particularly since Aboriginal health becomes a politically 'hot' issue from time to time and the figures are quoted in Parliament. The writer agrees wholeheartedly with the Bureau's concern, but on the other hand feels that a little light shed on Aboriginal health and vital statistics is healthier than complete darkness. The ultimate issue is surely not whether the figures are an absolutely accurate representation of events and differences, but whether useful information and insight can be gained in spite of acknowledged defects in the data. Until such time as accurate data are available, there is no option but to make the best of the existing situation.

[11] Suicide in this manner came to public attention in 1963 with the publication of a photograph in the press of a Buddhist protester immolating himself in Saigon.

The type of mortality data upon which most of the preceding analysis has been based ceased to exist after 1967, unless it is still being collected by the various departments responsible for Aboriginal welfare on their own initiative. One hopes not only that this information is still being collected, but also that it will be published periodically. Unless this is done, current and future trends in Aboriginal health and ecology revealed in demographic and mortality statistics will be even more obscure than they have been up to 1967.

Because the data from the States vary in completeness, in relevance to the total State full-blood populations, and in the period covered, it would be unrealistic to attempt to combine them to produce statistics for Australian full-blood Aborigines as a whole (except for data acquired *in toto* from national censuses). As far as the mortality and vital statistics are concerned, there is more to be learned from looking at the differences and similarities between States than at a composite value for all States.

There are many similarities. One that stands out is high death rate in infancy and childhood from the same leading causes—gastroenteritis, acute respiratory infections, and prematurity—typically causes of death of poor communities in poor environments. The leading causes of death in older age-groups also have the same pattern in the different States, but in this case the pattern is not very different from that for the older age-groups in all societies, including non-Aboriginal Australia.

Differences in the risks of death at various age-groups, including the infant mortality rate, have been noted. The risks of death in infancy and childhood appear to be highest in the Northern Territory, followed by Queensland and Western Australia in that order. Although it has not been possible to calculate or even estimate rates in this age-group for South Australia and New South Wales, the high proportions of deaths in infancy and childhood suggest a high infant and childhood mortality rate in the small full-blood populations of these States also.

In nearly all mortality statistics, the full-blood Aborigines compare unfavourably with the Maoris of New Zealand and the Indians of the United States—to name two indigenous minority populations in a roughly equivalent social and administrative situation. Furthermore, differences in death rates between Aboriginal populations are not as marked as differences between Aborigines and Maoris or Indians. Why is this so? Is it the result of factors peculiar to Aborigines, such as heredity, environment, or culture, or is it due to defects in the delivery of health and medical care to Aborigines? This important question cannot be answered at this point, and in any case it must be pointed out that both

the Maori and the American Indian figures generally refer to composite full-blood and mixed-race totals for the countries concerned: that is, they include a higher proportion of part-Maoris and part-Indians than the full-blood Aboriginal populations include part-Aborigines. The limited evidence for part-Aboriginal populations is that they usually have lower infant and child death rates than the full-blood Aborigines.

There appears to be an association between infant and childhood mortality rates and the extent of white settlement, the proportion of full-blood to total Aborigines, the proportion of full-blood Aborigines to the total population of States, and the proportion of full-bloods resident on settlements and missions. It is not implied that such associations are cause-and-effect relationships, of course, but one common underlying factor may be a 'cause'—the extent and duration of full-blood Aborigines' exposure to (and involvement in) white Australian rural society, and the degree of acculturation resulting from miscegenation apparent in the so-called full-blood communities of the more settled areas. The proposition is purely hypothetical, of course; its proof or refutation would require in-depth social and medical investigation on a very broad front. It is the identical hypothesis, however, to that which would explain the somewhat lower death rates in the part-Aboriginal populations, even where they are exposed to environmental and economic conditions as bad as (if not worse than) those for full-blood Aborigines in the north and centre of Australia.

The proportional mortality ratio aged 50 and over ranks the populations studied as follows in order of 'health, including demographic conditions':

Western Australia, Queensland	1
South Australia, New South Wales	2
Northern Territory	3

There are some doubts as to whether Queensland deserves equal ranking with Western Australia (because of an apparent under-identification of deaths in infancy and childhood).

The PMR(50+) reflects the interaction of age-distribution of the living and the age-distribution of deaths, and Western Australia's ranking therefore reflects a relatively 'old' population, a relatively low rate of natural increase in the past few decades, as well as a relatively high expectation of life in the early 1960s. If the birth rate remains as high as it appears to be at present, or rises further, a fall in the PMR(50+)

would be expected. Similarly, a fall in the Northern Territory birth rate would be expected to raise the PMR(50+). As will be shown in the next chapter, the PMR(50+) estimated for New South Wales part-Aborigines ranks this population near the Northern Territory full-blood Aborigines —primarily because of the former's very 'young' population.

New South Wales's full-blood population has all but disappeared, and would appear to have gone well beyond the point of no return as an ethnic entity. This raises the question of the extent to which various demographic and mortality indices have been influenced by the depletion of full-blood populations (that is, populations of persons with more than half Aboriginal ancestry) through intermarriage with part-Aborigines of around half or less Aboriginal ancestry. The offspring of such unions, if regarded as part-Aboriginal (and if reasonably numerous) would lead to an apparently 'older' full-blood and apparently 'younger' part-Aboriginal age-distribution. Such an effect, if it occurs, would be expected in States like Western Australia and Queensland, and perhaps South Australia, but not in the Northern Territory (with part-Aborigines a small minority both within and outside the full-blood category) or in New South Wales (with almost no full-bloods at all). Such an effect is purely speculative, but its possibility adds another doubt to the validity of indices for some States, and is another argument for the abandonment of any sort of classification of Aborigines based on the degree of Aboriginal ancestry. As has been pointed out already, such a classification has been necessary in this study because the data have been determined by it: it may also be of use in identifying differences between the health ecologies of 'true' full-bloods and part-Aborigines. From a practical viewpoint, what the distinction really tries to establish is the degree of adherence to traditional Aboriginal culture rather than the degree of Aboriginal ancestry, but it is a poor tool for this purpose outside Arnhem Land in the Northern Territory or limited areas in Queensland, Western Australia, and South Australia.

The remaining Australian States—Victoria and Tasmania—have not entered into the discussion because they have no full-blood Aborigines.

6 DEMOGRAPHIC AND MORTALITY PATTERNS IN PART-ABORIGINAL POPULATIONS

The part-Aboriginal population of Australia outnumbers the full-blood population by about two to one at present, and with its higher birth and natural increase rates it can be predicted that its majority will increase in the immediate future. This increase will be further accelerated if miscegenation between full-blood Aborigines and others continues or increases. Unless the health status of part-Aborigines is very much higher than that of the full-blood population, part-Aborigines will be the major contributors to the Aboriginal 'health problem' for the foreseeable future, although for various reasons—particularly their statistical invisibility—this contribution will not be so noticeable as that of the full-blood population.

Part-Aborigines are concentrated in Griffith Taylor's 'Economic Australia' for obvious historical reasons; the greatest numbers are in Queensland (about 30,000), New South Wales (23,000) and Western Australia (12,000), although their proportion to State or Territory total population is highest in the Northern Territory (7 per cent) (see Tables 1 and 2).

New South Wales is potentially the most fruitful source of data on part-Aborigines—including health and related data—for several reasons: it has a relatively large proportion of the total for Australia (about 30 per cent); the distinction between full-blood and part-Aboriginal poses no problem, since the former do not contribute significant numbers and do not contribute any who could be considered to be living in a traditional (tribal) setting; and environmental circumstances are sufficiently varied between the dry outback, the warm North Coast and the colder

South Coast and Tablelands to represent most of the environmental settings of part-Aborigines elsewhere in 'Economic Australia'.

Closer, more complete settlement of New South Wales predates that in other States (possibly excepting Victoria and Tasmania) and the ecological circumstances of New South Wales part-Aborigines were stabilised earlier. Lessons to be learnt from past and present circumstances in New South Wales might thus be expected to provide forewarning of problems which may arise in the north of Australia as settlement and development proceed.

The Northern Territory has been used in this study as the model for full-blood mortality statistics, mainly because Northern Territory full-bloods were more easily identifiable and better documented. Although New South Wales has been chosen to provide the part-Aboriginal model, the potentiality for better data is no more realised in published figures than in any other State. However, being the State of residence of the writer, the opportunity to carry out personal research in the State's death records has influenced the choice, together with the writer's personal involvement in social and morbidity surveys in New South Wales and greater familiarity with the part-Aboriginal environment.

The demographic, social, economic, and environmental circumstances of New South Wales part-Aborigines have been described elsewhere in the series Aborigines in Australian Society (Long, 1970; Rowley, 1971; Jones, 1970; Beasley, 1970), and some general remarks which are also applicable to the health ecology of New South Wales part-Aborigines have been made in earlier sections of this study. Before considering the New South Wales part-Aboriginal mortality data, however, some salient points in Aboriginal ecology relevant to health and mortality are considered briefly.

THE ECOLOGICAL PATTERN IN NEW SOUTH WALES

The mortality data to be presented below cover the period from 1950 to 1964. Viewed from 1971, this period covers the end of an era in New South Wales part-Aboriginal history characterised by public ignorance of any Aboriginal 'problems', paternalistic segregation of institutionalised communities, and official neglect of—as well as local antipathy towards—Aborigines living on the fringe. In the last few years there has been a quite dramatic change in public and official attitudes towards Aborigines in New South Wales, and a noticeable rise in part-Aboriginal morale. Assistance in housing, education (particularly pre-school, secondary,

and tertiary) and in other fields of social welfare, the extension of medical and hospital insurance to poor families, together with greater social acceptance and the gradual reduction of local 'colour-bars',[1] have made it difficult to equate New South Wales part-Aboriginal ecology in the 1950s with the situation today—without suggesting that the epidemiological factors underlying New South Wales Aboriginal disease and mortality patterns have changed significantly.

Census figures for the period 1950-64 are available for 1961 only. In spite of doubts as to the completeness of part-Aboriginal census enumeration, the distribution of the census-enumerated population in the various statistical divisions of New South Wales gives a picture of where the Aboriginal population was concentrated.

About 90 per cent of New South Wales Aborigines enumerated in the 1961 Census were outside the Sydney metropolitan area, as against about 44 per cent of the white population. For the white non-metropolitan population, about two-thirds were classified as 'urban'—living in country towns and provincial cities—as against one-third of the Aborigines. In relation to health, these figures place about two out of three Aborigines (classified as 'rural') without ready access to doctors and hospitals, and sanitary and other health-related services provided in cities and country towns, as against a figure of about one in seven whites. As the latter group, generally speaking, would have had the means and the personal transport to reduce this handicap considerably, if not entirely, a majority of the New South Wales Aboriginal population can be seen as having been separated from convenient health, medical and related services and facilities available to the non-Aboriginal population.

However, this separation was not quite as stated. Aboriginal stations (supervised settlements) had dispensaries, a member of the staff (usually the manager's wife) to treat minor ailments and give out medicines, and a vehicle to transport the more seriously ill to the doctor or hospital, and some sort of water supply, sanitation, and garbage disposal service was maintained. The reserves and fringe settlements lacked these services and facilities in most cases, but many were often close enough to towns—just outside the town boundaries—to place the inhabitants within walking distance of health, hospital, and medical facilities in the towns.

[1] These changes are well illustrated by an article 'Walgett—then and now' in *New Dawn*, a magazine produced by the Department of Child Welfare and Social Welfare, Vol. 2, No. 5 (August 1971). Although this article seems to gloss over many remaining problems, its appearance in a government publication is an index of recent changes in official attitudes, while its content speaks of marked changes in white and Aboriginal attitudes.

Table 29 shows the distribution of the 1961 census-enumerated Aboriginal population of New South Wales by Statistical Divisions of the State. About 70 per cent of the non-metropolitan Aborigines lived in the north and west of the State, as against only 25 per cent of non-metropolitan whites (Demography, 1963). The northern and western area amounts to about 53 per cent of the total area of the State. Furthermore, the North Central Plain, Central Plain, and Western Divisions—about 43 per cent of the area of New South Wales, or 130,000 square miles—contained 40 per cent of the non-metropolitan Aborigines, but only 7·4 per cent of the non-metropolitan whites. The density of the total population of this area was less than one person per square mile.

Just as the full-blood Aborigines were concentrated in 'Empty Australia', so the part-Aborigines were concentrated in the relatively empty areas of 'Economic Australia' (at least in New South Wales). This has

Table 29: *Aboriginal population (full-blood and mixed-race together) of the Statistical Divisions of New South Wales, 1961 Census figures, together with percentage of all-races population*

Statistical Division	Aborigines *			Percentage of total
	Males	Females	Total	(all races)
Western	1,238	1,151	2,389	3·8
North Coast	1,254	1,098	2,352	1·4
North Central Plain	746	749	1,495	4·3
Central Plain	744	700	1,444	5·0
Northern Tableland	492	471	963	1·7
Riverina	481	455	936	1·0
South Coast†	454	440	894	0·4
Central Western Slopes	376	358	734	1·1
Hunter and Manning	355	341	696	0·2
North Western Slopes	315	270	585	0·8
Central Tableland	176	212	388	0·2
South Western Slopes	85	152‡	237	0·2
Southern Tableland	56	39	95	0·1
County of Cumberland	709	781	1,490	0·65
Total metropolitan and urban	2,871	2,954	5,825	0·17
Total rural	4,610	4,263	8,873	1·6
Total New South Wales	7,481	7,217	14,698	0·38

* From unpublished tables, Bureau of Census and Statistics, Canberra.
† Excludes 143 at Jervis Bay-Wreck Bay, included with A.C.T.
‡ The high proportion of females in the S.W. Slopes Division is due to the presence of an Aboriginal Girls Home at Cootamundra, and employment of girls from this home in the surrounding district.

important implications in relation to the provision of public health services, and to a lesser extent medical and hospital services—not only to the Aborigines but to the total population of such areas.

The Aboriginal population of metropolitan Sydney amounted to nearly 1,500 in the 1961 Census, and nearly 2,600 in the 1966 Census, but independent estimates—largely guesswork—by various individuals and bodies put the socially-identified Aboriginal population of Sydney at between 10,000 and 15,000, and steadily increasing due to both natural increase and urban drift. For New South Wales as a whole, the Department of Territories' 1966 estimate of 23,000 (quoted in Table 1) is over 50 per cent greater than the census population of 14,219 in 1966, because the latter attempted to exclude those with less than half Aboriginal ancestry. It is likely that the difference between the census and socially-defined numbers would be greater in metropolitan Sydney than elsewhere —because of greater opportunity for self-identification in censuses as 'white' in the metropolitan environment.

In passing, it is an interesting thought that if the 23,000 estimated New South Wales Aborigines all lived in metropolitan Sydney, they would still contribute less than 1 per cent of the city's population, and would be outnumbered by several foreign-born non-British migrant groups—notably those from Greece and Italy; they would still be a minority among minorities.

All estimates of the age-distribution of New South Wales Aborigines indicate a high proportion under the age of 15—around 50 per cent[2]—a characteristic of a population with a high birth rate, and the associated large numbers in the average Aboriginal family and household. The relevance of these observations to health has been mentioned earlier, the chief consequences being low *per capita* income and all the health handicaps that this implies, and difficulties with housing and the domestic environment arising from household numbers as well as household income. If the non-productive age-groups are arbitrarily defined as those under 15 and those over 60 (with reference to the support of dependants rather than to contribution to the total economy), the figures in Table 30 illustrate the relative position of New South Wales Aborigines around 1961, based on census data.

From Table 30, if dependants are considered to be those under age 15,

[2] The lowest figure was for the census-enumerated population with '50 per cent or more' Aboriginal ancestry in 1966—48·3 per cent. The figure for 'mixed-blood Aborigines' in the 1961 Census was 51 per cent, and several surveys of rural New South Wales Aborigines have produced figures of around 53 per cent in recent decades.

Table 30: *Percentages in non-productive age-groups relative to the support of dependants, New South Wales Aborigines and others, c. 1961, both sexes**

Population	Year	Percentage 0–14	Percentage 60 and over	Percentage both groups
N.S.W. Aborigines[1]	1961	50·9	6·6	57·5
Maori[2]	1962	49·2	3·4	52·6
S. African ('coloured')[3]	1958	44·1	4·5	48·6
U.S. Indian[4]	1960	42·2	6·8	49·0
S. African ('asiatic')[3]	1958	42·9	2·8	45·7
N.S.W. rural whites[5]	1961	34·1	11·2	45·3

* Both sexes have been grouped together, since unemployed females contribute in labour and other ways to the support of dependants in their households, and contribute financially through receipt of child endowment and other social service benefits.

Sources:

[1] Jones, 1970, p. 10.

[2] New Zealand Population Census 1961, Vol. 8, *Maori Population and Dwellings*, Wellington, Government Printer.

[3] United Nations, *Demographic Yearbook 1961*.

[4] United States Census of Population 1960, *Non-white population by race*, Washington, Government Printer.

[5] Census of the Commonwealth of Australia 30th June, 1961, Vol. 1, New South Wales, Part 1, *Analysis of Population in Local Government Areas and in Non-Municipal Towns of 1,000 Persons or more*, Canberra, Government Printer.

and producers those not accounted for by those under 15 and those aged 60 and over, it can be seen that only in the New South Wales Aboriginal and the Maori populations (from among those for whom figures are given) do dependants outnumber producers. In the New South Wales rural white population, many children are 'dependent' well after the age of 15, but the addition of these will not alter the ranking shown in the table.

BIRTH, DEATH, AND NATURAL INCREASE RATES IN NEW SOUTH WALES

The Annual Reports of the Aborigines' Welfare Board, recently disbanded, gave the annual populations, births, and deaths for the Aboriginal stations under its jurisdiction, and was, indeed, the only State organisation of this kind to do so in a complete and consistent manner for a part-Aboriginal population.

The births, deaths, and population figures for fourteen New South Wales Aboriginal stations were extracted and tabulated for the period 1954–5 to 1963–4. During this ten-year period, the average population of these stations was 2,655, there were 1,107 births and 309 deaths. These figures yield a crude birth rate of 41·7 per 1,000, a crude death rate of 11·6 per 1,000, and a natural increase rate of 30·1 per 1,000.

During the period the population of these fourteen stations remained fairly steady—the numbers ranging from 2,563 in 1963-4 to 2,771 two years earlier—although births exceeded deaths by about 800 over the ten years. The numbers must have remained steady through a net emigration from the stations equal to the natural increase—that is, the stations appear to have contributed about 3 per cent of their population each year to the non-institutionalised segment of the New South Wales part-Aboriginal population and must therefore have been a slowly diminishing minority among the total numbers in the State. The birth, death, and natural increase rates in the non-station population are unknown, but it can be assumed that their net gain from the stations was quite small—something like one-half of one per cent per annum—compared with their gain from their own natural increase.

The crude birth rates for the individual stations during the period ranged from 57·5 per 1,000 at Boggabilla to 30·6 per 1,000 at Burnt Bridge (near Kempsey). However, because the station populations were not self-contained population isolates, the differences between stations will reflect local social and economic conditions as these affect the numbers and age-groups entering and leaving station communities, rather than fertility alone.

AGE AND CAUSE OF DEATH IN NEW SOUTH WALES

No details of age or cause of death were given in the Aborigines' Welfare Board reports except for a few isolated deaths mentioned in the text of the reports from individual Aboriginal stations.

A relatively small number of full-blood Aboriginal deaths have been examined in the section dealing with full-blood mortality, these data coming from lists prepared for the purpose of excluding full-blood deaths from New South Wales vital statistics prior to the 1967 referendum.

It has been stated earlier that New South Wales full-blood Aborigines were neither full-blood in the same genetic sense that could be applied to most full-bloods in North, Central, and South Australia, nor tribally-oriented. There appears to be little or no reason to attempt to distinguish between New South Wales Aborigines of more than 50 per cent Aboriginal ancestry and the remainder, since the two groups are not segregated from one another physically or socially in the western parts of the State—where most of those recorded as full-blood in censuses have been living. With very few exceptions, the New South Wales Aboriginal population may be regarded as of mixed descent, with a majority having less than

half Aboriginal ancestry. This is not to say that New South Wales Aborigines do not show a wide range in characteristics such as physical appearance, skin colour, or adherence to some aspects of traditional culture, nor is it implied that New South Wales Aborigines are socially homogeneous or possess a group identity.

However, when inquiries opened up the possibility of obtaining Aboriginal mortality data from the State's death registrations, there was no reason to be concerned with differentiating between those of different degrees of Aboriginal descent, and this simplified the problem of identification.

With the co-operation of the New South Wales Registrar-General's Department, searches were made in New South Wales death registers covering the period 1950 to 1964, to identify and abstract age, sex, and cause of death data for Aborigines in New South Wales. The most difficult obstacle was that Aborigines were not distinguished as such in the records of death; Aboriginal identity had to be inferred from clues supplied with the death notification. The chief clues were places of birth, marriage, residence, death, and burial, and secondary clues were supplied by surnames of the deceased, his parents and spouse, the first names of the deceased, his parents or his children (many first names were soon recognised as being unique or almost unique to the Aboriginal population). Another valuable clue was the name and address of the informant—frequently an officer of the Aborigines' Welfare Board or a close relative of the deceased. A check list was compiled of all known Aboriginal surnames, local place names for Aboriginal communities (including certain streets in country towns where Aborigines were concentrated).[3]

As the search proceeded, this list was added to, and refined, and the local registers where Aboriginal deaths were most likely to appear were identified. It became apparent that a search in the metropolitan district registers had a very low yield, as the only metropolitan deaths which could be easily identified as relating to Aborigines were those where the residential address of the deceased or a relative was given as La Perouse and the surname was exclusive to Aborigines. Therefore the metropolitan registers were ignored, and the search concentrated in country districts known to be inhabited by Aborigines. Approximately seventy registration districts yielded nearly all the deaths retrieved, and entries for these and adjacent districts were searched for each year.

As can be imagined, it was a particularly tedious and time-consuming

[3] The criteria for the identification of Aboriginal deaths are given in Appendix III.

project, justified only by the impossibility of gathering such data by other means. The search yielded 2,213 deaths for the fifteen-year period. The relevant data on age, sex, date of death, primary and contributory causes of death were coded, transferred to punch cards, and tabulated by computer. The causes of death were coded from the original description according to the 7th revision of the International Classification of Diseases, and should therefore be comparable with contemporary cause-of-death statistics for Australia.

It is not claimed that the data are complete, even for non-metropolitan Aboriginal deaths. Where there was doubt, or the possibility of error in the identification of a death as having occurred to an Aboriginal, the record was not included in the series, and deaths occurring to non-metropolitan Aborigines but registered in the metropolitan districts would have been missed. It is claimed only that the data are a fair sample of deaths occurring to those who could be socially or residentially identified as Aboriginal with relative ease, and were resident outside the Sydney metropolitan area. The data cannot be used directly to compute death rates.

AGE AT DEATH

Table 31 shows the deaths within age-groups as a percentage of total deaths for New South Wales non-metropolitan Aborigines for three five-year periods from 1950. The figures may be compared with those in Tables 11, 16, 22, and 25 for various full-blood Aboriginal populations, and in Table 11 with those for Maori, American Indian, and Australia.

Table 31: *New South Wales Aboriginal deaths by age-group, as percentage of total deaths, for 1950–4, 1955–9 and 1960–4*

Age-group	Percentage of total deaths for period 1950–4	1955–9	1960–4
0–	35·9	33·2	30·4
1–	9·2	8·1	8·0
2–4	5·6	4·2	3·0
5–9	1·8	2·2	1·9
10–19	1·7	3·3	2·4
20–29	3·6	2·4	3·5
30–39	5·2	7·2	5·1
40–49	6·8	9·1	6·8
50–59	7·3	7·9	10·5
60 and over	22·7	22·4	28·5
Number in series	715	669	829
PMR (50+)	29·9	30·3	39·0

The distribution of deaths by age-group is disturbingly similar to that for the Northern Territory in 1964-5, and indicates a very high mortality in infancy and childhood even if the higher proportion of children in the New South Wales Aboriginal population is allowed for. It appears likely that infant and childhood mortality in New South Wales was as high in the three periods considered as it was in Western Australian and Queensland full-blood populations in the early 1960s.

Although it is not possible to calculate infant and other age-specific mortality rates directly, it is possible to estimate *minimum* rates for these parameters. The estimation here is based on the assumption that the crude death rate for New South Wales Aborigines is 10 per 1,000 population (a conservative assumption—the crude death rate estimated for the fourteen New South Wales Aboriginal stations for 1954 to 1964 was 11·6) and the crude birth rate 40 per 1,000 (low estimate) or 50 per 1,000 (high estimate). From these assumptions, it follows that births will equal either four times or five times the total deaths, depending on which birth rate estimate is used. The total and infant deaths are available from the data, and it is thus a simple matter to calculate not only estimated minimum infant mortality rates, but also to estimate the minimum population at risk from which all deaths were drawn. (If the age-sex distribution of a fair sample of the same population was known, it would also be possible to estimate minimum age-sex specific death rates.)

The calculations for the infant mortality rates give the following results:

	Infant mortality per 1,000 live births	
	High estimate	Low estimate
1950-4	90	72
1955-9	78	66
1960-4	76	61

It is emphasised that the low estimate is still a minimum value—it would be raised if either the crude death rate was higher than 10 per 1,000 or the crude birth rate lower than 50 per 1,000.

The estimated population at risk for the three periods—the numbers from which the death data were obtained—rose from 14,300 in 1950-4 to 16,580 in 1960-4, these representing five-year averages.

If the estimated natural increase of 3 per cent per annum is applied to 14,300, the increase should have been to about 19,200 by the midpoint of 1960-4. The discrepancy—about 2,600—is no more than might be expected due to underestimating the crude death rate in 1950-4, or under-retrieving deaths in 1960-4, and is not important to the argument advanced

that New South Wales non-metropolitan Aborigines had an infant mortality rate which was at least between three and four times that for Australia during the period considered.

A minimum second-year mortality rate per 1,000 live births may be calculated from the same assumptions already discussed. Based on a high estimated birth rate of 50 per 1,000, the estimates for the three periods are as follows:

1950-4 19 per 1,000 live births
1955-9 16 per 1,000 live births
1960-4 16 per 1,000 live births.

These values were less than half that estimated for the Northern Territory in 1965-7 of 40 per 1,000 live births, yet was around three times the Maori equivalent rate and eight times that for Australia, and indicate that, like the Northern Territory full-blood population, New South Wales Aborigines faced exceptional risks to life in their second year.

For the remaining age-groups, minimum estimated age-specific mortality rates may be calculated from the data for the relevant age-groups during the period 1955-64 by dividing these into an estimate of the age-group population at risk. The latter values may be obtained by apportioning the estimated population at risk (from an assumed crude death rate of 10 per 1,000 $\times$ total deaths averaged for one year) according to the age-distribution determined for New South Wales part-Aborigines in the 1961 Census (reasonably close to the mid-point of the period) (Jones, 1970:10).

Table 32 shows the results of these calculations, compared with rates for equivalent age-groups in the New South Wales non-metropolitan total population for 1954.[4]

The pattern is one of higher death rates at all ages in the Aboriginal group, although the differences are slight between the ages of 5 and 29. The pattern suggests that the most fruitful age-groups within which to examine the causes of death are those under 5 years of age, and those aged 30 and over. Although the rate in the 30-39 age-group is not particularly high (although three times the non-Aboriginal rate), this is an important age-group in terms of Aboriginal ecology because it includes many parents of young and adolescent children. The high proportion of mothers (and presumably fathers) in this age-group who have large

[4] The equivalent figures for 1961 would have been more appropriate; the 1954 calculations were available from another study, and are likely neither to differ materially nor to exaggerate the differences.

Table 32: *Estimated age-specific minimum death rates, both sexes, for New South Wales Aborigines 1955-64, compared with non-metropolitan rates for New South Wales, 1954*

Age-group	Death rates per 1,000 non-metropolitan population	
	Aborigines	Total*
0-4	21·5	7·5
5-9	1·2	0·7
10-19	1·2	1·0
20-29	2·0	1·5
30-39	6·2	1·9
40-49	11·5	4·3
50-59	22·7	10·9
60 and over	73·2	48·8
All ages	10·0	8·6

* The non-metropolitan total deaths will always include an unknown number of unidentified part-Aboriginal deaths, the rates thereby being somewhat inflated above the value for non-Aboriginal deaths.

families has been noted earlier—about one mother in twelve aged 30-39 having ten or more children if she has any children at all.

CAUSE OF DEATH

The leading causes of death during the whole period 1950 to 1964 have been listed in Table 33 in descending rank order as percentages of total deaths, and compared with the percentages for the same causes for Australia in 1961. In addition, an estimated minimum cause-specific death rate has been calculated for each cause listed, based on a crude death rate of 10 per 1,000, and compared with the known rate for Australia. The comparisons show some interesting differences between the two populations.

In most cases these differences are influenced by the age compositions of the two populations; the higher Aboriginal birth rate and higher proportion of children in the Aboriginal population, for instance, will act to raise the crude death rates for causes associated with childbirth and infancy. Causes of death particularly relevant to the younger age-groups will be examined in further detail in a subsequent table. The crude figures are suitable for other purposes, however—particularly for examining causes which are not common enough in the data to provide meaningful figures when further dissected by age.

In Table 33, attention is drawn in particular to deaths from pulmonary tuberculosis, rheumatic heart disease, homicide and suicide, towards the bottom of the table (they are not major causes of death), and nephritis/nephrosis in the middle of the table. With few exceptions, these are

causes of death of adults rather than children, and crude proportions and rates in the Aboriginal population, with relatively few adults, will not be exaggerated by the age structure of the Aboriginal population.

Table 33: *Leading causes of death, all ages, both sexes, New South Wales Aborigines 1950–64, compared with Australia 1961*

Cause of death*	% of total deaths		Rate per 100,000†	
	N.S.W. Aborigines	Australia	N.S.W. Aborigines	Australia
Pneumonia (B31)	14·7	3·2	147	27
Arteriosclerotic and degenerative heart disease (B26)	13·3	30·2	133	256
All other accidents (BE48)	9·0	3·2	90	27
Gastroenteritis, except diarrhoea of the newborn (B36)	8·6	0·5	86	4
Other diseases peculiar to early infancy and immaturity, unqualified (B44)	7·9	1·6	79	13
Vascular lesions affecting the central nervous system (B22)	6·6	13·5	66	114
Malignant neoplasms (B18)	5·9	15·4	59	130
Birth injuries, post-natal asphyxia and atelectasis (B42)	3·7	1·3	37	11
Nephritis and nephrosis (B38)	2·3	1·1	23	10
Congenital malformations (B41)	2·0	1·4	20	12
Malnutrition and anaemia (‡)	1·5	0·4	15	3
Motor vehicle accidents (BE47)	1·5	2·9	15	25
Pulmonary tuberculosis (B1)	1·6	0·5	16	4
Rheumatic heart disease (B25)	1·3	0·8	13	7
Infections of the new born (B43)	1·2	0·2	12	2
Complications of pregnancy, childbirth and the puerperium (B40)	0·9	0·1	9	1
Homicide (BE50)	0·45	0·17	4·5	1·5
Suicide (BE49)	0·3	1·4	3	12
All infective and parasitic diseases (Class I)	4·9	1·1	49	9
All diseases peculiar to early infancy (Class XV)	14·8	3·1	148	26

* Symbol in parentheses refers to the Abbreviated List of 50 Causes, 7th revision, International Classification of Causes of Death.
† Rate based on estimated minimum crude death rate of 10 per 1,000 for the Aboriginal group.
‡ ICD Code groups 290-293, with 280-286.

Deaths from nephritis and nephrosis are the end result of damage to the kidneys in earlier life, particularly as a result of the toxic delayed effects of streptococcal sore throats—an infection which would be anti-

cipated to be both relatively common and relatively neglected medically in the rural Aboriginal population. It is a little surprising to the writer that the proportion of deaths and the estimated crude death rate in the Aboriginal group was only twice that for Australia.

Deaths from rheumatic heart disease—again the end result of streptococcal throat infections—are also about twice as common in the Aboriginal group; nephritis and rheumatic heart disease together account for some 3·5 per cent of all Aboriginal deaths as against about 1·8 per cent of all Australian deaths. This is a reflection of events (streptococcal infections in particular) which will have occurred some fifteen years or more prior to death in most cases, because the disease sequelae are rarely rapidly progressive.

The same remarks apply to pulmonary tuberculosis—the initial infection in most cases will have predated death by many years. Tuberculosis deaths were estimated to be four times more common in the New South Wales rural Aboriginal population than in Australia as a whole—a reflection of higher incidence, perhaps, but also the expected outcome of delayed diagnosis and treatment. The majority of deaths from pulmonary tuberculosis occurred at ages beyond 30, and none occurred to children.

It is of particular interest that the figures show a relatively high death rate from homicide (three times that for Australia) but a low suicide rate (one-quarter that for Australia)—although both causes of death are of minor importance overall, and are based on small numbers. Whatever interpersonal stresses the Aboriginal population of rural New South Wales may have experienced during 1950-64, the solution appears to have been sought in aggressive rather than depressive activity, but the low suicide rate is more unexpected, and merits further investigation. As suicide is rarely resorted to in childhood, the low value for the suicide rate can be partly attributed to a low proportion of adults in the Aboriginal population, but this cannot account for more than a small part of the difference from the Australian value.

At all ages, Table 33 indicates that pneumonia, gastroenteritis, 'all other accidents' (i.e. excluding those due to motor vehicle or railway accidents, suicide and homicide) and diseases of the neonatal period were responsible for a disproportionately large number of New South Wales Aboriginal deaths when compared with Australia—a pattern which was generally similar to that noted for full-blood Aboriginal populations (see Tables 8, 9, 17, 18, 19).

Apart from accidental deaths, the excess mortality rate for New South Wales Aborigines was due mainly to infections at all ages and to deaths

in the neonatal period—a commentary on general community health status, environmental circumstances, and access to and use made of medical services. Since the 1950s, most infections have been susceptible to cure with appropriate antibiotics, and neonatal deaths preventable with adequate pre- and post-natal care of the mother and her child. It is not possible to say to what extent the shortfall in Aboriginal survival of these hazards to life can be attributed separately to poor general health and nutrition, poor use of medical services or poor delivery of the services needed; all three are doubtless implicated. The ultimate responsibility in all fields must rest, however, with the health service and with those who determine the socio-economic environment of Aborigines. Not least among the latter are the general public and the Aborigines themselves, although in the period covered by the mortality statistics the Aborigines had very little—if any—control over their circumstances.

Before leaving the crude cause of death data, a comment on deaths from congenital abnormalities. The incidence of all serious congenital abnormalities taken together is generally held to be about the same in all populations, but about half the deaths may be prevented by appropriate treatment—particularly surgical correction (C. B. Kerr, personal communication, 1971). The higher crude death rate from congenital malformations in the New South Wales Aboriginal group (two-thirds higher than for Australia) may be accounted for by the higher Aboriginal birth rate (probably at least twice that for Australia) without looking any further. However, the possibility remains that a proportion of deaths attributed to non-hereditary causes—i.e. infections, neonatal conditions, malnutrition—may be the outcome of hereditary defects of a biochemical, physiological or immunological nature. Unlike the physical and anatomical malformations, such abnormalities are difficult to detect—particularly in a rural setting and where sudden or early death precludes the carrying out of the investigations necessary to establish a diagnosis. This is to say that genetic factors in Aboriginal mortality cannot be dismissed as of no importance until the possibility has been researched with care. The identification of inherited factors against a background of severe environmental stress is an exceptionally difficult problem, admittedly, except in the case of those relatively rare abnormalities which are known to have a genetic basis and can be detected biochemically or immunologically.

On the other hand, of course, it is equally unreasonable to assume a genetic basis for Aboriginal susceptibility to certain diseases—such as measles or tuberculosis, or malnutrition due to malabsorption—without

convincing evidence. This is particularly true in the part-Aboriginal population where, if 'hybrid vigour' is a valid concept, increased resistance to disease might be expected.[5]

Table 34 lists the leading causes of death in infancy and childhood as determined from the New South Wales non-metropolitan Aboriginal data, compared with Australia for 1961. As well as giving the proportions dying from leading causes within age-groups, minimum death rates per 1,000 live births have been estimated for infants, and children in their second year of life. The percentages and estimated rates point to pneumonia and gastroenteritis as leading causes in all age-groups after the neonatal (under 28 days of age) period, and it is interesting to note the relative prominence given to malnutrition in the second year and worm infestation in the 2-4 age-group. As was suspected, the death rate in infancy from congenital malformations was only slightly higher in the Aboriginal group when based on estimated live births (4·4 per 1,000 *v.* 3·8), and the difference not statistically significant. Control of neonatal deaths (particularly those classed as due to 'immaturity'), together with deaths due to 'pneumonia' and 'gastroenteritis', and to a lesser extent 'accidents', would not only lower the infant mortality markedly, but would exert a greater effect relative to Australia in the 1-4 age-group.

The mortality pattern, both by age and cause, closely simulates that determined for full-blood Aborigines, particularly that of the Northern Territory. The New South Wales part-Aboriginal mortality experience was closer to that in the Northern Territory than to that for white Australians. While rural white Australians run slightly higher risks of death at all ages from most infections, the excess risks are minor when compared with the excess risks for Aborigines anywhere.

Because the New South Wales part-Aboriginal population (even including those classified as full-blood) is more 'white' than 'Aboriginal' in its overall genetic make-up, and because the New South Wales Aborigines co-exist geographically with whites with much lower risks of death, the argument that Aboriginal mortality patterns are determined by socio-economic rather than racial or climatic factors is supported.

An additional pointer to the socio-economic determination of Aboriginal mortality patterns is the pattern of high death rates in children aged 1-4, and the leading causes of death at these ages. This pattern is characteristic of populations under severe ecological stress, indicating a failure

[5] Although the part-Aboriginal population may be 'hybrid' in terms of racial origins, it may also be 'inbred' if marriages have been restricted to members of small communities over several generations.

Table 34: *Leading causes of death in infancy and childhood, New South Wales non-metropolitan Aborigines 1950-64, as percentage of total deaths in age-group and estimated minimum rate, compared with Australia 1961*

Age-group and cause	% deaths in age-group		Death rate per 1,000 live births	
	N.S.W. Aborigines	Australia	N.S.W. Aborigines*	Australia
Aged under 1 year				
Other diseases of early infancy and childhood (769-776)	28·5	27·9	18·8	5·5
Pneumonia (490-493)	22·8	7·1	15·0	1·4
Gastroenteritis (571)	14·5	2·1	9·6	0·4
Birth injuries, post-natal asphyxia and atelectasis (760-762)	8·3	25·3	5·5	4·9
Congenital malformations (750-759)	6·6	19·5	4·4	3·8
All accidents (E800-E965)	5·9	2·9	3·9	0·6
All causes	100·0	100·0	66·1	19·5
Aged 1 but under 2 years				
Pneumonia (490-493)	34·4	16·8	5·8	0·29
Gastroenteritis (571)	30·6	5·6	5·1	0·01
All accidents (E800-E965)	8·1	27·0	1·4	0·46
Malnutrition (280-286)	4·3	—	0·7	—
All causes	100·0	100·0	16·8	1·7
Aged 2 to 4 years				
Pneumonia (490-493)	17·2	7·7	†	†
Gastroenteritis (571)	17·2	2·1	†	†
All accidents (E800-E965)	17·2	35·9	†	†
Worm infestation (129-130)	9·7	0·4	†	†
Meningococcal and other meningitis (057, 340)	6·5	3·0	†	†

* Based on estimated crude death rate of 10 per 1,000 and crude birth rate of 50 per 1,000.
† Rates not calculated or estimated for the 2-4 age-group: it is likely that the Aboriginal 2-4 death rate is about ten times the Australian rate for the same age-group, which is 0·86 per 1,000 population.

to cope with—perhaps even to appreciate—the problems of rearing children from weaning age to the attainment of a degree of independent survival in later childhood. There is no evidence that this period was exceptionally dangerous for the full-blood Aboriginal child in the nomadic culture,[6] and it is a very safe period of life for white children in Australia. In fact, a white child in Australia, having passed his first birthday, is so 'safe' from threats to his life that little attention is paid to him by the

[6] Abbie (1966) quotes estimates that 7 per cent of nomadic Aboriginal children born died in the 1-4 age period (13 per cent having died in their first year). The derivation of this estimate is not given, but may well have been based on Professor Abbie's experiences with Central Desert Aborigines, who faced the most severe environment in Australia.

health authorities until his first school medical examination, other than to offer immunisation and other services available at all ages. While this may be appropriate in white Australian society, the mortality pattern suggests it is far from appropriate in the Aboriginal community, because the Aboriginal child after his first year faces as great or greater risks of death than the white child faces in his first few weeks—the most dangerous year for the latter until he reaches his sixties.

MORTALITY IN OTHER STATES

Part-Aboriginal mortality in other States has not been studied in detail except for the population of single communities—mostly settlements and reserves. The number of deaths occurring in a discrete Aboriginal or part-Aboriginal community is necessarily small, at about 1 per cent of the population per annum, so that analysis of such data by age-group or cause is not particularly informative. As far as can be judged from the small numbers of deaths reported for selected communities, the patterns seem to follow that outlined for New South Wales, and most of the variations from this pattern may be no more than might be expected from small samples.

There is one aspect of small community size that has not been mentioned so far, but deserves mention at this point because of its likely impact on Aboriginal health. This is the effect of small numbers upon the community's awareness of its own health problems. A community of fifty N.S.W. Aborigines—a typical small 'fringe' community—would be expected to average about two births per year, one death every second year and one infant death every eight years. Deaths at this frequency are rare and apparently random events. In a town of 5,000 people, however, the same relative risks would result in 200 births, fifty deaths, and twelve infant deaths in a year. It is apparent that a doubling of the infant mortality rate (say as a result of a widespread epidemic of gastroenteritis) would probably attract no attention in the small community but would be almost certainly remarked upon in the town of 5,000—at least by the local undertaker, minister of religion, general practitioner, hospital matron, and registrar of births and deaths. Provided that communication among the townspeople was as efficient as it usually is in small towns, it would be expected that the word would get around that it was a bad year for gastroenteritis; that individual parents and citizens would take some sort of avoiding action; and that the year would be remembered as an exceptional and unhappy community experience. A seed of knowledge—stimulated by 'drama', fear, and bereavement—has

been sown for the town, but not for a hundred communities of fifty persons who shared the identical experience without knowing it. For small communities, this time-dilution of relatively uncommon events to the point of apparent insignificance can only be countered by widening the communication network and concept of 'community'—a task for the statistician and the news media. The proposition is of course no different from that of publishing lottery results—who would invest in lotteries if the only wins one heard of were from tickets sold by one village newsagent?

In Queensland, the data used to calculate crude birth and death rates and an infant mortality rate for full-blood missions and settlements (published by Jose *et al.*, 1969: Tables I and II) has been used to calculate the same parameters for two mainly mixed-race Aboriginal settlements—Cherbourg in south-east Queensland and Palm Island off the north coast near Townsville, for the period 1962-6. The results are as follows:

	Cherbourg	Palm Island	Total
Crude birth rate per 1,000	46	39	42
Crude death rate per 1,000	16·5	14·4	15·4
Infant mortality rate per 1,000	153	106	129

The infant mortality rates for Cherbourg and Palm Island are considerably higher than the value calculated for full-bloods for six settlements around the Gulf of Carpentaria for the same period (86 per 1,000 live births), and make one wonder what extra hazards were faced by part-Aboriginal infants in these settlements. Both had relatively large populations (average for the period 1,164 and 1,415 respectively) compared with the full-blood communities, which averaged 468 persons for the period, and it is tempting to suggest that this, together with the more impersonal contact between the inhabitants and the staff responsible for health and medical care, and the greater likelihood that institutionalised *part*-Aborigines are already selected to an extent by socio-economic handicaps and domestic or personal problems, are important influences.

It would appear from the figures that death rates in Cherbourg and Palm Island children in their second year are also likely to be exceptionally high, but this cannot be confirmed from the data available to the writer. Sandars (1964), in a report of investigations into parasitic infestations at Cherbourg, observed that the 'death rate' of children aged 3 months to 5 years, and hospital admissions (presumably from the same age-group) 'fell dramatically' from 1962-3 after mass treatment of the population

with anti-helminthics. Her figures are for the number of deaths in this age-group, and rates cannot be calculated as no population base is given. Since Jose's data cover the period 1962-6—following the fall observed by Sandars—the infant and childhood mortality are likely to have been extraordinarily high before 1962.

In Western Australia, Edmunds and his colleagues (1970) have drawn attention to the disproportionate contribution of Aborigines to childhood mortality in that State. Aboriginal deaths were identified among child deaths (under 5) for the years 1967 and 1968, by methods similar to those employed by the writer for New South Wales Aboriginal deaths. In 1968, public hospitals helped the study in Western Australia by classifying deaths according to race—'white', 'part-Aboriginal' or 'full-blood Aboriginal'.

The data obtained by Edmunds and his colleagues show that whereas the Aborigines represent about 2·5 per cent of the total population of Western Australia, they contributed about 20 per cent of the infant deaths and 27·5 per cent of deaths in the 1-4 age group. Furthermore, if the infant deaths are divided into those occurring in the neonatal period (under 28 days of age) and the remainder post-neonatal (aged one month to one year), the former contributed 12 per cent and the latter no less than 42 per cent of the total for the State.

The authors also compare the ratio of neonatal to post-neonatal deaths in the white and Aboriginal groups, showing that whereas white post-neonatal deaths were an 'acceptable' 20 per cent of infant deaths, post-neonatal Aboriginal deaths were an 'entirely unacceptable' 56 per cent of infant deaths.[7] As the authors cautiously state, this high figure 'raises suspicions about the environmental conditions in the area concerned'.

Of deaths at ages 1-4 years, 43 per cent in the white group were due to an infection, as against 92 per cent in the Aboriginal group. Although the authors were not able to calculate rates for death in the various age-groups, it is evident that the pattern of mortality is again like those for other Aboriginal populations considered in the present study. The authors' concluding words would be appropriate in any State or Territory:

> this relatively large number of deaths in Aborigines in the age groups concerned represents merely the exposed portion of a very large iceberg of ill health.
>
> Most of the severe, acute and recurrent infections, often combined with malnutrition, as well as less dramatic illnesses in Aboriginal children, receive intensive and

[7] 'Unacceptable' because the proportion of infant deaths occurring after the first 28 days of life has been reduced to less than 20 per cent in countries where the population is generally 'healthy' and maternal and infant health services are both adequate and effective.

adequate treatment which saves the majority of these patients. The costs in general misery and chronic ill health, as well as the financial burden on the people of the state, have not been calculated but must be very considerable.

The data referred to above were for both full-blood and part-Aborigines in Western Australia, where part-Aborigines are in the majority by a ratio of six to five according to the Department of Territories' figures quoted in Table 1. Of all deaths under age 5, 22 per cent were in the 1-4 age-group in Edmunds's series for all Aborigines in 1967–8, and 32 per cent in the figures for Western Australian full-bloods given in Table 13. Furthermore, the post-neonatal deaths were 56 per cent of all infant deaths in Edmunds's series, but 67 per cent of all infant deaths in the full-blood series. The implications are that part-Aborigines in Western Australia experience lower risks of death after the neonatal period than do the full-bloods, and that the latter were the major contributors to Edmunds's data for deaths under 5 years of age.

7 ABORIGINAL MORTALITY AS AN INDEX OF ABORIGINAL HEALTH

Although mortality statistics have been stated to reveal little about the health of the living, there is no doubt that Aboriginal mortality statistics provide some very positive information about Aboriginal health ecology—information which is not generally obtainable from morbidity statistics because of relatively greater gaps in knowledge on the subject of Aboriginal morbidity.

In terms of the relevance of the mortality pattern to health status, the iceberg analogy is valid in so far as it emphasises the greater bulk of the underlying disease pattern and the various factors which lead to death as a result of disease. But whereas the total bulk of an iceberg may be estimated from the bulk of the visible part, no valid estimate of morbidity can be made from mortality except that the incidence of disease must at least equal the incidence of deaths from the disease.

The advent of death removes the 'cause' from the morbidity picture of a population, and it is obvious that a reduction in mortality achieved through therapy, particularly mortality from diseases which are prolonged in their course, or tend to recur in the same individual, will *raise* morbidity prevalence rates. This is only to say that the prevention of death by the vigorous treatment of serious established diseases will not create a healthier community as judged by morbidity. Community health status can only be improved through prevention of disease (reduction in incidence) and complete and lasting cure of established disease (reduction in prevalence beyond that resulting from a fall in incidence). It is a strange paradox that the overall morbidity status of Western societies—in terms of the proportion of the population enjoying good health—may in fact be

falling due to the effects of successful palliative medicine and the prolongation of life, and be lower than the morbidity status of many 'primitive' communities where mortality is high and the expectation of life is short. However, Western societies have chosen, to a degree, to exchange child health and child mortality problems for geriatric health problems and to meet the cost, so far, of prolonging life without necessarily prolonging health.

Accepting that Aboriginal mortality statistics may be a poor indicator of levels of health (in terms of the prevalence or incidence of physical illness and disability), they nevertheless point to broader health patterns, particularly in relation to the physical, economic, and social environment. One of the most significant features of the Aboriginal mortality pattern lies in the age and cause distribution relative to Australian norms—the high rates for death in early childhood, and in particular the high death rates in the second year. Such deaths may be 'caused' by specific diseases, but the pattern itself points to deeper, more fundamental causes: the inability of the Aboriginal community to cope very well with the rearing of children through a period which has become the safest period of life for white Australians, and for other highly developed Western societies.

This 'inability to cope' is relative, and it is necessary to remember that white Australians were coping no better at the turn of the century. Death rates for white Australian children have come down as the result of a number of factors—advances in the control and treatment of communicable and other diseases; improvement in the physical environment and in nutrition through rises in the standard of living; greater availability of health and medical services (particularly with increasing urbanisation and increasing expenditure by governments on services and subsidies); and not least an awareness of where the mortality problems lay and the ability to determine priorities for action. This last factor has been dependent on the long-term collection and collation of mortality statistics, and their comparison with those of other countries, some of which have led Australia in reducing hazards to life in childhood.

The technical knowledge and the major medical facilities to control or reduce childhood mortality already exist, but cannot be delivered to the Australian Aborigines because of cultural or social and economic barriers, 'a standard of living gap', and to some extent a physical and demographic barrier with the majority of Aborigines distributed in small communities in rural areas. Appropriate preventive and curative services need to be accepted and utilised by the target population as well as made available, and the writer is not suggesting that high Aboriginal childhood

mortality can be materially reduced without active participation by Aboriginal communities and individuals; but until such time as non-utilisation is the only factor operating (and even then?) Aboriginal childhood mortality can be looked upon as an operational index of Australian public health as well as an index of Aboriginal health.

From the humanitarian viewpoint, childhood deaths represent individual and family bereavements and suffering children, and are to be avoided because of this. From the ecological viewpoint, they represent wastage, not only of future human resources embodied in the dead child, but the personal, family and—to a relatively small extent in the Aboriginal context—community resources which have been expended in pregnancy, childbirth, and in feeding, clothing, and caring for the child up until the time of his death. For a family in poor circumstances, the necessary diversion of limited resources—physical and psychological—to nourish and provide for a child who subsequently dies before reaching a stage of self-sufficiency can be viewed objectively as the relative deprivation of the surviving members of the family to no purpose. This argument for the prevention of deaths in childhood stands even if there were no humanitarian arguments. There is also the proposition that reduction in infant and child mortality will be conducive to a fall in the birth rate and a reduction in average family size, although there seems to be little certainty in such a prediction unless the social and economic climates also change in an appropriate direction.

The cause-of-death figures for Aborigines, like the age at death, also indicate inability to cope with health hazards to young children; none of the causes—either for full-blood or part-Aboriginal populations—are unusual in themselves, but the high proportion of deaths from infection in an age when most infections can be prevented or controlled by appropriate antibiotic agents suggests factors operating at several levels. Firstly, a high incidence and prevalence of infection in Aboriginal communities; secondly, a high threshold for motivation to seek treatment; thirdly, difficulties in obtaining treatment; and fourthly, a suggested increased susceptibility to severe or overwhelming infection arising out of the synergism between malnutrition and infection.

The high threshold to motivation to seek treatment would appear to be a particularly important factor. This opinion is partly based on personal observations, and partly arises from the opinions expressed by others in everyday contact with sick Aborigines. It is often called 'apathy'—not a very appropriate term in either of its two usual senses of 'insensibility to suffering' or 'indolence of mind', and in any case a term which could

perhaps be applied as reasonably to the Australian public and to the authorities responsible for solving Aboriginal health problems as to the majority of Aboriginal mothers.

An Aboriginal child is likely to suffer from repeated episodes of diarrhoea, respiratory and other infections each year, most of which will be relatively trivial and will clear up spontaneously, and will not come to the attention of the health and medical services. The occasional infection from among the many will be severe and lead to hospitalisation, when the mother or guardian realises that it is no 'ordinary' episode, and a percentage of these cases will die—partly because treatment is delayed, and partly because the child is likely to be anaemic, malnourished to a degree, or suffering from other concurrent infections and disabilities. It is proposed that high childhood mortality may be thus attributed in part to a motivation-threshold which is kept high by the high levels of 'background' infection among Aboriginal children. The latter will be considered in the sections dealing with morbidity, and attention is drawn to it here only as another argument against attempts to solve the Aboriginal child mortality problem in hospitals, or in infant health clinics and doctors' consulting rooms.

If Aborigines are to be assisted to cope with the problems of child rearing, this assistance has to be introduced primarily at the household level—through the door of the dwelling, not the doors of the hospital and the clinic. Above all else this approach needs sufficient, competent, Aboriginal-accepted field staff—and this is a staffing problem which does not appear to have been tackled successfully in Australia as yet. It is not an easy problem to tackle because of the scattered distribution of Aboriginal communities, but until it is tackled a continued high mortality rate in Aboriginal children must be expected.

In 1968, the writer estimated from mortality statistics available for full-blood and part-Aboriginal populations in the early 1960s that Aborigines, comprising about 1 per cent of the Australian population, contributed at least 2 per cent of the births, 10 per cent of infant deaths, at least 28 per cent of deaths in the second year of life, and about 9 per cent of deaths of 2-4 year olds (Moodie, 1969:184).

Because of the deficiencies in the data, the figures quoted can be no more than intelligent guesses, but they indicate, perhaps better than all the other figures which have been quoted in this study, the severity of the child health problem in the Aboriginal population. In terms of numbers it is not a big problem to handle. The pity of it is that the situation outlined cannot be re-evaluated year by year, nor can steps be taken to

improve the picture be evaluated as to their successes and shortcomings unless the relevant data are collected, collated, analysed, and published.

The writer can imagine no greater potential stimulus to Australia (and to Aborigines themselves) to make a concerted attack on Aboriginal health, social, and economic handicaps than the regular production of the relevant statistics, and to this end Aboriginal communities and community leaders might be wise to insist that Aboriginal social identity be included in the appropriate State and Commonwealth records and notifications, as happens in the case of the Maori and the American Indian populations. The contribution of statistical visibility to the noticeably better mortality patterns of the latter two populations cannot be measured, but should not be underrated.

8 MORBIDITY IN ABORIGINAL POPULATIONS

There exists a considerable body of data on Aboriginal morbidity, most of it relating to disease prevalence in circumscribed communities. The aim of this section of the study is to piece together the published material relating to disease prevalence (to compare morbidity patterns in Aboriginal and other communities) and in this way construct a picture of the 'bottom of the iceberg' of the health of Aborigines during the last decade or so. It is possible to deduce something of disease incidence from disease prevalence studies if the data are related to age, but generally speaking the incidence of disease can only be accurately established from longitudinal surveys—of which there are very few in the field of Aboriginal health.

In order to appreciate the significance of the material to be presented, the reader needs to have a very clear idea of the difference between prevalence and incidence—two terms which have specific meanings epidemiologically, although they are frequently used as if they were interchangeable terms by medical writers and others.

Prevalence refers to the number or proportion of persons affected in a community at a point in time ('point prevalence') or over a defined interval of time ('period prevalence'). Incidence refers to the number of new cases (of diseases, crimes, car smashes or whatever) occurring within a defined interval of time. Both prevalence and incidence must be related to a population at risk before the numbers can have any relative meaning —if comparisons are to be drawn—although the numbers to whom the events occur, taken alone, indicate the absolute 'load' which may have to be handled in dealing with a problem. The latter is obviously essential information for planning purposes.

Prevalence studies are weighted towards detecting long-duration illnesses and chronic disabilities; transient illnesses, though they may be major contributors to the pattern of ill-health in a community, may be missed—particularly those diseases which tend to occur in epidemics or are influenced by season. Incidence studies, on the other hand, should detect the occurrence of transient illnesses if the period of the study is long enough, but unless combined with prevalence data it is likely that the presence in a community of low-incidence long-lasting illnesses may not be detected.

Certain short-term communicable diseases produce long-lasting or permanent changes in antibody patterns, and may be studied in a prevalence survey with serological and immunological techniques. Thus a picture of disease incidence may be reconstructed from age-specific test reaction patterns. Such reconstructions are prone to error through the effect (on the age-specific prevalence patterns) of changes in the age-specific incidence which commonly accompany ecological changes. It is not safe to assume a steady-state behaviour of a disease over a long period with respect to attack-rates at various ages, particularly if human behaviour and the environment are changing.

Morbidity data for Aborigines, as for most populations, are of several types. Firstly, there are health surveys in which an attempt is made to record all abnormalities in a total community, or a component of a community selected on the basis of age or other relevant parameter. 'All abnormalities' in this type of survey are usually restricted to those which may be detected through a standardised history-taking, clinical examination, and some basic laboratory tests such as haemoglobin estimation, or examination of the faeces and urine. Secondly, there are surveys directed towards detecting one or several—usually related—specific diseases. Such surveys may be for eye diseases, diseases of other organs or systems, worm infestations, serological reactions specific to certain communicable diseases, and so on. Within both these two types of survey, and within limits set by the range of diseases encompassed, the information retrieved is fairly complete, includes the population at risk, and allows calculation of rates which may be used for comparative purposes. It has been pointed out earlier that there are difficulties in comparing morbidity statistics for populations surveyed by personnel who have not received an identical training or developed the same skills, and that this is particularly so when the populations compared have different cultural backgrounds influencing their symptom-patterns or giving rise to unclassified diseases.

Thirdly, there are data arising from a voluntary act by members of

the population in presenting themselves for diagnosis or treatment—hospital admission, outpatient, baby health clinic, immunisation clinic, mass chest X-ray clinic, and notifiable disease statistics. Depending on what might be termed the 'co-operation' of the population such data vary widely in completeness, but generally speaking the data are not suitable for assessing community health or morbidity. Mass chest X-ray clinics are not to be confused with mass chest X-ray surveys; in the latter it is assumed that an effort is made to identify and include the total population for whom the survey is designed, including absentees and refusals.

There are several alternative ways of examining Aboriginal morbidity —by taking communities one at a time and looking at the total morbidity pattern, or as much of it as has been described; or by taking diseases and disease groups one at a time and looking at their prevalence in different communities. The first method is to be preferred if the object is to assess the overall burden of illness borne by a particular community, but the 'complete picture' it purports to present is something of an illusion unless the investigation has been exceptionally thorough and includes longitudinal studies by a multidisciplinary team. Because of the multiple disease entities and related factors which have to be handled concurrently, the 'total morbidity' approach may be particularly difficult for those with no medical background to follow.

On the other hand, the 'one-disease-at-a-time' approach is easier to follow and provides greater opportunity to examine epidemiological and ecological factors in Aboriginal ill-health, but suffers from the great disadvantage that it tends to compartmentalise the Aboriginal health 'problem' and its impact on the community.

The choice has really been decided for the writer by the piecemeal and fragmented nature of Aboriginal morbidity data, and the second approach has been selected—modified to the extent that related diseases and disease-groups will be considered together when this is appropriate.

As with Aboriginal mortality, it is not possible to describe or evaluate morbidity in the 'total Aboriginal population' of Australia. In this case it is not due to a particular deficiency in the quality or availability of data referring to Aborigines: in some ways, the health (morbidity pattern) of Aboriginal communities is better documented than that of Australia, or most Australian communities. It is equally impossible to evaluate 'total Australian population' morbidity; however, the considerable cultural, socio-economic and environmental homogeneity throughout the white Australian population—State by State, area by area, city by city—

allows generalisations to be made from small but representative samples with fair confidence. Representative samples of the Aboriginal population cannot be said to exist within a single community—particularly within institutionalised communities which are more frequently studied because they are 'captive', accessible and already defined.

The system which has been followed here is to examine published data on Aboriginal morbidity, following the sequence in which diseases are listed in the International Classification of Diseases as closely as possible.

The first category to be considered is the group 'Infective and Parasitic Diseases'. This category is also the largest in terms of data available, and second only to 'Accidents, Poisonings and Violence' in the number of separate entities it includes.

9 MORBIDITY FROM INFECTIOUS AND PARASITIC DISEASES

The infectious and parasitic diseases are the communicable infections with biologic agents which are spread from person to person in most cases, but the category includes some infections acquired from animals (the zoonoses) and a few diseases caused by free-living micro-organisms from the soil.

Infection plays a part in many other diseases not included in the category, but the infection is generally either non-specific with respect to the agent (as in 'bronchitis') or, if specific, considered to be secondary to another disease or a breakdown of body defence mechanisms (as in pneumococcal pneumonia). Certain acute respiratory infections, such as influenza, are included with other respiratory system diseases in a separate category, although a number of them are patently 'communicable' or 'infectious'.

The infectious and parasitic diseases are theoretically preventable by one or more of the classical approaches to communicable disease control—removal of the source of infection (by treatment, isolation); interruption of transmission (through control of the human environment and alteration of human behaviour); and protection of susceptible individuals (by immunisation and other prophylactic measures).

Communicable diseases, in their behaviour in populations, may be 'epidemic' (measles); 'endemic' (worm infestations); or 'sporadic' (tuberculosis, leprosy). In the untreated individual, communicable diseases may be 'acute' or transient (chickenpox, gastroenteritis); 'sub-acute' or more prolonged (typhoid fever, malaria); or 'chronic' (hydatid infections, syphilis). These characteristics of disease behaviour are not fixed for

specific disease entities—they merely describe how a disease is behaving in a population at a specified time, or in a specified individual, although each disease has its typical or usual pattern of behaviour.

Endemicity (or epidemicity), incidence, prevalence, the duration of infection, and host resistance or 'herd immunity' (acquired immunity in a population) are all interrelated. Apart from the duration of infection, and resistance, which reflect more the biological characteristics of the infecting organism and the human species in general, all are very much influenced by human behaviour and the domestic and community environment as determined by human behaviour.

INTESTINAL INFECTIOUS DISEASES

Except for protozoal diseases such as amoebic dysentery, the diseases in this group are generally acute illnesses and frequently occur as epidemics.

Cholera, the most devastating among this group, has never been recorded among Aborigines or identified in Australia since European settlement. There is currently an extension of this disease throughout the world—particularly the El Tor variant—and Aborigines may be threatened by it, and threaten then to constitute a reservoir for it, should it gain a foothold in metropolitan communities and spread from there. The danger of its introduction has been remarked upon in the 'Report from the Select Committee on Grievances of Yirrkala Aborigines' (1963:22). Because of a prolonged carrier state, El Tor cholera may be introduced into the developing north by workers in the mining and construction industries transferring from cholera areas. It is only in the Aboriginal communities that cholera is likely to spread, if introduced at all.

Typhoid fever has been a persistent disease among Aborigines, as it was in the white population half a century ago. There were outbreaks on at least two Arnhem Land missions in 1968 to the writer's knowledge, although no figures have been sighted. Most *Northern Territory Reports* up to the most recent (which does not discuss health) have noted sporadic cases, presumed to have occurred in Aborigines because of the localities given—Bathurst Island, Bagot, Maningrida, Oenpelli—between 1960 and 1965. Intensive immunisation campaigns and case-finding surveys appear to have limited the outbreaks which have occurred, and there is no evidence, in the absence of incidence figures, that typhoid fever is a major health problem.

Information available on *Salmonella* (other than *S. typhi*), *Shigella* and bacterial food poisoning among Aborigines is almost entirely limited to

the bacteriological findings from surveys of faecal samples. Rodda (1963:3) in Queensland reported species of *Salmonella* and *Shigella* as endemic 'in most populations' (it is not clear whether he was referring to Aboriginal populations exclusively) but it is clear that pathogenic strains of these organisms were not difficult to find at Palm Island Settlement, and he reports three strains of *Shigella flexneri* (a potent cause of bacillary dysentery in adults and gastroenteritis in children) among forty-four Aborigines at Woorabinda Settlement following a gastroenteritis outbreak in 1963, although he was not able to state positively that the outbreak was due to *Shigella* infection.

Lee (1953:3) investigated intestinal bacteria responsible for dysentery, gastroenteritis, and *Salmonella* food poisoning at Palm Island, and found the grouped organisms to have a high prevalence in children under two years of age (13 per cent), the prevalence falling progressively to 2 per cent in those over the age of 10 years. Lee remarked '. . . an immediate reduction in incidence and severity of infection in the youngest age-group should follow if weaning is delayed from the present six months to about nine or ten months' but he found the highest prevalence in those aged 18 to 24 months—a significant observation in view of the high second-year mortality in Aboriginal populations.

Sutton (1966), investigating the prevalence of heat-resistant strains of *Clostridium welchii* among Aborigines and others in the Lismore area of the New South Wales North Coast, has published some comparative figures. The heat-resistant strains can survive cooking and produce food-poisoning, particularly where refrigeration or safe food storage is not used. Sutton found that 19 per cent of Aborigines were carriers as against 6 per cent of the general public tested, and more importantly, that the percentage of Aboriginal households with one or more carriers showed a marked inverse relationship to the standard of hygiene:

Living conditions	Proportion of households with carriers	Percentage with carriers
'Good hygiene'	4/20	20
'Fair hygiene'	18/32	60
'Poor hygiene'	24/26	92

(After Sutton, 1966:71)

Of further interest was the finding that the prevalence of carriers varied only slightly between summer and winter, but there was a rapid turn-over in the strains (serotypes) detected in individuals, pointing to a high rate of re-infection from the Aboriginal environment.

The heat-resistant strains, together with the much more common heat-sensitive strains, are 'normal' inhabitants of the intestinal tracts of man and animals and are not pathogenic in this situation, but the carrier rate for the heat-resistant strains may be regarded as an index of exposure to other (pathogenic) organisms in the environment—particularly those which, like the *Clostridia*, can survive drying, such as the eggs of intestinal worms and the cystic forms of intestinal protozoa.

Amoebiasis, a protozoal infection (commonly symptomless) with the organism responsible for amoebic dysentery, is another indicator of domestic and community hygiene. The prevalence of carriers in Aboriginal communities has been investigated particularly in Queensland, where observations have been made over many years by the Queensland Institute of Medical Research. Some recent figures for the prevalence of amoebiasis in Queensland Aboriginal communities showed rates varying from 46 per cent at Mitchell River Mission to nil at Hope Vale Mission in 1970, among children under 16. Fluctuations in prevalence from year to year (from 7 per cent in 1969 to 46 per cent in 1970 at Mitchell River Mission) suggest the occurrence of epidemics on top of an endemic situation, as would be expected. The age-prevalence figures at Palm Island (1965), Mitchell River Mission (1969 and 1970) and Edward River Mission (1970) indicate that the highest prevalence is reached around the age of 5 years, with up to 80 per cent of children aged 3 to 5 being infected. Amoebic cysts have been recovered from soil samples from around drains and taps at Mitchell River.

It is believed that *Entamoeba histolytica* normally behaves as a harmless commensal unless stirred into pathogenic activity by other conditions—bacterial infection, malnutrition, whipworm infestation having been suggested as precipitating factors. In its pathogenic behaviour, it is not only a cause of debilitating recurrent or chronic diarrhoea and dysentery, but can cause infections elsewhere than in the bowel—in skin, around the genitalia, and in the liver (liver abscess) and the brain (cerebral abscess). A free living amoeba, *Acanthamoeba castellani*, occasionally invades the central nervous system, causing a meningitis. Among Aboriginal children amoebiasis is usually only one of many concurrent intestinal infections and infestations, and with its dual behaviour as a commensal and a pathogen it is difficult to establish its exact contribution to Aboriginal morbidity. That it does contribute, particularly among Aboriginal children in unhygienic environments anywhere in Australia, there can be no doubt.

Among a number of other protozoal species found in Aboriginal

populations, only *Giardia lamblia* has been suspected as a cause of morbidity, and then only when the infection is heavy. This organism may cause diarrhoea but does not cause ulceration of the bowel or spread to other organs, like *Entamoeba hystolytica*; like the latter it has been observed to reach heavier loads in growth-retarded Aboriginal children (Jose, Welch, and Frazer, 1970:17). Jose and his colleagues believed that these infections played no part in initiating growth retardation, but may have increased susceptibility to diarrhoea and respiratory infection, or interfered with the assimilation of food (Jose, Self, and Stallman, 1969); a significant growth spurt was produced in half of an infected group by treating this half with mepacrine—a fairly effective drug against *Giardia*. The organism was found in high loads in up to 70 per cent of growth-retarded children from Queensland missions and settlements (Jose and Welch, 1970). It appears to reach high prevalence earlier in life than is the case with *Entamoeba histolytica*; it had the same incidence of 70 per cent in the 0-4 and 5-9 age-groups at Palm Island Aboriginal Settlement in 1965, whereas the prevalence of amoebiasis was 48 per cent and 72 per cent respectively in the same age-groups at the same time (Sandars, Hutchins, and Tanner, 1965).

Diarrhoeal disease due to other or unspecified organisms completes the diseases within the group 'intestinal infectious diseases', and 'gastroenteritis' is usually recorded under this heading unless the case has been investigated in a microbiological laboratory.

Gastroenteritis due to virus infections is not well documented in the Aboriginal population, with the exception of an outbreak at Wellington —a town in the Western Slopes division of New South Wales—in late 1965. Of fifty-eight patients admitted to hospital, thirty-eight were Aboriginal, and Echovirus Type 1 was isolated from five of these (Field *et al.*, 1968). The authors comment that this was not an explosive, single-source epidemic—cases came from three separate Aboriginal settlements over a period of about two months. It particularly affected children aged 2 months to 2 years, and there were three deaths (race unspecified). The virus was also isolated from kitchen and laundry sullage water from two of the Aboriginal communities, the laundry sullage possibly being contaminated by nappy-washing. Poor hygiene and sanitation in the Aboriginal communities was held responsible for the higher incidence in the Aboriginal group.

With reference to this epidemic, the authors observed that the average time spent in hospital was 13 days—a contribution of very nearly 500 patient days from the Aboriginal group, and an indication of the potential

cost benefit of even quite expensive preventive measures and environmental sanitation. It is possible, however, that hospitalisation was prolonged because of the laboratory investigations or because of co-incidental diseases requiring treatment or investigation.

Gastroenteritis as a clinical entity (unspecified as to cause) is mentioned throughout Australia as a cause of morbidity in Aboriginal children, but there are no figures for its incidence in the community. There are some figures for hospital admissions: Davidson (1957) stated that of 1,779 Aboriginal admissions in the North and North-West areas of Western Australia, 154 were due to 'diseases of the digestive system', of which '115 are accounted for as diarrhoea or gastroenteritis' (p. 54). In this series, over three times as many admissions were for respiratory diseases, over twice as many for both diseases of the skin and connective tissue (mostly infections, including scabies) and accidents and violence. The proportionate deaths from these causes in Western Australia relative to one another are generally similar, except that skin diseases do not feature.

HELMINTHIASIS

Helminthiasis, or worm infestation, is considered next although it appears towards the end of 'infectious and parasitic diseases' in the International Classification. The group includes parasitic infestations of the blood, lymph, and other tissues, but few of these infestations are known to have occurred in Australia in recent years. The balance of this group—worms parasitic in the bowel—are an important cause of morbidity in the Aboriginal population, and the group is considered next because the intestinal parasites share epidemiological features with the intestinal infections.

Tapeworm infestations are common, particularly infestations with the minute ('dwarf') tapeworm, *Hymenolepsis nana*. This parasite is not regarded as harmful except when present in enormous numbers, when it may cause abdominal pain, diarrhoea, and nervous phenomena such as convulsions (Manson-Bahr, 1960:977). Mackerras (1955:11) found a prevalence of 25 per cent at Palm Island Aboriginal Settlement. Welch (1969:14) found this parasite at high prevalence among children at seven other settlements and missions in Queensland, the overall prevalence in children being 28 per cent, and the prevalence in children 11-15 years being 63 per cent. In this series, it was the second most common parasite found, being led by whipworm. The parasite has also been recorded in Aboriginal communities on the North and South Coasts of New South

Wales,[1] in the Victoria River-East Kimberleys area of north-west Australia,[2] and in East Gippsland, Victoria,[3] and the distribution may be assumed to be Australia wide (although possibly absent from the drier parts of the interior), with higher prevalence rates in the tropical areas.

The large beef tapeworm, *Taenia saginata*, has been recorded occasionally, two infestations being reported by Sandars, Tanner, and Kay (1966: 12) from Cherbourg Aboriginal Settlement.

The most serious infection is that with *Hydatids*, an intermediate stage of the dog tapeworm *Echinococcus granulosus*, which normally passes its intermediate stages in sheep and cattle and commonly in kangaroos. Dogs infected from feeding on hydatid-containing offal are necessary to produce the infection in man, via tapeworm eggs excreted in the dog's faeces. As the stage in man cannot be detected by examination of the faeces, morbidity data depends on cases of hydatid infection diagnosed after symptoms are produced, aided by a somewhat non-specific skin test (the 'Casoni' test).

Stein and McCully (1970:848) surveyed major hospitals in Western Australia for the period 1957-67, and counted forty-five confirmed cases of hydatid disease, of which thirteen were Aborigines (31 per cent). The authors calculated that the annual incidence for Western Australia as a whole was 0·4 cases per 100,000 population, but for the Aboriginal population it was almost 7 cases per 100,000, nearly eighteen times higher. They also noted that only nine cases were officially notified in Western Australia during the period—an indication of the unreliability of notification figures for establishing the incidence of disease.

Caseley-Smith (1959:485) found nine out of twenty-three Casoni tests (for hydatid infection) positive among a group of nomadic Pintubi Aborigines at Haast's Bluff, Central Australia, but pointed out that false positive tests may be produced by other helminth infestations.

The Aborigines tested were stated to have been 'drawn from divers places; many had been living on stations under near-European conditions'. Warner *et al.* (1957) found no positive Casoni tests among eight Aborigines from Yuendumu, north of Haast's Bluff.

Aborigines appear to be at high risk of hydatid infection because of their close association with 'camp' dogs, and their rural distribution where

[1] The writer found a prevalence of 3·4 per cent among children in a South Coast community in 1971.

[2] Black (1950: 11) reported *H. nana* from two out of twenty-three faecal specimens examined.

[3] Stevenson (1961: 547) reported an incidence of between 7·4 per cent in 1957 and 3 per cent in 1959 at an Aboriginal settlement (Lake Tyers), with nil incidence in white children from the same area.

dogs are easily infected from offal of livestock and kangaroos slaughtered for food. It is most probable that the infection in kangaroos has followed the introduction of infected sheep or cattle, but under Aboriginal conditions the kangaroo-dog cycle, with man as an occasional accidental host, may continue even where livestock are absent.

Hydatid infection in man is a serious, often disabling and frequently fatal disease requiring skilled diagnosis and surgical treatment. It has a long incubation period. Its diagnosis in Aborigines dying in remote areas is unlikely without a careful autopsy as it mimics internal tumors and abscesses.

The nematode worms commonly causing bowel infestations among Aborigines are hookworms (*Ancylostoma duodenale*, *Necator americanus*), *Strongyloides stercoralis*, roundworms (*Ascaris lumbricoides*), whipworms (*Trichuris trichuris*), and threadworms (*Enterobius vermicularis*). Except for *Strongyloides* these worms do not multiply within the body, and the worm load is therefore proportional to the number of eggs swallowed or, in the case of the hookworms, the number of larval forms penetrating the skin, and indicates the degree of environmental pollution with human faeces and the personal habits of those infested.

With the possible exception of threadworms, all are important contributors to Aboriginal morbidity, both directly and indirectly, and deserve full consideration.

The 'common' hookworms, *Ancylostoma* and *Necator*, have for long posed a public health problem in north Australia, where in the past the infestation was present in all racial groups—Aboriginal, Chinese, white, and Pacific Islander. The Australian Hookworm Campaign of the 1920s largely eradicated it from the white and Chinese population of north Australia, but it has persisted among the Aborigines. Its prevalence has declined among Aborigines with the advent of more effective drugs and improvement in community hygiene, but it is still a problem in many areas.

By far the largest number of 'notifications' of hookworm disease in recent years have come from the Northern Territory, with lesser numbers being reported from Queensland, New South Wales, and Western Australia. It may be safely assumed that the majority of cases reported are Aboriginal. In the Northern Territory, the notification rate is likely to equal the case detection rate, since faecal specimens obtained from surveys and other sources are examined in government laboratories and Aborigines are rarely treated by the relatively few private practitioners. This may account in part for the high Northern Territory notification

rate, but the figures cannot be used to calculate a prevalence rate in the absence of figures for numbers surveyed. The only recent figures for prevalence available to the writer are contained in a statement in the *Annual Report of the Commonwealth Director-General of Health for 1963-4*, which states (p. 38) '... the instance of hookworm ... has formerly approached 100 per cent infestation. In some instances, levels as low as three per cent have been reached'. There were 257 notifications for hookworm in this period, from an unreported number of faecal examinations. Assuming that the disease was confined to the northern division with a population of around 10,000 Aborigines,[4] a *minimum* prevalence rate of about 2·6 per cent in the northern division can be postulated.

The initiation of a full-scale hookworm campaign was implied in the *Report* for 1962-3, the emphasis being on improved sanitation in Aboriginal communities.

In Western Australia, Elphinstone (1960:61) reported the hookworm-endemic areas to be Forrest River Mission, Wyndham Ration Camp, Beagle Bay Mission, and Kalumburu Mission. The report stated that most of the children at Forrest River, and all the children at Kalumburu were passing hookworm ova. With the combination of climate and soil types, hookworm appeared to be a focal disease in the Kimberleys.

Three years later, Elphinstone (1963:90) reported the detection of hookworm among Aborigines at several cattle stations (Bow River, Argyle Downs, and Kimberley Downs) and another mission—Mowanjum—near Derby. He made the observation that if it was ever necessary to prohibit movement of Aborigines out of endemic areas to control the spread of hookworm, 'considerable disorganisation of the cattle industry would inevitably result'—an interesting comment!

Another three years later, Elphinstone (1966:66) reported hookworm among Aborigines recently settled at Kununurra, on the Ord River. Meanwhile the endemic areas were being brought under control with mass drug administration, and a method of killing infective larvae in soil around laundry and drinking taps with a layer of coarse salt was being investigated.

Twenty-four samples of human faeces taken from the ground during a survey of Aborigines in the Central Desert of Western Australia in 1964 yielded one infection with *Hymenolepsis nana* only, the remaining twenty-three specimens being free of protozoal cysts or helminth ova

[4] Hookworm does not appear to be a problem in the southern division, because the infective larvae cannot survive in the soil due to dryness and the extreme diurnal and seasonal temperatures.

(Elphinstone, 1971:299). The common occurrence of eosinophilia[5] in this Desert population—almost half of sixty-one persons tested showing an elevated percentage of eosinophils—was unexplained. Elphinstone has suggested that visceral *larva migrans*[6] due to ingestion of the ova of the dog roundworm (*Toxocara canis*) may have contributed to the eosinophilia.

In Queensland, hookworm was found in only four out of 526 faecal specimens (0·8 per cent) from children at seven settlements and missions in the late 1960s (Welch, 1969:14). The highest figure for recent years was 7 per cent at Edward River (Welch and Frazer, 1970:19). Prolonged efforts to eradicate hookworm from Queensland Aboriginal settlements have not been completely successful, but the prevalence appears to have been reduced to quite low levels, from rates of over 60 per cent around 1960 to less than 1 per cent in 1970.

In New South Wales, hookworm has remained a problem in the North Coast Aboriginal settlements, although no recent figures are available. The New South Wales hookworm endemic area peters out north of Sydney for climatic reasons, and hookworm is not a problem on the South Coast, or inland in western New South Wales.

Mobidity from hookworm disease is related to the worm burden in the individual and not the prevalence in the community, because the presence of a few worms has little or no ill-effect, although hookworm ova will still be detectable in the faeces. However, high worm burdens tend to be uncommon in areas of low prevalence. The worm burden is estimated from an egg count (per gramme of faeces) and without data on egg counts it is virtually impossible to draw conclusions as to the consequences of hookworm infestation. Furthermore, the consequences of a specified worm burden will vary according to the nutritional status of the individual—particularly his iron intake.[7] Anaemia in Aborigines from hookworm-endemic areas used to be referred to as 'hookworm anaemia' but there is an increasing amount of evidence that the anaemia (which is common in Aboriginal communities outside hookworm-endemic areas) cannot be attributed to hookworm as the sole cause. This

[5] Eosinophilia—an increased number of eosinophilic white blood cells commonly associated with various helminth infestations.

[6] The dog roundworm larva wanders through the body tissues and may encapsulate or disintegrate in vital organs of the accidental human host—the eye and the brain being particularly prone to damage in this way.

[7] Hookworms suck blood from the lining of the intestine, and the principal effects of hookworm disease are believed to arise from chronic blood loss, and the accompanying iron depletion.

is not to say that high hookworm loads will not contribute to anaemia or produce other effects by damaging the intestinal lining of affected individuals. This will be discussed further when considering morbidity from anaemia.

Strongyloides stercoralis, a 'toothless hookworm' having a slightly different life cycle and epidemiological pattern, is another common parasite in Aboriginal communities, and is not limited by climate to the same extent as the 'classical' hookworm in Aboriginal populations, *Necator americanus*. Its important differences from ordinary hookworm lie in its ability to multiply within the body of the human host as well as to spread through an infective larval stage in the soil; and in its habit of embedding itself within the intestinal lining where it is protected to a degree from being dislodged by drugs. Its chief effects appear to arise from damage to the lining of the upper intestine and from allergic manifestations (Hunter, Frye, and Swartzwelder, 1967:450). As with ordinary hookworm, morbidity appears to be related to worm load, but the pathological effects of strongyloidiasis suggest that there is a greater likelihood of permanent sequelae to chronic infestation. As with hookworm and roundworm the larval forms in their passage through the lungs may cause a pneumonitis. Infestation is often associated with a high eosinophilia, and may cause a 'creeping eruption'.

The prevalence of this parasite is not well documented for Aborigines in the Northern Territory and Western Australia. In Queensland, surveys in the late 1960s revealed a prevalence of around 7 per cent among children from seven settlements, the highest figure being for Mornington Island, with eleven out of eighty-five specimens positive (about 13 per cent) (Welsh, 1969:14). Its prevalence was 5 per cent among children in a New South Wales South Coast community surveyed in 1966-7 (Hausfield and Moodie, 1967). It was not reported among parasites found by Stevenson (1961) among Aborigines in East Gippsland, Victoria.

The prevalence of *Strongyloides* based on the examination of single faecal specimens is particularly likely to be lower than the 'real' prevalence in the community when compared with most other intestinal worms.[8] As a chronic disease which may lead to invalidism (Hunter, Frye, and Swartzwelder, 1967:451), and because it is distributed beyond the boundaries of the hookworm endemic areas, it may be of more importance in Aboriginal morbidity than is generally suspected.

[8] Because special techniques are necessary to find *Strongyloides* larvae in small numbers in faecal specimens, and their excretion may be intermittent. Confirmation of diagnosis may require a biopsy of the duodenal mucosa.

Roundworm infestation is widely distributed in the Aboriginal population, but appears to be more prevalent in the southern communities. Only five infestations were detected among 482 children from North Queensland settlements in the late 1960s (Welch, 1969:14)—a prevalence of about 1 per cent. No infestations were found at Mitchell River and Edward River Missions in 1970. The prevalence at Palm Island in 1964 was 10 per cent before mass treatment (Sandars, Hutchins, and Tanner, 1965:10). At Cherbourg Aboriginal Settlement in southern Queensland, 19 per cent of all children under 15, and around 30 per cent of children aged 2 to 6 years, were infested in 1965, seven months after discontinuing mass treatment. After four mass treatments, in 1962, the under-15 prevalence was 12 per cent, and in 1961, before mass treatment was commenced, the prevalence was 33 per cent (Sandars, Tanner, and Kay, 1966:12).

In New South Wales, the Reports of the Director General of Public Health show a roundworm prevalence of 46·5 per cent among Aboriginal children from North Coast reserves and settlements in 1954, the highest figure being 64 per cent at Burnt Bridge Aboriginal Reserve, near Kempsey. Mass treatment programs commenced at this time had lowered the prevalence to around 16 per cent by 1959. Figures for later surveys are not available.

In a New South Wales South Coast Aboriginal community, Hausfeld and Moodie (1967) reported the prevalence among children as 33 per cent in 1966-7, and a further survey in the same community in 1971 revealed a prevalence of 37 per cent, after treatment on a voluntary household basis (Moodie, unpublished).

In Victoria, Stevenson (1961) reported a prevalence of 55 per cent (settlement) and 64 per cent (non-settlement) in East Gippsland Aboriginal children before treatment, the first figure quoted falling to 14 per cent after repeated mass treatments.

To the writer's knowledge, ascariasis is rare in the Northern Territory and no figures have been sighted for its recent prevalence in Western Australia and South Australia.

Here, then, is a parasite which appears to be of greater importance in temperate south-eastern Australia, although it overlaps to some extent with hookworm in warmer climates. It would be anticipated as a problem among Aborigines in south-eastern South Australia and south-western Western Australia, on climatic grounds, because optimal development of viable eggs occurs at 25°C (Hunter, Frye, and Swartzwelder, 1967:431).

Its importance as a cause of Aboriginal morbidity arises from two

separate consequences of infestation—one from the adult worms and the other from the larval stage. The adult worms, exerting their main effects through interference with the digestion and absorption of protein, will have greater impact upon malnourished children—proportionate to the worm load. Bunched worms may also cause intestinal obstruction. It has been shown, however, that a single adult worm may have serious consequences when it migrates to an abnormal site, so that worm load is not the only determinant. The larval stage, which burrows through the intestinal wall into the bloodstream after escaping from the swallowed egg, migrates via the lungs and air passages before passing into the intestine for the second time and growing to adulthood. In their passage through the lungs the larvae give rise to inflammation—ascaris pneumonitis—the extent of which appears to be determined by the number of larvae migrating concurrently, and hence to the number of eggs swallowed at the same time, or within a short period. This in turn will be influenced by the density of *Ascaris* eggs in that part of the environment from which the infestation is contracted. The soil is usually contaminated by a child's faeces—either directly or through soiled clothing or nappies being rinsed under a tap or in a dish of water. The eggs become infective after an interval of three weeks or so, depending on temperature, and may remain infective for prolonged periods under suitable conditions.

The severity of ascaris pneumonitis, and its frequency in a community, are obviously related to the chances of acquiring a large 'dose' of eggs, and among the factors which determine this chance will be the worm load in those who cause the environmental contamination.

Such contamination is not entirely a matter of lack of 'proper hygiene'. At a well-run (and worm-free) mission home for Aboriginal children, the writer once observed by chance a small child—a new arrival at the home—involuntarily soil the place where he was standing in the playground because of an uncontrollable attack of diarrhoea. An event such as this can produce, unobserved, a localised area heavily contaminated with *Ascaris* eggs and other intestinal pathogens, despite reasonable precautions by responsible adults. Months later, a piece of food or a toy dropped on the ground may easily transfer a large number of worm eggs to a child's mouth.

Where there are small children, intercurrent or recurrent diarrhoea among those infested with intestinal worms is likely to increase the degree of environmental contamination with worm eggs and protozoal cysts by the mechanism referred to above. Lack of toilet training, poor facilities for washing soiled clothing, as well as a lack of proper toilet

facilities will each add their contribution. The prolonged survival of many intestinal pathogens in soil is another factor. It is not surprising that mass treatment programs, even where the drugs are administered under supervision, have had limited success in Aboriginal communities in achieving eradication of roundworm, although the direct and indirect benefits of reducing worm loads should not be ignored when considering the effects of mass treatment on morbidity.

Whipworm is another intestinal nematode commonly infesting Aboriginal communities, and infestation rates may be exceptionally high. Welch (1969) reported a prevalence of 49 per cent among children at seven Queensland Aboriginal settlements, the highest rate being at Yarrabah—91 per cent. Other rates published have been those for the South Coast of New South Wales of 78 per cent, reaching 97 per cent among children at one Aboriginal settlement in 1966-7 (Hausfeld and Moodie, 1967); and for Lake Tyers Settlement in East Gippsland a rate of 93 per cent in 1959 (Stevenson, 1961).

At Mitchell River and Edward River Missions in North Queensland, where there were no *Ascaris* infestations found in 1970, the whipworm prevalence was 28 and 36 per cent respectively in children under 15 (Welch and Frazer, 1970:19).

Light infestations with whipworm are believed not to have significant morbid effects, but heavy infestations in young children have been held responsible for diarrhoea and related abdominal symptoms, severe anaemia, weight loss and weakness. Unlike *Ascaris*, the whipworm larvae do not migrate through the lungs but develop from the larval to the adult stage in the intestine after hatching out of the swallowed egg. The adults interlace themselves through the lining of the lower intestine, causing a degree of oozing from the damaged tissues. It has been suggested, with some experimental evidence, that whipworm predisposes to concurrent invasion by *Entamoeba histolytica*, and that where both are present in an individual whipworm is no less a cause of morbidity (Jung and Beaver, 1951:556). The association of whipworm infestation with anaemia is of particular interest in the context of Aboriginal child health.

As with hookworm, whipworm prevalence rates are not necessarily related to whipworm morbidity rates, and estimates of worm loads based on egg counts are rarely reported. The estimation of worm burdens from egg counts may be subject to considerable error, particularly in that there is little general agreement as to how many eggs a female whipworm produces per day—figures quoted range from 300 to 7,000.

Whipworm eggs require several weeks to become infective, and the

embryo may not become fully mature for perhaps six months: thereafter, in a moist, shaded environment, it may remain infective for at least five years—an indication of the difficulty of eradicating this infestation once it becomes established in an Aboriginal community.

The part played by individual helminth and protozoal infestations in Aboriginal morbidity patterns is hard to assess: in the social and environmental situation where these parasites flourish, infection of individuals with multiple species is a very common occurrence.

The prevalence figures, while not translatable into morbidity figures, are excellent indicators of the risks of acquiring almost any sort of intestinal infection and the extent of environmental pollution with human faeces.

Aboriginal children almost invariably have higher rates of infection or infestation than adults, and in childhood the highest rates appear to coincide with the ages at which children are most likely to spend much of their time playing in the dirt and around drains and taps. Young children in the same age-group are most likely to contaminate their environment with faeces. Thus the cycle of infection may be maintained in the small children in a community—adults and older children tending to be 'accidental' hosts but not sources. While adequate, safe toilet and laundry facilities are necessary to protect the environment from contamination, the eradication of intestinal infections from Aboriginal communities cannot be achieved with drugs and the provision of physical facilities alone; changes in the behaviour of children, and changes in the attitudes of adults to the behaviour of their children, are also necessary. Even with changes in behaviour, the long-term persistence of infectious agents such as the helminth eggs in a suitable environment will not allow quick results in an eradication program—an unfortunate but largely unavoidable source of discouragement to the Aboriginal communities and to those working to improve their health. In the face of many handicaps, the extent to which Queensland has succeeded in reducing worm infestations—particularly hookworm—is to be commended, although this State would appear to be committed to maintaining its anti-worm measures for many years more if prevalence levels are to be kept low. The figures for Queensland, as with the figures for other States, are derived from studies in supervised communities, and the intestinal infection rates in the 'fringe' communities and among those moving to the cities are generally unknown: they are likely to parallel those of the supervised communities prior to the introduction of mass treatment and preventive measures.

There can be little doubt that the intestinal infections, particularly worm infestations, are an important source of what might be called 'social morbidity'. So long as 'everybody knows' that such infestations are common in Aboriginal communities, prejudices from whites against Aborigines are likely to be reinforced—in schools, at community facilities such as swimming pools, and elsewhere, although the arguments advanced are often spurious. The effects upon general social acceptance of Aborigines caused by various infections—many of which are medically trivial—will be taken up later. They are referred to here because the writer has often heard and read of prejudicial views in connection with worm infestations, these being diseases which may cause excessive reactions of revulsion in otherwise tolerant people (perhaps because worms such as roundworms are large and often visibly active when expelled).

In addition to the direct consequences of specific intestinal infections, there is an unmeasurable synergism with malnutrition, and some evidence for synergism and increased susceptibility between the intestinal infections themselves and between intestinal and other infections—a cumulative total morbidity which cannot easily be dissected into its component parts in a human population. It is likely, for instance, that parasitic infestations affect the psychological performance of schoolchildren adversely. Dawson (1963) investigated the psychological performance of iron mine workers in Sierra Leone treated for intestinal parasites and malaria, with a control group, and found a significant improvement in performance of several tests in the treated group, although he was unable to conclusively rule out a placebo effect in his treated group. There is a need for a careful assessment of the impact of parasitic infestations on Aboriginal performance at school and in employment—particularly with respect to parasites such as *Giardia*, *Strongyloides*, and whipworm which are often assumed to be of minor importance.

TUBERCULOSIS

Tuberculosis has been considered an important disease in any community, and the control of tuberculosis one of the major activities and responsibilities of a health department. Both the general standard of living of a population and the success of specific control measures are reflected in tuberculosis incidence, prevalence, notification rates, mortality rates and hospitalisation rates. The disease is important to affected individuals even when it is asymptomatic because treatment often requires prolonged hospitalisation and isolation, prolonged follow-up care, and the more

advanced cases may suffer permanent physical incapacity and occasionally death. In countries such as Australia there is very often a social stigma attached to the sufferer or ex-sufferer, and employment potential may be permanently reduced for this reason although a 'permanent' cure has been achieved.

The near-eradication of tuberculosis from the white Australian population has been achieved partly through the anti-tuberculosis programs of health departments and partly as a by-product of improved living standards, and advances in treatment have reduced the period of hospitalisation and isolation to an average of a few months for cases detected early in the course of the disease. The economic handicaps resulting from diagnosis as much as from morbidity have been reduced by social welfare assistance—it being noteworthy that for decades this was the only communicable disease for which a special sickness pension was available.

Until recently, one of the most useful methods of measuring the incidence and prevalence of tuberculosis in a community was the 'natural' reactor rate to the tuberculin skin-test (the Mantoux test and its variations). This test indicated whether an individual had acquired a tuberculous infection in the past, not whether he had an active infection—the majority of acquired infections terminating spontaneously—but the reactor rate was an index of exposure to infective individuals in the community surveyed.

For Aborigines, this test has lost much of its value in recent years for two reasons: firstly, the natural reactor rates have been obscured by B.C.G. vaccination in the Aboriginal population;[9] and secondly because a number of organisms related to *Mycobacterium tuberculosis*, the so-called 'atypical mycobacteria', which appear to be commonly responsible for minor infections in Aboriginal communities in the north, are now known to cause cross-reactions with the tuberculin test. Tuberculin test surveys carried out before the introduction of B.C.G. vaccine are mentioned below, the results being comparable one with another and indicating exposure to infective cases.

In 1950, King *et al.* (1951) carried out a survey for pulmonary tuberculosis among Aborigines of the Kimberley and Port Hedland districts of Western Australia, X-raying tuberculin reactors and some others. They found fifteen cases of pulmonary tuberculosis among 3,209 Aborigines surveyed, only two cases being classified as 'active'. The authors commented that the prevalence, at less than five cases per 1,000 was lower than in the white population at that time. The Mantoux test results

[9] B.C.G. vaccination increases immunity to tuberculosis infection but also causes a positive reaction to the tuberculin test.

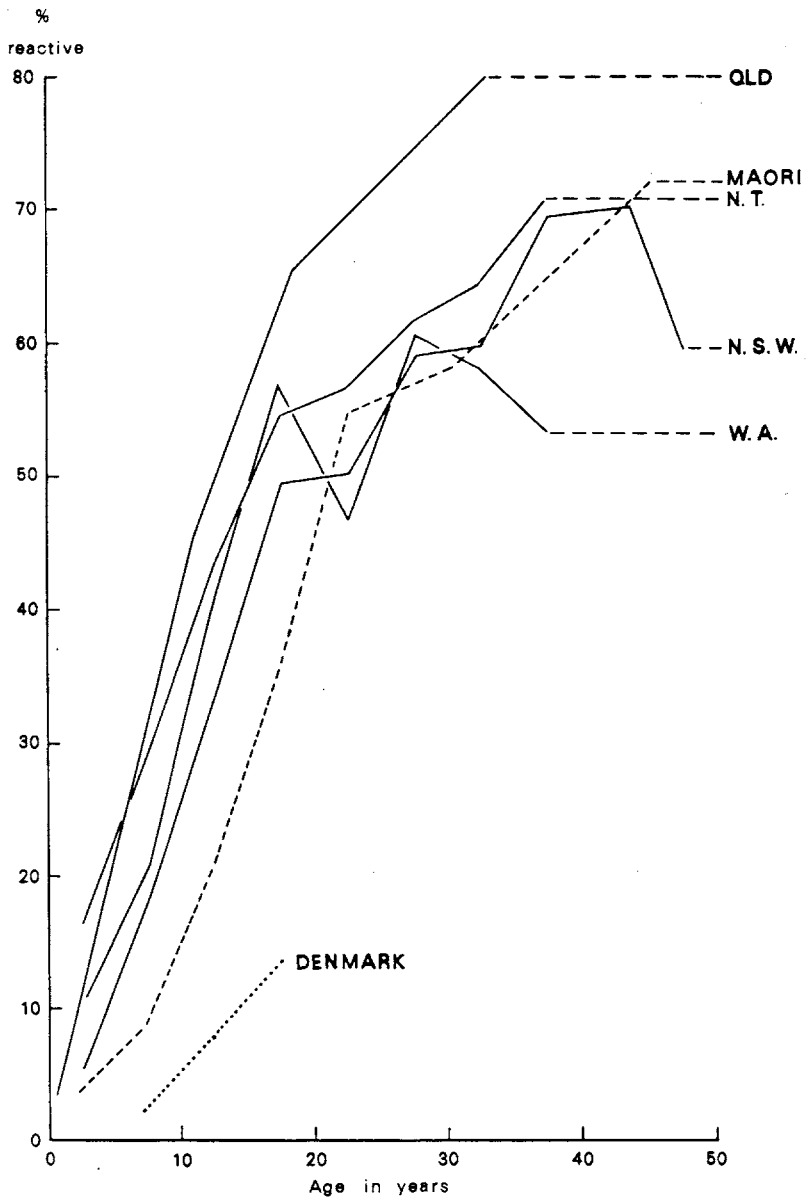

Fig. 2. Tuberculin reactor rates by age-groups, both sexes. Denmark is included because it represents optimum figures. Figures for later ages are not available

showed a significantly lower proportion of natural reactors at all age-groups except in those under 5 years when compared with the proportion of reactors in the white population of the Perth metropolitan area. As expected, Aboriginal reactor rates were higher in 'town' communities (Derby, Wyndham) than in 'desert' communities (Jigalong, Pallotine), though still lower than rates in metropolitan whites. Non-reactors under age 45 were given B.C.G. vaccine. The tuberculin reactor rates are shown in Fig. 2.

In Queensland, Macken (1952) carried out a similar type of survey among 6,600 Aborigines on settlements and missions in the east and north, including the Torres Strait Islands. Excluding the latter, the survey revealed an active case rate of 21 per 1,000, and a reactor rate somewhat higher at each age-group than in Western Australia (see Fig. 2). The author commented on the frequency of active infection in Aboriginal nurses and midwives, and in the Aboriginal household servants of white Australians who had come to Queensland 'for their health', and stated his personal impression that there was an increased racial susceptibility to tuberculosis among full-blood Aborigines. B.C.G. vaccination was commenced in 1950-1.

In the Northern Territory, Watsford *et al.* (1956) reported a tuberculin survey among 5,259 full-blood Aborigines, a little over a third of the estimated total numbers in the Territory at the time (1951-5). Aborigines included came from communities visited and from those attending hospital: the relative proportions coming from these sources are not stated. The tuberculin reactor rates by age-group were intermediate between those determined for Western Australia and Queensland, although nearer to those for Western Australia (see Fig. 2).

Age-specific tuberculin rates were higher in long-established settlements near white centres of population than in recently established settlements in remote areas, and conversion to tuberculin-positive reactions occurred at earlier ages, pointing to tuberculosis having been introduced with white settlement, and encouraged by environmental conditions in the settled Aboriginal communities. The reactor rates (all ages) for Oenpelli and Hermannsburg Missions, both settled by the 1890s, were 59 per cent and 71 per cent respectively, while for Yuendumu and Yirrkala, both settled since 1939, the rates were 31 per cent and 17 per cent respectively.

Among Darwin schoolchildren, the authors noted markedly higher tuberculin rates in the part-Aboriginal children when compared with those for white children, as follows (Watsford *et al.*, 1956: Table VI):

Age-group	Tuberculin reactor rate per cent White children	Part-Aboriginal children
5-9	5·5	24·8
10-14	10·2	43·9
15-19	25·0	54·5

indicating higher exposure to infective individuals in the part-Aboriginal environment.

In New South Wales, Andrew (1958, 1959) reported briefly on the results of tuberculin surveys of Aboriginal communities, reactor rates varying between 39 per cent[10] and 27 per cent for different areas of the State. No details were given, and I am indebted to the New South Wales Department of Health for access to Dr Andrew's data, from which age-specific reactor rates have been calculated.

Table 35: *Age-specific Mantoux reactor rates, by sex, for 45 Aboriginal communities, New South Wales, 1958-63*

Age-group	Males Positive/total	%	Females Positive/total	%	Both sexes Positive/total	%
0-4	35/674	5·2	31/599	5·2	66/1,273	5·2
5-9	125/616	20·3	100/582	17·2	225/1,198	18·8
10-14	162/430	37·7	141/465	30·3	303/895	33·9
15-19	70/128	54·7	103/222	46·4	173/350	49·4
20-24	43/78	55·1	93/193	48·2	136/271	50·2
25-29	50/73	68·5	90/163	55·2	140/236	59·3
30-35	37/56	66·1	85/148	57·4	122/204	59·8
36-39	35/41	85·4	54/87	62·1	89/128	69·5
40-44	26/31	83·9	57/86	66·3	83/117	70·9
45-49	26/33	78·8	33/66	50·0	59/99	59·6
50 and over	81/109	74·3	90/124	72·6	171/233	73·4
Total	690/2,269	30·4	877/2,735	32·1	1,567/5,004	31·3
Ages unknown	11/65	16·9	23/57	40·4	34/122	27·9
Total	701/2,334	30·0	900/2,792	32·2	1,601/5,126	31·2

Note: B.C.G. conversions, where identified, have been counted as tuberculin negative.
Source: Unpublished data, Tuberculosis Division, New South Wales Department of Health.

The results calculated from Andrew's data are given in detail in Table 35, as they are not accessible in the medical literature. In the survey, 5,126 individuals were tested and read (Mantoux test—5 T.U. of Old Tuberculin given; reaction of 5 mm diameter or more recorded

[10] A figure of 39 per cent is derived from the numbers tested and reactive in Andrew's report for 1958, but a figure of 41 per cent for this survey is quoted in the next year's report.

as positive). Where surveys had been repeated, the earliest survey which appeared to give the most complete age-sex coverage was selected. This selection resulted in the inclusion of communities which had been surveyed before and the negative reactors given B.C.G. vaccine, and in many such communities it was evident from the records that difficulty was experienced in re-identifying all the B.C.G.-vaccinated individuals—usually children under 5. In estimating 'natural' reactor rates from these data, known B.C.G.-conversions have been classified as tuberculin-negative—i.e. as if they would have remained tuberculin negative if B.C.G. vaccine had not been given. It has been assumed that the inclusion of some unidentified B.C.G. conversions as natural reactors would be counterbalanced to about the same extent by counting all identified B.C.G. conversions as tuberculin-negative—that is, denying any possibility that some individuals recorded previously as tuberculin-negative would have been naturally converted to tuberculin-positive status between surveys. This manipulation cannot be defended statistically, but is unlikely to inflate the 'real' natural reactor rate above its true value. The results calculated in this way show a straight-line rise in reactor rates with age, which would be expected in the absence of a large number of unidentified B.C.G. conversions.

The New South Wales Aboriginal reactor rates by age-group generally parallel those for the three full-blood groups (Northern Territory, Western Australia, and Queensland) but are a little lower at all age groups. The results are plotted in Fig. 2.

The methods used in tuberculin-testing Aborigines in the Northern Territory, Queensland and New South Wales were the same—Old Tuberculin as the skin-test agent, and reactions of 5 mm or more recorded as positive. The Western Australian report (King *et al.*, 1951) does not mention the agent or the criterion of positivity, but it may be assumed that the technique was the same as in the other States, and that all four survey results may be compared. Most tuberculin reactor rates for overseas populations are based on reactions to another agent (usually the purified protein derivative of tuberculin—P.P.D.) and different criteria of positivity (usually 10 mm diameter of reactions) and cannot be compared in detail with Australian rates. The steepness of the age-group reactor rate profile curves, however, may be compared with those for the Aboriginal populations, and when this is done it is seen that the Aboriginal profiles are rather 'average' on world standards, being neither as steep as those for countries where tuberculosis has been a major health problem (Somaliland, Philippines) or as gradual as in countries where

tuberculosis has been well controlled (Denmark, England). The New South Wales Aboriginal tuberculin-reactor profile was very similar to that for Uganda (Nyboe, 1960) and South India (Frimodt-Møller, 1960).

There are no published data for the incidence or new active case rates for New South Wales Aborigines. K.W.H. Harris (personal communication, 1966) has estimated that the new active case rate among New South Wales Aborigines in the early 1960s was around 1 per 1,000. This estimate may be compared with the equivalent rate of a little under 0·4 per 1,000 for New South Wales whites in the early 1960s;[13] of 2·5 per 1,000 for the Maoris (1960);[11] and 3·4 per 1,000 for American Indians (1959).[12]

The lower incidence among New South Wales Aborigines when compared with Maori and American Indians may be more apparent than real because of the probable under-identification of Aborigines in notifications of tuberculosis. There is no obvious epidemiological explanation to account for the difference. The Aboriginal domestic environment in rural New South Wales is likely to be as poor and as overcrowded as that of Maoris and American Indians, although the New South Wales communities, on the average, are probably smaller and the degree of close personal contact between households may be less because of different social behaviour patterns. However, the Maori tuberculin reactor rates by age-group (comparing rates obtained by different techniques) appear to be a little *lower* than those for New South Wales Aborigines, and the same can be said for several American Indian communities for which the writer has seen figures.

Early records suggest that pulmonary tuberculosis was a major health problem in many Aboriginal communities. From 1864 to 1899, 13 per cent of all deaths at Point McLeay Aboriginal settlement in South Australia were attributed to pulmonary tuberculosis, and from 1880 to 1899, 28 per cent of all deaths at Point Pearce settlement in the same State were attributed to the same cause.[13] Tuberculosis receives frequent mention in reports on Aboriginal settlements in other States in the past, although no incidence rates can be obtained from these reports.

[11] Calculated from data published in the World Health Organisation, *Epidemiological and Vital Statistics Report*, 1962, Vol. 15, No. 5, and the United Nations *Demographic Yearbook*, 1961.

[12] United States Department of Health, Education and Welfare, Public Health Service, *Indian Health Highlights*, 1964 edition.

[13] From data collected by Mrs Elaine Treagus, supported by the Aborigines Project of the Social Sciences Research Council and assisted by Dr F. Gale. I am indebted to Professor C.D. Rowley for permission to quote from these data.

Today it appears that pulmonary tuberculosis is no longer a serious health problem among Aborigines, although the incidence is generally somewhat higher than in the white population. Case-finding surveys, B.C.G. vaccination, and effective treatment have probably contributed more to the decline in pulmonary tuberculosis among Aborigines than they have among whites, as the latter have also benefited from a marked rise in the general standard of living. There may be a danger that pulmonary tuberculosis will persist, and possibly increase, in the larger rural and metropolitan Aboriginal communities if anti-tuberculosis measures, no longer of such importance to the white community, are abandoned or scaled down in Aboriginal populations. It should not be difficult to continue anti-tuberculosis measures—based on mass X-ray, skin testing, and B.C.G. vaccination—in the supervised settlements and missions in north Australia. In the rural-urban fringes, on reserves and in metropolitan part-Aboriginal communities in southern Australia, however, the problem of continuing tuberculosis control is not so easily met. Special measures which single out the part-Aboriginal members of mixed communities will be recognised by part-Aborigines as 'discriminatory', and if resisted on these grounds are unlikely to be very successful. Preventive measures offered to the general community are unlikely to be used by those part-Aboriginal households who need them most: these are the households which also frequently fail to attend for free immunisations against polio, diphtheria, whooping cough, or make full use of free school medical services. Skilled general community health field programs which are accepted by part-Aborigines without being regarded by them as discriminatory probably offer the best prospect of a continuing (and improved) control of pulmonary tuberculosis, other communicable diseases, and—in fact—all health hazards of particular importance in the Aboriginal population.

Tuberculosis affecting other organs—bones, joints, intestine, genito-urinary system and central nervous system—has been reported in Aboriginal populations but no incidence or prevalence figures are available. Extra-pulmonary tuberculosis is commonly a result of infection with bovine tuberculosis, as well as the result of dissemination from an infection in the lungs with human tuberculosis. The primary source of bovine tuberculosis is milk from tuberculous cattle, and as Aborigines generally obtain milk from the same sources as the rest of the Australian community—that is, from TB-tested herds—bovine tuberculous infection is unlikely to be a threat to the Aboriginal population at present.

As stated by Seabury (1967:183), 'the epidemiology of tuberculosis is

extremely complex, and susceptibility to infection, pathogenesis and clinical manifestations are not uniform in all racial groups'. In Seabury's view, 'the differences may reflect factors of immunity arising from the duration of tuberculosis within the population group' and 'malnutrition ... is of major importance in the infection, morbidity and mortality'. There is as yet no convincing evidence that tuberculosis in Aborigines is influenced by genetically-determined factors peculiar to Aborigines. As with the majority of communicable diseases, a genetically-determined component of susceptibility is difficult to isolate because genetic constitution, behaviour, environment, nutritional status, and personal contact are all mediated through kinship in one way or another. Hypotheses that attempt to prove genetically-determined racial susceptibility to tuberculosis, but do not take into account the factors mentioned above (and the added effect for Aborigines of intercurrent non-tuberculous infections or malnutrition in converting a quiescent 'healed' primary infection into an active infection) may be safely disregarded.

Because of environmental circumstances, it is likely that the incidence of non-tuberculous mycobacterial infections is higher in Aborigines than in the general population. This is certainly true with respect to leprosy—considered in the next section—but very little information is available on the prevalence or incidence of the 'anonymous' mycobacteria—bacteria usually identified in routine sputum cultures in tuberculosis laboratories but distinguished from *M. tuberculosis* by their behaviour in the culture. The pathogenicity and epidemiology of these 'infections' remains somewhat obscure; some have been identified as either causing or being associated with pulmonary disease, and many in the group are believed to be identical with organisms recovered from soil, water, and domestic animals (Abrahams, 1965). Bolliger and Kiewiet (1969:1205) found more than three times as many 'anonymous' as tuberculous infections among 'chest investigation' sputum samples in the Townsville area in 1967-8, and comment that the 'incidence [i.e. prevalence] of acid-fast organisms in sputum among Aborigines ... living in the laboratory's area is much the same as among the population as a whole. However the proportions of anonymous mycobacteria and *M. tuberculosis* were equal ...' rather than three to one as in the general population of the area investigated. It is probable that the higher proportion of anonymous infections in the white population merely reflects a lower prevalence of tuberculous infection, but the capacity of at least some of the anonymous mycobacterial infections to produce positive tuberculin reactions casts some doubts on the tuberculin test as a

valid index of the occurrence of tuberculosis infection in Aboriginal communities. Abrahams (1965:788) has also pointed out that anonymous mycobacteria, like B.C.G. vaccination, appear to confer some immunity to tuberculous infection, and may explain the rarity of juvenile and osseous tuberculosis in Queensland, since it has been shown that the prevalence of anonymous mycobacterial infections is much higher in Queensland than in the southern States.

Skin ulceration due to *Mycobacterium ulcerans* has been reported by Quinn and Crotty (1963:317) in three part-Aborigines from the Croker Island area of the Northern Territory.

In general, it would appear that mycobacterial infections other than tuberculosis and leprosy are not 'communicable diseases' and few of them are primarily responsible for disease and disability. Both absolutely and relatively, they do not appear to be particularly important in Aboriginal health, although further knowledge about their epidemiology and pathogenicity may alter this view. The possibility that they confer some cross-immunity to tuberculosis and leprosy should be borne in mind.

LEPROSY

Leprosy, remarkable for the apparent low infectivity of cases (or high resistance to the development of 'disease' in those infected, which amounts to the same thing), a relatively low degree of morbidity among total cases and a negligible mortality, is also remarkable for the intense feelings of horror and revulsion it has generated in Western society—perhaps in all societies which have been influenced by Christianity, Judaism, and Islam. Partly arising from a confusion of 'real' leprosy with more mutilating diseases such as syphilis and yaws in past centuries, this emotional reaction has nevertheless given leprosy more prominence as a 'health problem' of Aborigines than it possibly deserves. This has placed leprosy in the almost unique position wherein the *diagnosis* of leprosy is often a major personal and social catastrophe, while the disease itself may cause no physical handicap.

As a result, Aborigines in widely separated areas of north Australia have referred to two primary classes of illness when speaking to white people—'big sick' (leprosy) and 'little sick' (all other illness): a classification quite unrelated to morbidity or mortality in the ordinary sense.[14] For Aborigines, however, the social consequences and the occasionally

[14] See Hargrave (1968) for a brief but very well informed account of Aboriginal attitudes to leprosy.

severe physical handicaps are an inescapable fact, and the attempt to control and eradicate leprosy in the Aboriginal population a very worthwhile pursuit. The liberal approach to control, employing domiciliary treatment where possible, pioneered by Hargrave in the Northern Territory, has done much to reduce the social consequences of diagnosis in the context of Aboriginal society, but leprosy is likely to remain a major contributor to prejudices against Aborigines, and its presence in the Aboriginal population an increasing psychological handicap in their relations with white Australian communities, until it has been eradicated.

There is little doubt that leprosy was introduced into the Aboriginal population by contact with infective Chinese and Pacific Island labour during the nineteenth century (Cook, 1927). Leprosy, introduced near Darwin and Pine Creek in the Northern Territory, and into ports and mining areas in the Kimberleys and north Queensland, spread slowly from tribe to tribe particularly in the northern half of Northern Territory, where it is now endemic, with concentrated foci in Arnhem Land, and in the north-west of the Territory between the Daly and Fitzmaurice rivers (Hargrave, 1968:7). Hargrave reported it to be endemic also in the Kimberleys, but less so than in the Northern Territory, and to have almost disappeared in an active form from Queensland. No cases have been reported in the southern States in recent years, but before World War II cases were reported among New South Wales coastal part-Aborigines who had never been outside this area (Millard, 1913, 1928).

In the Kimberleys, Elphinstone (1966:64) reported that among 7,000 Aborigines, about 600 had, or had had, leprosy—a prevalence for past and present infection of around 8·6 per cent. His figures for admissions to the Derby leprosarium indicate an average 'new active case' rate of about 1·4 per 1,000 from 1964 to 1966. The disease was confined to about fifty families. B.C.G. vaccine, offered throughout the Kimberleys in 1966 as an anti-tuberculosis measure, was also looked upon with hope as a possible means of increasing immunity to leprosy.

In the Northern Territory, the new active case rate averaged for the ten years 1957-66 was 2·8 per 1,000 Aborigines per annum,[15] it being assumed in the calculation that all new active cases were Aborigines. In 1966, living persons registered as leprosy patients amounted to 4 per cent of the Aboriginal population. Omitting the population of Central

[15] Based on 504 new cases reported in the National Health and Medical Research Committee Report of the 65th Session (1967), p. 132, and an average Aboriginal population of 17,906 calculated from population figures given in successive Annual Reports of the Northern Territory.

Australia, where leprosy is not endemic, these figures would be about 50 per cent higher for the northern half.

The Report of the 65th Session of the National Health and Medical Research Committee (1967:130) states, 'In Western Australia and the Northern Territory the disease is most commonly found in Aborigines, whereas in Queensland it is more generally spread between people of all racial groups'. It is not possible to make an estimate of the new active cases rate for Queensland Aborigines from the available data other than to state that the rate appears to be considerably less than 0·1 per 1,000 Aborigines. It is difficult to account for such a marked difference in estimated rates from those for the Northery Territory and Western Australia—a situation which is not paralleled by that for tuberculosis. Cook's study (1927) indicates that leprosy was well established in certain areas of north Queensland, although endemicity seems to have declined naturally in large areas of Cape York Peninsula by 1925.

It is an interesting but now untestable hypothesis that the endemicity of leprosy in Queensland may have been influenced by cross-immunity from tuberculosis and 'atypical' mycobacterial infections, known to be particularly prevalent in that State. The present distribution of leprosy in Queensland through 'all racial groups' appears, from the figures, to result from its very low incidence in Aborigines rather than from a higher incidence in other racial groups. C.E. Cook (personal communication, 1971) is of the opinion that the early rigid segregation of Queensland Aborigines on supervised missions and settlements, with very little social intercourse between or outside these communities, contributed to the decline of leprosy to its present low level. In contrast, Aboriginal lepers isolated on Channel Island in Darwin Harbour, Northern Territory, were sent back to their 'tribes' and to distant missions and settlements when Darwin was attacked by air during World War II, an event which probably increased the prevalence of leprosy in the north of the Northern Territory just before the increase in spatial mobility of Aborigines which followed the war.

There are many other factors which may account for differences in the incidence of leprosy in the three northern States' Aboriginal populations, including differences in case-finding methods and coverage.

The epidemiology of leprosy is no less complex than that of tuberculosis, and is less understood because the agent, *Mycobacterium leprae*, cannot be cultured in artificial media and cannot be tested for by the equivalent of the tuberculin reaction. In speaking of morbidity due to leprosy, one is speaking of morbidity due to diagnosable leprosy, and hence morbidity

due to an unknown fraction of total infections in a community—assuming that many infections never progress to detectable disease. Where diagnosis depends on the detection of a pathological change, the consequences of infection with leprosy are likely to appear exaggerated. Hargrave (1963) analysed 300 cases among Northern Territory Aborigines and concluded that 239 cases had disability as a result of the infection—nearly 80 per cent. Figures published by the National Health and Medical Research Council (1967:133) show that at about the time of Hargrave's study (which is not stated in his report) there were nearly 700 living persons registered as leprosy patients in the Northern Territory, including some part-Aborigines and others, but it is not known whether the balance of Aboriginal cases were free of disability or had disabilities to the same extent as the 300 cases studied. Estimating the Aboriginal population of the Northern Territory at the time to be something like 18,000, it may be estimated that about 2 per cent, and not less than 1·3 per cent of all Northern Territory Aborigines had disabilities of varying degrees of severity due to leprosy in 1963.

On a world basis, it has been estimated that nearly 40 per cent of sufferers have some disability as a result of their infection (Bechelli, 1966) and this would suggest a figure of 1·3 per cent of Northern Territory Aborigines, and about 3·4 per cent of Kimberley Aborigines were disabled. Even higher figures would be anticipated in the concentrated endemic foci in Arnhem Land and the north-west of the Northern Territory. To this figure must be added the proportion who are not physically handicapped, but on the other hand are most likely to be suffering from the infectious form of the disease and are likely to be removed from their families and social group for a period until their infectious state is controlled. Total morbidity as incidence in this sense (physical and social handicaps combined) will be close to the new active case rate annually, and the prevalence of past or present infection will exceed the prevalence of disability only to the extent that domiciliary treatment, the arrest of active disease, and the correction of deformities is possible.

DIPHTHERIA AND WHOOPING COUGH

There are no published references to outbreaks of these diseases of childhood in recent years. The *Northern Territory Report* for 1953-5 (p. 41) refers to a small epidemic of diphtheria at Banka Banka Station in February 1955. Both these diseases appear to be rare in full-blood com-

munities, due both to immunisation of Aboriginal children and the low incidence in the general population. As a result of inadequate immunisation, cases are most likely amongst the part-Aborigines in southern Australia, but such cases when notified would not be identifiable as being Aboriginal. Small size of Aboriginal communities and their relative isolation from one another tend to discourage prolonged endemicity of such diseases, but small epidemics are likely should the immunisation status of individual communities fall to a low level.

STREPTOCOCCAL SORE THROAT AND SCARLET FEVER

These have not been reported as endemic or epidemic in the Aboriginal population, although Jose and Welch (1969) have shown a high prevalence of isolations of Group A streptococci among children from Yarrabah, Arakun, and Doomadgee in North Queensland when compared with white schoolchildren in Brisbane. This organism is not only responsible for sore throat and, in some strains, scarlet fever; it is also responsible for the serious sequelae of rheumatic fever and glomerulonephritis. Thirty per cent of sixty Aboriginal children had Group A streptococci in their throats compared with 3·3 per cent of sixty Brisbane white children. Jose and Welch further showed that 58 per cent of the Aboriginal group had high levels of antibody to this infection (antistreptolysin—O) indicating recent or repeated streptococcal infection; this is compared with 8 per cent of Brisbane white children. Streptococcal infection is discussed further when considering rheumatic fever and rheumatic heart disease. Streptococcal sore throat is also an important cause of middle ear infection, which may lead to perforated ear drums, and less frequently to mastoiditis and meningitis. Streptococci and staphylococci are frequently associated in cases of impetigo.

The high carrier rates and antibody levels for streptococcal infections in North Queensland Aboriginal children are likely to be repeated in all Aboriginal communities where children are plentiful in the community and live in overcrowded dwellings. Scarlet fever is easily missed in dark-skinned children, and may not be suspected until peeling of the skin occurs after the infection has run its course. This may explain its absence from reports on illness among northern full-blood Aborigines, although another explanation is that first infection with scarlet fever-producing strains (subsequent infections do not cause a rash) may occur early in infancy when the rash may not appear.

POLIOMYELITIS

As with diphtheria and whooping cough, poliomyelitis does not figure in recent reports of illnesses in Aboriginal communities, and may be presumed to have 'disappeared' as a result of immunisation of the Aboriginal and white populations. Miles (1953) and Stokes *et al.* (1955) showed that before 1955 type II poliomyelitis virus was endemic in the Northern Territory Aboriginal population tested. Over 90 per cent showed serological evidence of past infection with type II polio virus by the age of 5 years in 1952. Antibodies to poliomyelitis virus types I and III showed a more patchy distribution, with evidence that epidemics in localised communities were infrequent, but that no communities had escaped infection during the lives of those tested. Stokes *et al.* commented on the apparent rarity of paralytic poliomyelitis in the Aboriginal population, but could offer no evidence that there was inborn racial resistance to paralytic sequelae from poliomyelitis infection.

SMALLPOX

Smallpox is currently absent from Australia, but earlier epidemics, particularly the first epidemic in 1789 in the year following settlement at Port Jackson, are believed to have resulted in high mortality in the Aboriginal population. Although smallpox is no longer a problem in this country, the first outbreak is of particular interest to a study of Aboriginal health for two reasons: firstly, it must have provided enormous initial impetus to the decline in Aboriginal numbers following white settlement,[16] and quite apart from its physical and mortal effects the epidemic must have been a terrifying experience for the Aborigines who probably had no previous experience of such a virulent, fatal epidemic disease. Secondly, the sketchy evidence available (Cumpston, 1914) suggests the disease spread widely through south-eastern Australia, and may have reached as far as the west, north-west and north coasts from its presumed point of introduction on the east coast of New South Wales. This implies a greater degree of contact between tribes and local groups than has usually been allowed, and suggests that the nomadic Aborigines before contact with European settlers may not have been so

[16] Tidswell (1898), quoted by Cumpston (1914: 172) stated that the epidemic of 1789 spread over the whole continent, was maintained until 1845, and killed one-half of the Aborigines in the southern part of Australia.

isolated from one another as to discourage the spread of other communicable diseases, once introduced. However, smallpox is a particularly infectious disease, and furthermore it is likely that the devastating effects caused those still incubating the disease to flee to adjacent tribal territory, speeding the disease on its way across the continent.

Some writers have argued that the disease may not have been smallpox (at least in some of its alleged appearances among Aborigines) but a particularly virulent form of chickenpox. Some support for this argument comes from the absence of recorded smallpox among members of the 'first fleet', and the non-infection of settlers during the first epidemic, when many of them were presumed to have been non-immune.

CHICKENPOX AND MEASLES

These 'infectious diseases of childhood', particularly measles, have caused epidemics in isolated Aboriginal communities. Chickenpox has not drawn much attention in recent decades, probably because epidemics of this infection do not seem to have caused serious morbidity or sequelae. Measles, on the other hand, is not infrequently accompanied by serious complications—bronchopneumonia, middle ear infection—particularly in malnourished populations and where 'virgin soil' epidemics affect all age-groups. The last recorded major outbreak of measles among Aborigines occurred in the Northern Territory between October 1965 and October 1966, involved 35 Aboriginal communities, and affected 26 per cent of the 8,920 Aborigines in these communities (Langsford and Vawser, 1969). The authors state (p. 225), 'measles has caused great morbidity and not insignificant mortality on missions, settlements and pastoral properties', but no details of morbidity or mortality have been reported for this epidemic. With the advent of a measles vaccine (first employed on a large scale in the epidemic referred to above) measles may be expected to follow diseases such as whooping cough and poliomyelitis and become of minor importance in the supervised communities, but continue to be a cause of morbidity in many 'fringe' communities.

MOSQUITO-BORNE VIRUS DISEASES

Knowledge of the prevalence of human infection with mosquito-borne viruses in Australian Aborigines has proliferated over the last twenty years, but is confined to Queensland and to a lesser extent the Northern

Territory and Western Australia. Most of the research in this field has been carried out by the Queensland Institute of Medical Research, and is continuing. With refinements in serological and tissue-culture techniques, virus groups are becoming increasingly subdivided and the epidemiological picture more complex. The relationship between infection and morbidity is not very clear, and it is extremely difficult to even hazard a guess at the overall impact of these infections, and their sequelae, on the health status of Aborigines.

In 1961, the arbovirus situation appeared comparatively simple, knowledge being restricted largely to dengue and Murray Valley encephalitis. Dengue is thought to be confined to a man-mosquito-man cycle in Australia, although dengue-type antibodies have been found in north Australian bats (Work, 1967). The epidemics are infrequent, and are probably initiated by travellers returning from South-east Asia. Murray Valley encephalitis, on the other hand, together with an ever-increasing number of other viruses which have been isolated in the last ten years or so, appears to involve man only as an accidental event in a vertebrate-mosquito-vertebrate cycle. The majority of infections in this latter group appear to be asymptomatic in most cases, and the prevalence of antibodies to some of these viruses may have little or no relevance to Aboriginal ill-health.

While dengue epidemics appear most likely to cause temporary incapacity of the greatest number involved in an epidemic, Murray Valley encephalitis is likely to result in occasional serious sequelae, and deserves most attention on this basis. Mackerras (1951: 9-12) reported an outbreak of MVE on Mornington Island in the Gulf of Carpentaria in which three cases developed severe paralysis. The clinical case incidence appears to have been around 21 per cent, and 24 per cent of the cases required hospitalisation. The three cases severely paralysed represent nearly 1 per cent of the population of about 350—a not inconsiderable proportion to develop serious handicaps. This outbreak appears to have been a major stimulus leading the Queensland Institute of Medical Research to pursue the detailed study of arboviruses in Queensland. As a result of investigations throughout northern Queensland, the prevalence of antibodies to dengue, Murray Valley encephalitis, some Afro-Asian arboviruses such as 'Sindbis' (believed to be a cause of epidemic polyarthritis) and a number of distinct 'new' arbovirus entities such as 'Kunjin' and 'Kokobera' have been documented. While serological cross-reactivity between many of these viruses has made it impossible to be certain of the exact serological prevalence of each, there is no doubt from the figures

that north Queensland Aborigines have been repeatedly involved in arbovirus epidemics and live in areas where zoonotic arboviruses are endemic in local fauna and livestock. Doherty *et al.* (1959:9), testing sera from children and young adults on ten Cape York and Gulf missions and settlements in 1957, found arbovirus antibodies in 71 per cent, with most of the reactions being to Murray Valley encephalitis[17] (60 per cent of those tested) and concluded at the time that the last spread of this disease occurred in 1954.

An arbovirus first isolated from mosquitoes collected at Townsville and subsequently named 'Ross River Virus', has been suspected since 1964 of being responsible for 'epidemic polyarthritis', and it now appears likely that this virus, rather than the serologically-related Sindbis virus, is responsible for epidemics of this disease in north Queensland and the Northern Territory. Inquiries made from doctors by the Queensland Institute of Medical Research indicate that a minority of patients (probably not Aboriginal) infected with Ross River Virus suffered recurrent joint pains and swellings for up to nine months after the initial illness.

In Queensland, the different arboviruses (as detected serologically in man) show varying prevalences from place to place, and varying degrees of endemicity over time—a characteristic of most vector-borne diseases because the main vectors are influenced by climate, season, and terrain.

In the Northern Territory, Doherty *et al.* (1968:6, 1969:5) commented that antibodies to Group A arbovirus (includes Ross River Virus) and to Group B arbovirus (dengue, Murray Valley encephalitis, etc.) were present among sera which included samples from Aborigines, and that 'results on Aboriginal children suggest a lower infection rate than . . . in north-west Queensland'. Beech *et al.* (1953) had found that half of the Aborigines tested in a survey of Aboriginal communities throughout the Northern Territory showed antibodies to Murray Valley encephalitis (implying past infection with MVE or another related Group B arbovirus) and suggested endemic foci in the north of the Northern Territory. An epidemic may have occurred in the Northern Territory in 1951, but according to Beech *et al.* was not noticed to produce sufficient clinical disease to be recognised.

In Western Australia, Stanley and Choo (1961) demonstrated antibodies against Group A (polyarthritis) arbovirus in Aborigines in the

[17] Later reports usually refer to the 'MVE-Kunjin-Alfuy-Japanese B encephalitis' group, or 'Murray Valley Encephalitis subgroup', indicating the decreasing confidence in the specificity of serological tests—a consequence of the identification of 'new' serologically cross-reacting viruses.

Central Desert (100 per cent);[18] Kimberleys (50 per cent); Leonora—south-central Western Australia (22 per cent); and Carnarvon (8 per cent). The prevalence of antibodies to Group B (Murray Valley encephalitis, etc.) was nil in the Central Desert, 92 per cent in the Kimberleys, 4 per cent at Leonora and 17 per cent at Carnarvon.

It is apparent, therefore, that arbovirus infections have been very common in Aborigines throughout northern and central Australia; that Group A arboviruses may be responsible for temporary morbidity from polyarthritis, while some members of Group B (in particular, strains of Murray Valley encephalitis) may be responsible for more permanent sequelae including paralysis. Encephalitis-producing arboviruses are known to cause mental deterioration or intellectual impairment of varying degrees in a proportion of cases (Work, 1967:39) and studies on the association between MVE and intellectual impairment among Aboriginal schoolchildren are necessary before such infections can be dismissed as being unimportant contributors to Aboriginal morbidity. Unfortunately, from the viewpoint of this suggestion, research on arboviruses has concentrated on the serological, virological, and entomological aspects, with little or no attempt to correlate this information with clinical or psychological studies. The need for further research cannot be better indicated than by considering the implications of this statement by Work: 'Some who display an apparent complete recovery [from arbovirus encephalitis] may later show emotional instability under stress or inability to concentrate, or may just no longer do well in school or at other demanding mental tasks.'

INFECTIOUS HEPATITIS

This viral disease is believed to be spread by the faecal-oral route, and if the high prevalence of other intestinal pathogens in Aboriginal populations is an index of potential transmission, infectious hepatitis would be expected to be a major health problem among Aborigines. Curiously, the disease is barely mentioned among the reports of illnesses among Aborigines, although various Northern Territory Reports mention it under 'Health' and imply that outbreaks occur in Aboriginal communities without stating this directly.

'Australia' antigen, a factor in the blood which appears to be associated with 'serum' hepatitis and may be a hepatitis virus or a part of the virus,

[18] These percentages are for positive reactions to only one of two different tests carried out. The other test gave somewhat lower reaction rates, but with similar rankings.

has been stated to be present in 5 per cent of the Aboriginal population tested (Blumberg and Melartin, 1966)—the details of the population surveyed not being given. Barrett (1970) states that no 'Australia' antigen was detected in thirty-eight sera from Aboriginal children, presumably in Queensland.

Apart from the limited evidence deduced from Northern Territory reports that infectious hepatitis is endemic and occasionally epidemic in Aboriginal communities, the contribution of this disease to Aboriginal morbidity remains obscure.

TRACHOMA

Trachoma ('sandy blight') is one of the few diseases believed by Abbie (1966) to have been endemic in the Aboriginal population prior to white settlement. Mann (1957b), on the other hand, believes trachoma was introduced by European and Chinese settlers. Whatever its origins, trachoma has been very prevalent among Aborigines, although often stated to be a relatively mild disease in most cases, with a low incidence of complications and of blindness. Mann has found prevalence of trachoma to be around 60 per cent in Aboriginal populations of the Warburton Ranges, the South West, the Eastern Goldfields and the Kimberleys in Western Australia, as against a prevalence of 2-8 per cent in the white population of the same areas. Without treatment, Mann (1963) states that approximately 12 per cent of Aborigines who contract trachoma suffer impaired vision, so that a 'low incidence of complications and of blindness' in Western Australian Aborigines would still be sufficient to cause blindness (partial or total) in 7 per cent.

A survey of Northern Territory Aborigines by Flynn (1957) showed an overall prevalence of trachoma of 62 per cent out of 4,876 examined. The prevalence varied from 54 per cent in the north (Darwin and Gulf), to 61 per cent in the Victoria River-Barkly Tablelands area, to 77 per cent in the Alice Springs area. Flynn showed that the infection was much more likely to be severe in the Alice Springs area—the ratio of healed cases with impaired vision to total healed cases rising from 8 per cent in the north to 42 per cent in the south. The proportion with impaired vision out of the total examined ranged from 2 per cent in the north to 17 per cent in Central Australia.

The greater severity of the disease in the drier areas has been attributed to the combined effects of the stress on the conjunctiva produced by the hot, dry, dusty climate and repeated secondary infection due to poor

personal hygiene, the use of clothing and blankets which are rarely washed, and flies.

Trachoma has been found in high prevalence in the drier areas of South Australia—96 per cent at Musgrave Park in the north-west of the State, and 74 per cent at Coober Pedy in the central north, and was responsible for visual disability in 3 per cent of South Australian Aborigines (Moore *et al.*, 1965). The prevalence of infection and morbidity in Queensland and New South Wales is not documented as far as the writer is aware.

Hardy *et al.* (1967) have demonstrated that the conjunctival cytopathology associated with trachoma is improved when hygiene is improved, and further improved with local and systemic antibiotics. It is a disease which is therefore sensitive to public health action and an improvement in living standards. The prevalence of blindness and lesser degrees of visual impairment among Aborigines may be expected to diminish markedly with effective community health programs, although the prevalence of benign infections may not be so easily reduced in the Aboriginal environment.

MALARIA

Malaria has not been recorded as having been transmitted in Australia since the last endemic focus was eradicated from the Aborigines in the Roper River area of the Northern Territory by mass drug administration in 1962. Malaria was previously endemic (but not highly endemic) in several areas in north Australia, and epidemics have been reported.

Although malaria has been eradicated or has died out throughout northern Australia, the *Anopheles* vectors are still present. The increased tempo of northern development, coupled with the transfer of labour and personnel to the north from malarious areas in Papua New Guinea and South-east Asia, threatens the reintroduction of malaria into the Aboriginal population. There is also a possibility of the introduction of 'new' vector mosquitoes from New Guinea or South-east Asia, many of which are much more efficient as vectors than the naturally-occurring species.

The main threat to Aboriginal health from the reintroduction of malaria is unlikely to be malaria itself, but the diversion of health resources to achieve eradication again before the disease becomes more firmly established. Changes in Aboriginal ecology in northern Australia may heighten or lessen the threat from malaria, depending on the ability of the preventive health services to adapt to changing conditions, and continued vigilance is necessary.

SYPHILIS AND OTHER VENEREAL DISEASE

There is no doubt that the venereal diseases occur amongst Aborigines, and many references to them are to be found in the medical and popular literature on Aboriginal health. The prevalence of venereal disease is speculative, however, and the extent of morbidity from venereal infections is generally not known. The lack of a clear picture of the occurrence and effects of venereal diseases arises from several interrelated factors: the reluctance of cases of venereal infection to report for diagnosis and treatment; reluctance of individuals in a survey to submit to examination of the genitalia; and lack of a specific test (serological or skin test) with the exception of tests for *lymphogranuloma venereum*. Although there are specific tests for treponemal infection, these do not differentiate between venereal syphilis, endemic (non-venereal) syphilis and yaws except in areas where the latter two treponemal infections may reasonably be excluded.

Thus serological surveys for treponemal antibodies in the Aboriginal populations of north and central Australia do not provide the epidemiological picture of venereal syphilis that they might do in the white population. Yaws and possibly endemic (non-venereal) syphilis are disappearing diseases in the northern and central Australian Aborigines but the decline in prevalence appears to be recent, and in many areas transmission has not yet ceased. The problem of establishing the separate prevalence of venereal syphilis in such areas is discussed further on p. 165 when considering other treponemal diseases (yaws and endemic syphilis), and it is of no help to this study to list the various reports of individual cases of venereal syphilis in Aboriginal communities.

Gonorrhoea has been particularly prevalent in Aboriginal populations in the past—as judged from admission statistics for the 'Native Hospitals' in places such as Darwin forty or more years ago.[19] Its present prevalence is not apparent from published data. There is no really satisfactory serological test for gonorrhoea, the Gonococcal Complement Fixation Test being a relatively poor indicator of infection except in cases of advanced gonococcal arthritis, etc., lacking specificity and reliability (Garner *et al.*, 1972). The following figures may as easily exceed as equal or fall short of the real past prevalence of gonorrhoea, and are presented only as 'facts' which have relatively little epidemiological meaning.

[19] According to Hackett (1936), there were 289 admissions for gonorrhoea at the Kahlin Beach Native Hospital during the period 1929-33. These cases represent 23·4 per cent of total admissions during the period.

Allen (1963) surveyed three groups of Aborigines at Cundeelee Mission in the south-east of Western Australia, with the following results:

Aborigines from 'spinifex desert' country 7/66 (10·6%)GCFT+
Aborigines from South Australia 10/27 (37%) GCFT+
Aborigines from Kalgoorlie/Karonie 7/23 (30%) GCFT+

Moodie *et al.* (1970), from a 1968 sample survey of 1,541 Northern Territory Aborigines of all ages from localities throughout the Territory, reported positive GCFTs in only 21 (1·4 per cent). Some figures for the Darwin area sighted by the writer put the GCFT rate for the Darwin area at over 30 per cent in 1965, but such high figures for this area were not found in the 1968 survey. The difference may be explained by different criteria for positivity in different serological laboratories, and this in itself is a further reason for ignoring GCFT rates as indices of gonorrhoeal infection.

There are no recent figures for the clinical prevalence of gonorrhoea. Twenty-five years ago, Fryberg (1946) reported the prevalence of Aborigines 'suffering from gonorrhoea' at three Queensland Aboriginal settlements. It is not stated how the survey was carried out or the diagnosis established, but the figures quoted show a high prevalence at Cherbourg (5 per cent of males, 11 per cent of females) and somewhat lower figures at Palm Island and Woorabinda. Again, because they are out of date, such figures are barely relevant to the present study, and the same could be said for similar figures quoted by Musso (1944, 1945) for Western Australia.

Gonorrhoea in the acute stage is probably not responsible for much morbidity, and the effects of chronic infection (gonococcal ophthalmia of the new born, gonococcal salpingitis and sterility, and urethral stricture) do not figure in current reports on Aboriginal ill-health. Because of the opprobrium which the sufferer faces, and may always face, and the difficulty in obtaining adequate prevalence data from surveys of 'volunteers', the true prevalence of this infection is likely to remain obscure until a more specific serological test is developed. The present world increase in gonorrhoea and the development of antibiotic-resistant strains are likely to be reflected in the Aboriginal and part-Aboriginal population to the extent that part-Aboriginal women offer themselves for prostitution in the white community and promiscuity features in the Aboriginal communities. Taking into account general attitudes to promiscuity, and economic recessions in rural and urban areas, an increase in this health problem can be expected.

Lymphogranuloma venereum, a venereal infection with a 'large virus' related to trachoma virus (both now considered to be a small bacterium rather than a true virus), was reported as a microbiological diagnosis for the first time in Australia by Cook and O'Rourke in 1969. The patient, a migrant, had apparently acquired the infection in Australia. The authors say, 'The fact that the patient's most recent sexual contacts were with Aboriginal women [in the Pine Creek area of the Northern Territory] suggests that LGV may be present in the Aborigines, although it has never been reported in them'. If this disease is in fact present in the Aboriginal population, it is to some extent surprising that it has never been reported through the occurrence of its complications—particularly rectal stricture in women detected during hospitalisation for childbirth.

Ulcerating granuloma of the pudenda is another venereal disease which was common among Aborigines in the past—Hackett (1936) reported it as being responsible for 11 per cent of admissions to the Kahlin Beach Native Hospital during the period 1929-33.

Watsford and Alderman (1953:50) reported that, since August 1946, 59 cases (17 male and 42 female) had been treated at the Darwin Hospital, all being full-blood Aborigines from the northern half of the Northern Territory, mostly from coastal communities in Arnhem Land. This number represents 3·3 per cent of Aboriginal admissions during the period. The authors state that the 'incidence' (of hospital admissions for this disease) had been relatively static since 1946 'if we take into account the fact that more intensive investigations and surveys have been carried out over the last two years'. An occasional case had been reported from the Alice Springs area (i.e. the southern half of the Northern Territory).

The only clue to its apparent rarity in recent years is contained in a statement by Elphinstone (1960):

> Twelve years ago, this disease was very common throughout the Kimberley Division [of Western Australia]. It is now very uncommon. Since 1959, only two cases have been treated at Derby Native Hospital and a third at Kalumburu Mission. Presumably this welcome change has resulted from the natives seeking treatment, with antibiotics, at an earlier stage.

YAWS AND SYPHILIS

Early records frequently refer to 'syphilis' among Aborigines in many widely separated areas of Australia. Hackett (1936a), after examining these references closely, concluded that most if not all these references could be descriptions of yaws rather than syphilis. Certainly yaws has

existed among Aborigines in the humid north of Australia and was an Aboriginal health problem of some magnitude up until World War II, since when clinical cases of early yaws have been rare. It is probable that yaws in the north was another one of the relatively few diseases that existed among Aborigines prior to white settlement, although it is not clear whether the disease was brought by the Aborigines when they populated the continent or was introduced through later contacts with traders from the islands to the north of Australia.

Pathological changes in Aboriginal bones from south-eastern Australia have been regarded as evidence of 'syphilis', but again such changes are possible with yaws or endemic (non-venereal) syphilis.

While it is possible to argue at length on the origins and nature of the treponemal (spirochaetal) disease in central and southern Australia in the past (climatically-modified yaws *v.* endemic (non-venereal) syphilis *v.* venereal syphilis *v.* a treponemal infection unique to southern Australian Aborigines), the Aborigines' past experience of this type of infection is important today because it makes it almost impossible to establish the prevalence of venereal syphilis throughout the present full-blood population of 'Empty Australia'. Seroreactivity to tests for 'syphilis', quoted below, reflect the pattern of a declining but sometimes still high prevalence of an endemic, non-venereal disease, and an unknown but probably low prevalence of venereal syphilis. Cases of venereal syphilis (primary sores) are observed from time to time, but so are early (secondary stage) cases of yaws in the north (Garner *et al.*, 1970).

In the Northern Territory, an extensive clinical and serological survey in 1968 (Garner *et al.*, 1972; Moodie *et al.*, 1970; Moodie *et al.*, in press) showed that 25 per cent of over 1,500 Aborigines examined had treponemal antibodies to the Treponema Pallidum Immobilisation (T.P.I.) test, while clinical evidence of treponemal infection was limited to the middle and late (generally non-infectious) stages and was uncommon. Seroreactivity rates were around 80 per cent in older adults in the north, around 60 per cent in the south, were lower with successively younger age-groups, and were nearly zero among children in Central Australia. In the northern half of the Territory, 10 per cent of children showed seroreactivity in the absence of clinical signs of treponemal disease, and seroreactivity among children under 5 years indicated that transmission had been occurring since 1963. The pattern was suggestive of a 'disappearing' disease which had not yet disappeared, and a disease in which most of the 'cases' were sub-clinical (not diagnosable on clinical grounds) at a point in time. A small number of high-titre serological reactions in

adults suggested that venereal syphilis was a component of this picture. The last stronghold of yaws appeared to be in Arnhem Land, and it was from an isolated group in the interior of Arnhem Land that two children with typical florid secondary yaws were observed (after admission to the Darwin Hospital) by the writer in 1968. Maningrida Settlement, established fifteen years ago on the north coast of Arnhem Land, showed the highest serological prevalence of any Northern Territory Aboriginal community surveyed, serological evidence of recent transmission to young children, and appears to have been the major focus of treponemal activity in the recent past.

Venereal syphilis was stated to be unknown in the Northern Territory Aboriginal population before 1939 (Hackett, 1936; C.E. Cook, personal communication, 1971). Since then there have been increasing opportunities for its repeated introduction and spread within the Aboriginal population, and with diminishing cross-immunity from yaws venereal syphilis threatens to be an increasingly important factor in Aboriginal morbidity in the Northern Territory.

In other States, surveys in recent years have revealed a rather similar picture—seroreactivity to tests for 'syphilis' common, clinical signs of active treponemal disease rare or absent, or confined to signs of long-standing, usually 'burnt-out' disease, and no clinical evidence of venereal (or congenital) syphilis.

In the south-east of Western Australia, Allen (1963) found 52 per cent (mostly adults) were treponemal antibodies (VDRL test) at Cundeelee Mission, with a subgroup originating from the spinifex desert country having 74 per cent seroreactors. There was 'almost complete absence of cutaneous lesions or late bone or joint changes' and no suspicion of congenital syphilis among the children. Further north, in the Great Sandy Desert of Western Australia, Elphinstone (1971) found 67 per cent of sixty-three desert Aborigines examined between 1958 and 1967 were seroreactors (Wassermann reaction) eight showing signs of late yaws and one with 'papillomata resembling early yaws'. Among twenty-five 'semi-sophisticated Aborigines' from the same area, 24 per cent were seroreactive. Davidson (1957) reported a serological prevalence of 'somewhere between 20 per cent and 30 per cent' for Aborigines in the north and north-west of Western Australia, and remarked 'only seven cases of yaws [were] admitted to hospital, along with five of granuloma [of the pudenda] and six of syphilis . . . late manifestations of a tertiary nature are seldom seen'.

In Queensland, Jose *et al.* (1969) reported only three children reacting

to the TPI test from among 481 children in missions and settlements throughout the State—a prevalence of 0·6 per cent. The three seroreactors came from communities in the Cape York Peninsula-Gulf area. There were fourteen children reactive to the less specific VDRL test (3 per cent), but the additional reactors were classed as 'false positive' reactors. Doherty (1966) reported eighteen TPI reactors among ninety sera (20 per cent) from Aborigines of all ages at Arakun and Weipa on the north-west coast of Cape York Peninsula. The majority of reactors were adults. Doherty (1967) mentions that mission staffs considered that 'yaws' was widespread in the area before World War II.

Thus the picture of treponemal disease among full-blood Aborigines is one of a relatively high prevalence of seroreactors in most adult groups, with a tendency to higher rates in the more isolated communities having less contact with centres of white settlement or industry; very few cases of active primary or secondary stage infection; a low prevalence of tertiary (late) disease; and no recorded evidence of congenitally-acquired 'venereal' syphilis. Sporadic cases of venereal syphilis are reported in Western Australia and the Northern Territory, and may also occur in Queensland (but are not reported in the literature). Age-group reactor rates to serological tests indicate a steadily declining incidence of endemic treponemal infection over the last fifty years or so, although transmission has occurred recently in some areas. The disappearance of the endemic treponemal infections may be attributed to the widespread use of penicillin and other antibiotics since the 1940s (for a variety of non-treponemal infections), and changes in Aboriginal living conditions in the past fifty years. The residual serological effects of past treponemal endemicity obscure the prevalence of sporadic venereal syphilis which, on both ecological and immunological grounds, has a better chance of spreading throughout the Aboriginal population now than in the past.

The prevalence of treponemal infection in the part-Aboriginal population in 'Economic Australia' is unknown; where it occurs it is likely to be due to venereal transmission, as the endemic (non-venereal) treponematoses are not recorded and probably do not now occur in this area.

LEPTOSPIROSIS

There are a large number of leptospiral infections of animals which are communicable to man in Australia. One or more serotypes cause 'canefield fever' in Australia, but it is stated that any serotype may cause severe symptoms (Yager *et al.*, 1967). Severe forms of the disease are

characterised by fever, jaundice, and haemorrhage, and a variable case mortality rate of up to half those infected. Milder cases, without jaundice, but with fever, headache, congested conjunctivae and meningeal signs, have a more favourable outcome. The disease is contracted through contact with the urine of infected animals (rats, dogs, livestock, bandicoots, etc.) in water or directly. The disease is more common in the tropics and among those in contact with livestock.

Following a number of cases at Lockhart River Mission on the east coast of Cape York Peninsula which resembled the benign form of leptospirosis, Mackerras and Emanuel (1957) found significant titres of leptospiral antibodies in the sera of one-third of sixty-two Aborigines tested. A later survey of Aborigines from the Gulf coast revealed antibodies in 10 per cent of 170 sera tested. Outbreaks are known to have occurred in the Northern Territory in recent years, and the disease may be presumed to occur, with varying incidence and prevalence, right across the north coast among full-blood Aboriginal communities. Its importance in the overall Aboriginal morbidity pattern cannot be estimated from the limited serological evidence, but it appears unlikely that it contributes more than relatively infrequent, short-term feverish illness, and negligible mortality. Cases of the severe form (with jaundice) may occur, but have not been noted in the literature. Its serological prevalence is an index of the presence of animal reservoirs in contact with man or contaminating the water supply.

MISCELLANEOUS INFECTIOUS AND PARASITIC DISEASES

Apart from those diseases which have already been discussed as being important causes of Aboriginal morbidity, or particularly common in the Aboriginal population, there are a number of relatively rare infections which have been recorded over the years, but which hardly figure in the morbidity pattern because of their rarity. Diseases such as psittacosis ('parrot fever'), 'Q' fever, and histoplasmosis are occasionally detected *post hoc* in serological and skin-test surveys and clinical cases of torulosis (cryptococcosis), actinomycosis, nocardiosis, chromoblastomycosis—all systemic or dermal fungus infections—and infections such as brucellosis, melioidosis, and toxoplasmosis are noted from time to time. The list grows steadily from year to year as diagnostic techniques are refined and carried to the more remote areas. As many of these infections are communicated to man more easily in tropical climates or where ecological factors (environment and behaviour) increase the chances of contact with

the infecting organisms, it is not surprising that the rarer infections are often first detected in Australia in the Aboriginal population, or that Aborigines contribute more cases than would be expected from their proportions in the total population.

Amongst the rarer infections listed, *torulosis* probably leads as a cause of morbidity and mortality. The agent is a yeast-like organism, *Cryptococcus neoformans*, which has been found in soil, bird excreta, and a variety of animals. In man it may produce skin infection only (pustules or ulcers), or a generalised infection, probably in the lungs at first, but frequently resulting in a severe and often fatal meningitis.

In the Northern Territory, Crotty (1965) reported ten cases among Aborigines, all presenting as meningitis, of whom eight died. The author commented that torula infections without meningitis were probably being missed and that the true incidence of torulosis was probably higher than indicated by his series. He noted that *C. neoformans* was isolated in Aboriginal cases of meningitis in Darwin more often than any other organism, and estimated that torulosis caused between 0·5 and 1 per cent of deaths 'in these people' (? Aborigines in Darwin).

In Queensland, Geaney *et al.* (1956) reported fourteen cases between 1949 and 1954, of whom four were Aboriginal. All except two non-Aboriginal cases died. The Aborigines in this series came from Yarrabah Mission (2 cases) and Woorabinda Settlement in Queensland, and Casino in northern New South Wales. Another case at Woorabinda was mentioned as having been recorded elsewhere.

Sutherland (1969) reviewed twenty-eight cases of cryptococcosis of the nervous system in Queensland between 1959 and 1968, of whom five were Aboriginal. From an estimated mean Aboriginal population of 17,103, he calculated the nine-year incidence among Aborigines at 29 per 100,000. This is an annual incidence of 3·2 per 100,000, compared with a calculated incidence of 0·18 in the non-Aboriginal population, a figure around one-eighteenth that for Aborigines.

In Western Australia, Elphinstone (1966) reported seven cases among Aborigines in the East Kimberley and Hall's Creek area since 1957, one being part-Aboriginal. All died from the infection. The author suggested that the infection in this area (detected in Aborigines only) may have originated from Top-knot pigeon droppings, as these birds were 'commonly found in, and around native camping areas'. He estimated the annual torulosis fatality rate among Aborigines of the area from which cases came at about 1 per 1,000, compared with an overall fatality rate of about 1 per 1,000,000 in the United States.

In South Australia, Richardson (1968) reported on twelve cases, of which five were Aborigines. However, four of the Aborigines came to Adelaide from the Northern Territory for treatment. No further details of the locality or origin were given.

Toxoplasmosis (infection with a protozoan parasite, *Toxoplasma gondii*) is common in some Aboriginal populations surveyed for antibodies. Cook (1959) found antibodies in 39 per cent of Aborigines at Lockhart River Mission on the east coast of Cape York Peninsula, and in 9 per cent on missions on the west coast. The prevalence in adults and children was very nearly equal. However, Kaufman (1967) states that approximately 50 per cent aged over 20 in the United States show antibodies, so the Aboriginal prevalence is hardly remarkable.

Toxoplasmosis is a curious infection in that, for such high prevalence figures for antibodies, very little is known about its method of transmission to man. It is found in every common kind of meat eaten by man, but is as common in vegetarians as in meat-eaters. It has also been found infecting pets, especially cats. The infection in meat is destroyed by light cooking. Congenital transmission in man can occur, but appears to be very uncommon in relation to the serological prevalence. It has been suggested that *Toxoplasma* can be transmitted in the eggs of the dog or cat roundworm (*Toxocara* species) and although this theory has recently been discarded as untenable, there is evidence that the cat may be an important source of infection in man (Hutchison, 1967).

The disease, when congenitally transmitted, may cause hydrocephalus, liver enlargement, and blindness from retinal damage, or may be limited to various degrees of retinal damage only. Presumably many congenital infections cause no ill effects. In adults, it may cause retinitis, or a prolonged low fever with glandular enlargement, pneumonitis, myocarditis, encephalitis, or skin rashes (Kaufman, 1967). These manifestations have not been reported in Aborigines as the effects of *Toxoplasma* infection, and the place of toxoplasmosis in Aboriginal morbidity remains obscure.

Fungal infections of the skin and scalp amongst Aborigines have been studied by Donald (1959), who states that the infection rate for *Trichophyton violaceum* (an important cause of scalp 'ringworm') was at least 30 per cent for South Australian part-Aboriginal children, being nearly as prevalent as in North Africa and Israel 'where the disease is most prevalent'.

Donald (1960) has also drawn attention to the risks of picking up *Trichophyton mentagrophytes* (a cause of body ringworm) from kangaroos. If kangaroos were naturally infected before white settlement, the disease may have been prevalent among nomadic Aborigines.

Although rarely responsible for serious morbidity—in the sense of crippling or disabling disease—the visible 'ringworms' on face, scalp, and body have important social consequences for Aborigines (particularly part-Aborigines) in mixed communities. Like 'worms' and 'headlice', their prevalence reinforces discriminatory attitudes and behaviour by white Australians and therefore contributes in no small way to the assimilation barrier. 'Ringworm' is a prime example of diseases which have greater psychological and social than physical consequences, particularly in a plural society.

ECTOPARASITES

This final category of 'infective and parasitic diseases' includes head and body lice and scabies. Such conditions are medically trivial in that they produce little morbidity and no mortality, and the agents are not known to be vectors of any disease currently occurring in Australia. Their main effects are skin irritation, and sometimes a degree of secondary infection brought on by scratching. Severe effects are uncommon.

However, these infestations are of utmost importance to the Aboriginal population because of their social consequences, as was noticed by Williams (1965) and Aitchinson (1965). They contribute to prejudice and practices of discrimination and segregation in mixed communities. Part-Aboriginal schoolchildren in mixed schools may suffer considerable indignity when singled out by teachers for inspection and treatment for headlice. Reactions by many white Australians to headlice appear to be as strong as they are to diseases such as leprosy and 'worms', and the social consequences for infested individuals within a high school group can be imagined. It is not difficult to postulate life-long psychological consequences for an Aboriginal teenager 'discovered' at a mixed school to be suffering from headlice infestation and accidentally or intentionally identified in the course of treatment, or when he is excluded from school until the infestation has been eradicated.

Such infestations are favoured by an overcrowded home environment, and are easily passed between children who have to share a bed. Current fashions for long, uncombed or 'afro' hairstyles among teenage boys seem to favour both the persistence of headlice infestations, once acquired, and spread from one child to another.

In a New South Wales coastal part-Aboriginal community, the writer observed 16 per cent of children with 'nits' during a medical survey in 1966-7. A repeat survey of the same community in 1971 showed a

prevalence of 35 per cent. Part of the increase may be attributed to a more careful examination for the presence of 'nits' during the second survey, but there was a noticeable increase in the infestation rate among teenage boys—who had changed from 'short-back-and-sides' to long or shoulder length hair styles over the five-year period.

Davidson (1957), reporting on a survey of Aborigines in the Warburton Range area of the Western Australian desert observed, 'A number . . . had head lice but this condition was much less frequent than it is among natives and half-caste natives in our country towns'.

Scabies does not appear to be particularly common, although mild infestations, with little or no 'scabies rash', are easily missed in dark-skinned individuals.

COMMUNICABLE RESPIRATORY INFECTIONS

The International Classification of Diseases places diseases such as influenza and the 'common cold' within a separate major category covering all diseases of the respiratory system; in reviewing causes of Aboriginal morbidity, it is more convenient to consider these respiratory infections together with the other communicable diseases which have been reviewed above.

With a few exceptions, such virus infections are usually acquired by inhalation of infective droplets or particles in the air, produced by an infective individual coughing, sneezing, or talking. However, in situations where personal hygiene is of a low standard, there are many opportunities for such infections to spread by the direct transfer of nasal, oral, and conjunctival secretions—on fingers, towels, clothing, bedding, and possibly by flies. The predilection of flies to 'feed' around lips, nostrils and the inner corner of the eyes, and the tolerance of 'outback' Aboriginal children and adults to large numbers of flies in constant attendance at these sites is well known. In Aboriginal communities it is not therefore a safe assumption that respiratory infections are spread exclusively by the airborne route, or that such diseases may be controlled by measures directed primarily against airborne spread.

The major contribution of respiratory infections to Aboriginal mortality has been noted in earlier chapters, but the mortal effects of the acute 'communicable' respiratory infections are somewhat indirect. People do not die of the common cold, and rarely die of influenza; however, people do die of the secondary bacterial infections (pneumonias) to which the common cold and influenza predispose. There are other

predisposing factors such as age, climate, smoking, state of immunity, nutritional status, 'general health', and so on, making it difficult to determine the contribution of the acute virus infections to respiratory morbidity and mortality in the individual case, and impossible to determine for a population without detailed longitudinal study.

The morbid effects of the acute respiratory infections may be divided into the effects of the primary infection—a transient episode with varying degrees of incapacity as may be experienced by any individual with 'flu'; the effects of the secondary infections which follow in a proportion of cases—bronchitis, pneumonia, again with varying degrees of severity but occasionally causing serious acute illness and death; and the cumulative effects of repeated primary infection—a somewhat more controversial association with chronic naso-pharyngitis, chronic sinusitis, chronic middle ear disease, chronic bronchitis, etc.

In this section, it was intended to limit the review to the acute primary infections themselves, although the secondary and cumulative effects of the 'simple common cold' and 'influenza' are likely to be responsible for the greater morbidity in terms of 'man-years' of illness and incapacity. The intention cannot be fulfilled, however, because there are practically no data for either incidence or prevalence of 'colds', influenza and other acute upper respiratory tract infections other than the often repeated statements that these infections are very common, and some subjective data obtained by questioning individuals as to their experience of 'acute chest infections' in the recent past.

In a survey reported by Gandevia (1967), Pintubi and Walbiri subjects at Papunya, Northern Territory, were questioned as to the occurrence of 'acute chest illness' in the past two years (1963 to 1965). The criterion of 'acute chest illness' was that antibiotics had been administered for a chest infection, and this criterion does not separate the communicable respiratory tract infections from exacerbations of bronchitis or occurrences of pneumonia precipitated by factors other than the communicable infections. It is of considerable interest, however, that Gandevia found a history of 'acute chest illness' to be over twice as common (85 per cent) among Pintubi who had entered the settlement from the desert within the past eighteen months when compared with Pintubi who had been at the settlement for at least five years (38 per cent). The implications were that the 'new' Pintubi were being exposed to 'new' communicable respiratory infections, to which the 'old' Pintubi had acquired a degree of immunity. Gandevia also found signs of current, presumed '*chronic*' bronchopulmonary disease more prevalent in the 'new' group (79 per

cent) than in the 'old' group (36 per cent), and postulated that this may have been due to a relative failure by the 'new' group to adapt to the communicable infections, although environmental factors may have contributed.

From a survey of New South Wales and South Australian Aboriginal households with a white control group, designed by C.D. Rowley,[20] data were obtained for a history of 'contagious respiratory infection' occurring in the four weeks immediately preceding the questionnaire. The white 'control group' was drawn from white households next door to Aboriginal households in the Eyre Peninsula region of South Australia, and may be assumed to represent the lower end of the white socio-economic spectrum. The results for children under 16 years of age were as follows:

	Proportion	Percentage
New South Wales Aboriginal children	33/689	4·8
South Australian Aboriginal children	3/102	4·1
South Australian white children	6/49	12·2

The result is unexpected in that the white group appeared to have three times the incidence of 'colds' and 'flu' as the Aboriginal group in South Australia, both groups being surveyed coincidentally in space and time. The difference does not prove a particularly high statistical significance, however,[21] and may also be contributed to by different 'standards' of parental diagnosis. Attempts to analyse such figures point to the problems inherent in the comparison of questionnaire results obtained from different subcultures in a population. The determination of the real incidence and prevalence of the more transient infections is dependent on longitudinal studies in depth for those diseases for which there is no suitable *post hoc* serological test. The respiratory virus infections, as a group, are difficult to investigate serologically because of the large number of different serotypes which have to be identified.

Probably the most important knowledge to be gained from current and future research in the field of respiratory disease and functional incapacity is the relationship between the transient communicable infections common in childhood and the chronic diseases—bronchitis

[20] Professor Rowley was, at the time, Director of the Aborigines Project for the Social Science Research Council of Australia. For further details of the survey, see Rowley's *The Destruction of Aboriginal Society* and *Outcasts in White Australia*, in the series Aborigines in Australian Society.

[21] The difference in percentages would occur by chance slightly more frequently than once in twenty similar samples.

and emphysema—common in later adult life. Chronic respiratory disease in Aboriginal populations will be taken up in a later chapter.

Except for a few communicable disease groups—such as puerperal infection, some infections of the newborn, and some skin infections such as impetigo—this concludes the review of the impact of the infectious diseases on the Aboriginal population, looking at disease entities one at a time.

Communicable diseases stand out as very important contributors to Aboriginal ill-health, and to mortality. There is no evidence to suggest that any of the infectious and parasitic diseases in Australia are restricted to the Aboriginal population, nor that Aborigines have escaped any of the diseases present in Australia. However, the Aboriginal population contributes much more than its 'fair share', for a large number of the infections which have been discussed—such as leprosy, worm infestations, torulosis—are of no real significance in the pattern of white Australian morbidity.

White Australians are protected by their largely metropolitan and urban environment, where food handling, water supplies, excreta disposal and building standards are controlled; where disease vectors and animal reservoirs are limited in their contact with man; and where relative affluence and social and legal sanctions on behaviour maintain conditions which do not favour the transmission of disease directly from man to man.

With many of the infectious diseases, children form the major human reservoir, and the high proportion of children in Aboriginal communities with a high local density of population favours the persistent infections like the parasitic diseases. On the other hand, the small size of most Aboriginal communities limits the endemicity of short-term infections like measles, so that these infections tend to be more epidemic in their behaviour than in the larger white communities.

It is apparent from an examination of those infectious diseases which have been studied in geographically separate areas that prevalence and incidence may vary widely—each disease being influenced by local environmental conditions and human ecological patterns. Although there is an extensive body of knowledge on the epidemiology of the great majority of communicable diseases, and the clinical and pathological effects of manifest illness are well documented, less is known of the overall impact of these diseases on a population in terms of the proportion of those infected which will suffer varying degrees of disability, how long

the ill-effects may last, how serious the incapacity will be in a given socio-economic setting. This is to say that not only the incidence and prevalence are variable between communities, but for a given disease and the same incidence and prevalence, the effects are likely to vary also. The effects will also be varied by the presence or absence of other diseases, infectious or otherwise.

It may be concluded that it is hardly possible to compare Aboriginal communities in their morbidity from the infectious diseases, or be able to say that, overall, this community is 'healthier' than that community. One population may be afflicted with leprosy, hookworm, leptospirosis, and yaws, the other with tuberculosis, roundworm, head lice and gonorrhoea as major components of their morbidity patterns. It is only when one community has a lower incidence of *all* important communicable diseases that it may be assumed that it is 'healthier' from the viewpoint of this group of diseases, but even in this case the 'healthier' community may be worse off because of the social or economic consequences of its residual morbidity.

It is this problem of measurement and comparability that has drawn attention to the need for new measurements of health which are not disease-based, like the measurement of 'functional capacity' referred to earlier in this book. If the need for a new approach to the measurement of health and the weighting of diseases is admitted, it can be argued that the determination of priorities for communicable disease control, based on clinical or pathological criteria, may be wide of the mark in terms of 'health' in its broadest context. In the long term, it may well be that for part-Aborigines on the fringes of white society infestations with headlice are more important than gastroenteritis or pneumonia—a viewpoint which would be quite absurd from the strictly medical aspect.

10 MORBIDITY FROM MALNUTRITION AND THE ANAEMIAS

These diseases occur somewhat later in the groupings used in the International Classification of Diseases, but have been advanced here because they are of particular relevance to Aboriginal health and have association with the communicable diseases. The major categories in the International Classification are 'Endocrine, Nutritional and Metabolic Diseases' and 'Diseases of Blood and Blood-forming Organs', but it is convenient to consider the nutritional and haematological components together. Endocrine disturbances, such as goitre, diabetes mellitus, gigantism and dwarfism, occur in the Aboriginal population, but the documentation is not extensive and little is to be learned from the examination of occasional reported cases.

Wise *et al.* (1970) reported twenty-two cases of diabetes mellitus among 210 urbanised Aborigines from the Davenport Reserve, Port Augusta, South Australia—a prevalence of 10·5 per cent. Among those aged 20 years and over, the prevalence was 19 per cent. The authors compare these figures with a prevalence of 2·3 per cent in a white Australian community at Busselton, Western Australia, from a survey employing the same diagnostic criteria. The authors concluded that the higher prevalence in the Aboriginal group may have been due to Aborigines having 'evolved without the ability to cope with the high carbohydrate loads inherent in Western civilisation', and noted a familial pattern of cases and an association of diabetes with age, obesity and myocardial ischaemia.

However, when the community is divided into those with and those without definite diabetes (including a number with 'borderline' diabetes

in the normal group) there is no statistical association of diabetes with obesity.[1] Furthermore, an analysis of the published data shows that there was a likely association[2] of diabetes with part-Aboriginal origins and with having been 'civilised' for ten years or more (surely these two factors are themselves associated) which does not particularly support the hypothesis for inability to cope with a high carbohydrate diet as an Aboriginal trait.

It is of interest from the nutritional viewpoint that Wise and his colleagues classified 21 per cent of females and 2 per cent of males as 'obese'.

Wise *et al.* also reported a communication from A. Proust to the effect that 10 per cent of Bathurst Island Aborigines from the north coast of the Northery Territory showed high blood sugar levels suggestive of diabetes.

'Malnutrition' is a term which covers a wide range of nutritional abnormalities from starvation and famine through varying degrees of dietary deficiency and dietary imbalance to minor deficiencies of specific nutrients; it includes obesity due to excessive calorie intake, and may be extended to cover nutritional disturbances caused by malabsorption of various dietary components—'endogenous malnutrition'.

Starvation and frank deficiency diseases such as beri-beri and scurvy have not been described in the morbidity patterns of Aboriginal populations in the recent past, so that ten years ago it would have been difficult to persuade the 'authorities' that the Aboriginal population *in general* suffered from a nutritional disadvantage. While it was obvious that Aboriginal and part-Aboriginal adults were, on the average, shorter and lighter than European adults, the difference tended to be ascribed to hereditary factors. In recent decades—particularly in racial groups where improvement in the quantity and quality of food has been marked, such as in Japan and in Japanese-Americans—it has become apparent that the average physical size of adults is determined more by a combination of nutrition and freedom from infection in early life than by inheritance, although the latter may play a major role in determining parameters associated with body build and in influencing the adult dimensions ('physique') of individuals. Ten years ago it may have seemed reasonable to derive separate height and weight standards for different 'races',

[1] Wise *et al.* claimed a highly significant proportion of new cases of diabetes (discovered during the survey) were obese. It is possible that the low proportion of obese subjects among the known diabetics (1 in 21) reflected calorie restrictions accompanying treatment.

[2] $0{\cdot}1 > p > 0{\cdot}05$ from χ^2 test.

whereas today it is generally accepted that standards derived from populations where nutrition is 'optimal' and infection rates are low may be regarded as potential standards for most 'races' or populations.[3] There may be a degree of ethnocentric enthusiasm involved in this view, and it would certainly appear that Western 'average' standards for weight (or weight relative to height) may exceed the 'optimum' for a physically active adult. This implies that growth rates for Western children, if determined from mass survey data, may exceed the optimum, particularly with respect to weight for age. Nevertheless, growth standards based on mass survey data from healthy, well nourished populations, such as the 'Harvard Standards', may be accepted as being nearer to the 'potential' than standards derived from nutritionally-underprivileged populations.

There is one situation where achievement of the potential for growth may be dangerous, even fatal. This occurs when a relatively over-nourished foetus is unable to pass through a relatively small pelvic opening during birth, and both mother and foetus may perish. Aboriginal women in Central Australia have been noted to have small pelves (Davivongs; 1963) and Parsons (1964) noted low birth weights among Pitjantjatjara women. It is possible that maternal pelvic size and birth weights in this situation were both determined by nutrition, although it seems possible that selection pressures operating over thousands of years, perhaps, would produce an inheritable factor for 'low birth weight' which could be either maternal or foetal in operation. It remains to be seen whether, with improved nutrition, selection pressures can still operate alongside obstetrical intervention and produce an inheritable factor for a wider pelvic opening to accommodate larger babies. If not, the proportion of obstructed labours which have been noted in detribalised Aborigines and in developing countries may persist and increase as maternal-foetal nutrition improves.

To return to the main theme of this chapter, it has become apparent that growth parameters, as a sensitive index of the nutritional status of a population, indicate that many Aborigines have been at a nutritional disadvantage compared with white Australians—at least since changes in Aboriginal ecology following white settlement, and possibly (but not certainly, in the wider context of health) before white settlement. Increases in growth parameters for the white populations of Western countries indicate that the children of fifty years ago were at a growth disadvantage compared with children today, but children fifty years hence may grow

[3] Genetic factors determine overall adult size in some populations, as evidenced by true 'pygmies'.

faster and become taller and heavier than those of today. Thus there are no absolute standards for optimal growth, but a comparison of growth parameters for Aboriginal communities with standards such as the 'Harvard Standards' allows a comparison between Aboriginal communities in terms of their relative shortfall from the mid-twentieth-century optimum. The measurement of the extent of any shortfall poses some problem, however, since a growth index for a community (of children) must take the age of the individuals into account. That is to say that comparisons between populations of children at different ages, which may have different age-distributions, are only valid if the overall growth indices for the populations incorporate a correction for age—and to a minor extent in the case of small children, a correction for sex. There are several alternative solutions to this problem, none of which are entirely satisfactory, and in the absence of the raw data it is not easy to compare the findings of surveys using different methods of measuring the growth deficit. A brief explanation of the different methods of assessing growth parameters is given in Appendix I, which may be helpful to those readers who are not familiar with the problem.

Recent studies relevant to nutrition among Aborigines may be divided into two categories—those concerned with the growth of children, and those searching for evidence of specific nutritional deficiencies from clinical, biochemical or dietary survey data. In many studies, both approaches have been combined, but it is convenient to consider growth retardation first.

GROWTH RETARDATION

Parsons (1965) observed that Pitjantjatjara infants from the western border between the Northern Territory and South Australia had low birth weights (average 6¾ lbs), but grew rapidly up to the age of four months, catching up with the weight of white Australian infants. Thereafter, however, their weights fell off progressively so that by the age of 2 years Pitjantjatjara infants were about seven pounds lighter than 2-year-old white children (in Melbourne).

Kettle (1966) observed an almost identical growth pattern among Aboriginal children up to 5 years of age from coastal Arnhem Land missions in the Northern Territory. She also noted that the introduction of communal feeding of infants and small children in 1958 had very little effect on this pattern of diminished growth rates after the first four months or so. The low birth weights were attributed to an inadequate maternal diet, and the rapid growth rate in the first four months to the Aboriginal

custom of 'demand feeding'. No explanation was offered in this study for the fall away in growth rates after 'five to six months' but the author concluded, 'much more information is required about the effects of repeated infections, especially gastrointestinal infections . . .'. It may be assumed that the impact of these infections had increased with the aggregation in a poor and crowded environment of a formerly nomadic people, and it is a pity that the early observers, who had contact with Aborigines in their 'natural' state, were more interested in the physical measurements of adults (for studies in physical anthropology) and made few observations on infants and children. The earlier observers were, of course, handicapped in not having access to records of birth from which to determine chronological age, but a comparison of height-weight ratios would be informative today. Kettle noted that the fall-off in the increase in height was more gradual than the fall-off in weight, and in terms of percentiles below the standard remained within the standard range. Up until the age of about 1 year, Kettle's figures show that coastal Arnhem Land infants were above standard values for height (length).

Propert *et al.* (1968) reviewed Parsons's and Kettle's findings and added further data on birth weights and growth rates up to one year for Aborigines from Mornington Island (Gulf of Carpentaria), Santa Teresa (Central Australia) and full-blood and part-Aborigines from twenty-seven localities in Western Australia. The additional full-blood data followed the same pattern of low birth weight, rapid weight gain in the first four months, then a marked slowing in weight gain. Western Australian part-Aborigines showed 'standard' birth weights, but the same pattern of a fall away in weight increase from the age of about 5 months to levels which were intermediate between the full-blood Aboriginal and Melbourne white values at 1 year. The authors were not able to identify the factors responsible for the slowed growth rate, but suspected a combination of the effects of poor diet, lack of parental care and skill in feeding infants, and the occurrence of infection.

Studies to determine causes of Aboriginal growth retardation have been pursued in Central Australia and Queensland. Maxwell and Elliott (1969) reported on their studies at an unidentified settlement in Central Australia which had been investigated since 1965.[4] For the whole group, 68 per cent were at or below the 10th percentile for weight and 59 per

[4] Although not named by the authors, the population size and tribal groups represented identify the settlement as Papunya, 130 miles north-west of Alice Springs. This is a 'desert' area and the Aborigines, when living in the nomadic state, had to range in small family groups over relatively immense areas to hunt and collect sufficient food.

cent at or below the 10th percentile for height. The authors comment that 22·5 per cent being below the 3rd percentile for weight (i.e. below the 'standard' mean minus 1·88 standard deviations) 'argues that these children are malnourished by white standards'. They also noted that approximately one-third of children were below the 10th percentiles for chest and head circumference. The most marked abnormality was noted to occur between the first and third birthdays, but a similar level was recorded in 11-year-olds. An apparent 'recovery' of lost weight-gain in 4-year-olds was suspected to have been produced by excess mortality among the most undersized younger children. The authors state,

> These children would appear to fit into the category of protein-calorie malnutrition of early childhood, which may have its onset in the first year of life. The exaggeration of the abnormal percentiles in the 2nd and 3rd years of life may also reflect a post-weaning phenomenon . . . The persistence of malnutrition in the older age group suggests that these children never fully recover.

One of the most serious implications of Maxwell and Elliott's study is the possibility of intellectual handicapping by retarded brain development in early childhood. While the authors were not able to conclude that reduced brain size accompanied reduced head circumference, they noted a report from Africa in which there was a correlation between reduced head circumference and reduced 'intelligence quotient', and in their own study showed a correlation between reduced head circumference and malnutrition indicated by failure to gain weight.

In looking for causes, the authors noted that Aboriginal children brought up from an early age by white foster parents in 'good surroundings' approximated white norms in their growth parameters; that there was no biochemical evidence of a deficiency of human growth hormone in the settlement group; that lactose maldigestion in Aboriginal children (due to loss of lactase enzyme activity) coupled with a lower lactose content in Aboriginal breast-milk and a fall in breast-milk production after six months appeared to be depriving Aboriginal infants of a proportion of the calories available to white children, particularly in later infancy.[5] The authors attempted to correlate growth retardation with infection, but found that a history of infection was so frequent that the attempt was futile—over two-thirds of children had a history of gastroenteritis and of ear-nose-throat infection, and over four-fifths had a previous history of chest infection. There was, however, an association between malnutrition and mortality.

[5] Lactose, or milk sugar, cannot be metabolised unless converted to dextrose and galactose by the enzyme lactase, secreted by the pancreas and the intestinal lining.

In the Central Australian population studied, Maxwell and Elliott found no evidence of kwashiorkor—a protein deficiency syndrome—nor was there any 'real decrease' in plasma protein levels, and they concluded that the pattern of malnutrition they described was 'probably attributable to calorie lack'.

In Queensland, Jose and Welch (1970) examined the growth measurements of 2,250 children on six settlements since 1967. They found growth retardation (based on height-age and weight-age indices) in up to 56 per cent of children aged 6 months to 3 years at four of these communities, but there were only 16-18 per cent with growth retardation at two missions where supervised infant feeding programs were in operation. Overall, the authors reported a 16 per cent prevalence of severe growth retardation, which was accompanied by anaemia and infection.

Jose and Welch also carried out extensive clinical and biological testing for evidence of specific deficiencies, which is described below, and concluded that,

> nutritional deficiency in pregnant mothers and infants, and inadequate early infant feeding were considered primary initiating factors, but infections, and subsequent intestinal malabsorption, were important secondary factors precipitating and maintaining growth retardation. Intestinal parasites . . . reached heavier loads in growth-retarded children, but played no part in initiating the syndrome.

They also noted that a high proportion of children who had died from 'gastroenteritis' or 'pneumonia', or who suffered from deafness, had a previous history of growth retardation.

Jose and Welch confidently labelled the growth-retardation pattern as being one of protein-calorie malnutrition, and there is confirmatory evidence in their clinical and biochemical findings that the protein deficiency component was greater than in Maxwell and Elliott's study in Central Australia. They classified three out of 600 growth-retarded children (0·5 per cent) as suffering from kwashiorkor. However, the pattern, timing and extent of weight-deficiency in the two studies were identical as far as can be judged from the different ways in which the findings are presented. Neither group of researchers found any definite evidence to support a genetic basis for the growth deficiency, except that the Central Australian study suggested that there may have been a genetic component in the lactase deficiency.

Jacobs and Clements (1968) have reported a longitudinal study for full-blood children up to the age of 4 from twelve missions and settlements in the northern part of the Northern Territory—an area which is climatically and economically similar to north Queensland in many

respects. The survey was conducted during 1966 and 1967. The authors' conclusions with respect to growth failure are similar to those reached by Maxwell and Elliott in Central Australia and Jose and Welch in Queensland, but in observing the growth of children over a period, the authors noted that, beyond the first six months of life, approximately half the infants showed retarded growth, and many of these 'make little or no further weight gain during the remainder of the first year'. They also observed that many infants who had maintained satisfactory growth rates during the first year fell behind in the second and third years.

Communal feeding for infants, commencing some time in the second half of the first year, did not appear to prevent the growth retardation except at one community where a new nurse's influence in persuading all mothers to attend the communal feeding centre regularly led to a striking increase in weight-gain among the infants and children. The authors commented that, elsewhere, attendances at the communal feeding centres,

> tended to be irregular and often surprisingly low. Furthermore it is our impression that the Aboriginal women are often not at ease in these centres and make their visits as brief as possible, so that the quantities of food eaten by many of the children are probably much less than would be eaten by white children of the same ages.

It is not surprising that the most efficient method of preparing meals for large numbers (the 'communal feeding centre') did not prove to be effective in alleviating malnutrition if it disregarded human behavioural factors. As the authors state: 'Aboriginal attitudes to infant feeding and child management in general in the settlement situation have never been subjected to more than the most superficial exploration'.

The study mentions the high incidence of communicable disease and the generally poor domestic environment. Hookworm was stated to be present in 60–70 per cent of pre-school children in the northern part of the Northern Territory; diarrhoea, impetigo, boils, and upper respiratory tract infections were extremely common in infants and young children, and 27 per cent of those examined had discharging ears. Records from the communities surveyed showed that for the period 1960 to 1966 inclusive (seven years) the infant mortality rate had been 112·2 per 1,000 live births, with post-neonatal deaths accounting for 61 per cent of infant deaths (see p. 116n.). Deaths in the second year of life accounted for nearly a quarter of the total deaths in the first two years, and from Jacobs and Clements's figures it is possible to calculate a second-year mortality rate of 32·6 per 1,000 live births—a figure which is very close to that calculated for the Northern Division for 1965-6 (Table 6) of 30 per 1,000.

Jacobs and Clements concluded that the pattern was one of protein-calorie malnutrition caused by underfeeding and recurrent episodes of infection, particularly diarrhoea.

The studies reviewed so far were for full-blood Aboriginal communities in the Northern Territory, mixed full-blood and part-Aboriginal groups in Queensland, and full-blood and part-Aboriginal groups separately in Western Australia. In New South Wales, with its exclusively part-Aboriginal population, there are relatively few figures for growth rates. Data for heights and weights by age and sex for New South Wales North Coast reserves, collected about 1951 during a worm survey by Dr T.L. Dunn, showed that between the ages of 5 and 15 nearly every child's height and weight lay in the lower half of the distribution for New South Wales non-metropolitan white children, a few values being just below the mean minus two standard deviations. In a South Coast community studied in 1966-7 (Hausfeld and Moodie, 1967), the pattern was similar up until the age of about 8 years, although a proportion of heights and weights in this instance were just above the standard mean values for age and sex. From age 9 onwards, however, the values for heights and weights were evenly distributed across the standard range (plus or minus two standard deviations), with a small proportion of teenage girls showing abnormally high weights for age (they would be classed as obese). The pattern suggests that either the younger children had experienced a relatively greater degree of growth failure *since* about 1958, or that half the children from around the age of 9 onwards had been able to climb into higher 'growth channels' by way of improved nutrition or reduced infection. Neither explanation is particularly plausible. It is not generally held that children whose increase in height is persistently below average can achieve over-average heights through improved nutrition in the second half of childhood, although the same remarks do not hold for weight. There is no obvious reason for a deterioration in nutrition around 1958, nor any evidence that the prevalence or range of infections increased suddenly at this time. It is possible that children with very poor growth rates usually died in early childhood before 1958.

Edwards (1970) estimated that 31 per cent of part-Aboriginal children from Walgett, in north-western New South Wales, were growth retarded according to Jose's criteria. He also found about two-thirds to be below the 10th percentile for head circumference, possibly reflecting impaired brain development and impaired intellectual capacity.

In the Sydney metropolitan area, Lickiss (1970) found the following

proportions of Aboriginal and white children below the 10th percentiles for height and weight:

	Height		Weight	
Aboriginal pre-school children	20/32	62·5%	18/34	53%
Aboriginal schoolchildren	19/49	39%	15/37	41%
White primary schoolchildren	6/50	12%	6/43	14%

Lickiss's graphs for height and weight by age were similar to those for the South Coast community described above. Her data showed statistically significant associations[6] between failure to reach the 10th percentile values for growth and absence of a simple nuclear family, non-cohabiting parents, a 'problem-drinking' father, overcrowding, frequent changes of residence, and breast feeding for less than one month —all more or less predictable. Surprisingly, there was no significant association with low *per capita* income, mean *per capita* income, or mean number of children living.

Comparing the New South Wales findings with those in other States, it appears that growth failure (protein-calorie malnutrition) has been milder where it has occurred on the coast, but still sufficient to produce a noticeable growth deficit in the majority of young children. The New South Wales data described do not cover the important period from birth to 2 years, and some simple but extensive surveys of Aboriginal growth patterns in this age period should provide a more sensitive index of nutritional status and trends.

NUTRITIONAL DEFICIENCIES

General deficiencies in protein and calories are implied when a community is judged to be affected by growth retardation. A more specific calorie deficit has been implicated in Central Australia where lactose maldigestion and lactose-deficient breast-milk has been detected, but is unlikely to be confined to Central Australia.[7] The aim in this section is to examine other examples of specific nutritional deficiencies detected by clinical and biochemical survey.

The most recent detailed search for specific deficiency states has been made by Jose and his colleagues in Queensland (Jose and Welch, 1970), in the same community in which growth parameters were examined. In 600 growth retarded children, Jose diagnosed deficiencies in Vitamin A

[6] Statistical significance calculated for Lickiss's data, using χ^2.

[7] Harris *et al.* (1970) have reported lactase deficiency in fourteen out of nineteen intestinal biopsy specimens removed from hospitalised New South Wales Aboriginal children.

(3 per cent), Vitamin D (1·8 per cent), and Vitamin C (0·7 per cent) on clinical grounds, from evidence of skin and conjunctival changes, rickets and scurvy respectively. In a smaller group of forty-two growth-retarded children, there were low biochemical (plasma) values for albumin, folic acid, iron, carotene (Vitamin A precursor) and Vitamin C. Treatment of groups of deficient children with Vitamin C, Vitamin D, and iron by injection produced growth-spurts; the high doses of Vitamins C and D required to correct the biochemical deficit were seen as indicators of malabsorption of these vitamins from the intestinal tract. A group of infants aged 5 to 9 months followed for four months showed the onset of growth retardation in 61 per cent, and the retarded group 'showed significant decreases in serum iron and ascorbic acid levels before the onset of growth retardation'. The 'normal' 39 per cent were more often breast fed, and more often received supplementary solid foods by the age of two months. It is particularly significant that growth retardation was invariably precipitated by an episode of 'diarrhoea', but no intestinal parasites were detected until after the onset of growth retardation, when three infants showed *Giardia* infestations. Twenty-three out of twenty-seven infants who subsequently died from 'gastroenteritis' or 'pneumonia' showed abnormal weight loss for at least a month prior to death. The authors also found a significantly poorer primary school performance (in terms of average marks obtained in examinations) for children whose records showed growth retardation during infancy (although this result would be expected on environmental or 'home-atmosphere' grounds as well as on nutritional grounds—that is, growth retardation and poor school performance, while associated, may both be attributed independently to social and economic factors).

Jose and Welch reported that pregnant Aboriginal mothers had low plasma Vitamin C levels when tested in the last three months of pregnancy, suggesting that the newborn started with a disadvantage at least in terms of ascorbic acid stored before birth or obtained in breast-milk.[8]

The complex interrelationships between socio-economic circumstances, parental attitudes, the physical environment, infection, health behaviour (with respect to health and medical services) and nutrition are the bugbear of the investigator searching for prime causes of ill-health. In the

[8] Ascorbic acid (Vitamin C) is 'stored' in the body, but regular intake is required to maintain adequate normal growth. An analysis of breast-milk ascorbic acid from four Arnhem Land groups in 1948 (Hodges, 1960) showed generally low levels, although there was little correlation between ascorbic acid levels in breast-milk and the mother's blood.

field of nutrition, there are added difficulties in evaluating border-line biochemical and clinical findings, and in assaying body stores of vitamins.

Taking these difficulties into account, the studies in Queensland have been of high value in 'proving' the malnutrition story in that State, and identical or very similar patterns of nutritional disease may be assumed to occur in other States and in the Northern Territory—wherever Aborigines exist in similar circumstances. It is unfortunate that the detailed clinical, biochemical, and dietary studies carried out in 1948 during the American-Australian Scientific Expedition to Arnhem Land were not published until 1960, as the findings for three Arnhem Land missions foreshadow the findings in Queensland and the Northern Territory nearly twenty years later. In many ways the Arnhem Land study was a model interdisciplinary investigation, the team including a nutritionist, a physician, a dentist, a biochemist, social and physical anthropologists, and food analysts, again pointing the way to the ideal method of researching a socio-medical problem like malnutrition.

The team in Arnhem Land noted low birth weights, adequate growth early in infancy, and the fall away in growth rates from around 4 months onwards. Protein and calorie deficiency was suspected, but the authors were uncertain as to optimal growth rates for Aborigines and did not commit themselves in drawing comparisons between Aboriginal and white Australian growth rates. They noted anaemia probably due to iron deficiency,[9] and suspected Vitamin A and riboflavin deficiency in some cases. They found no clinical evidence of a deficiency of thiamine or ascorbic acid. The prevalence and range of infections was well documented. Biochemical deficiencies in ascorbic acid in plasma and breast-milk were noted in some communities. The biochemical testing was limited to that which could be carried out in the field with portable equipment.

In western New South Wales, Kalokerinos has observed a dramatic improvement in the condition of moribund Aboriginal infants and young children given large doses of ascorbic acid by intramuscular injection (Kalokerinos, 1969, 1971). Kalokerinos's observations, carried out without laboratory facilities or university or institutional support, are somewhat empirical, and some of his conclusions are unsupported by documented or tested evidence (nor were they advanced as anything more than hypotheses). Nevertheless, Kalokerinos's view that many of the

[9] This may have been due to folic acid deficiency.

clinical features found in desperately ill Aboriginal children are the result of 'acute ascorbic acid deficiency' precipitated by infection cannot be refuted even though cases of chronic ascorbic acid deficiency (scurvy) appear to be quite rare. The properties of ascorbic acid as a medicament (rather than as a vitamin) are suggested by its reported activities as a detoxicating agent and 'cell stimulant' in acute infections and heart failure (Martindale, 1941). The rapid disappearance of large amounts of ascorbic acid from the adrenal cortex is the basis of a method of assaying 'adrenocorticotrophic hormone', and the adrenal ascorbic acid 'stores' may thus play an important part in physiological reactions to severe acute stress. In view of Jose's clinical and biochemical evidence for ascorbic acid depletion and malabsorption in 'scurvy-free' malnourished Aboriginal children, Kalokerinos's theories deserve full investigation with support from the academic medical fraternity. Unfortunately, Kalokerinos's somewhat flamboyant and 'pioneering' approach to the subject, and his 'non-scientific' claims appear to alienate him from the support he needs and has asked for. These aspects in no way conceal what may be a most important 'discovery' or 're-discovery', with widespread applications in Aboriginal child health. Acute ascorbic acid deficiency, if it is a reality, cannot be regarded as the basic problem, nor parenteral ascorbic acid the answer—there is still the underlying socio-economic situation which fosters a wide range of deficiencies and other synergistic conditions. In this sense, the socio-economic 'answer' can solve the 'problem' before the problem is fully elucidated. It seems that the lack of knowledge of the social—not the biological—aspects of Aboriginal malnutrition is the main barrier to progress in this field.

Beri-beri, due to Vitamin B_1 (thiamine) deficiency has not been reported among Aborigines in recent decades.

The urinary thiamine/creatinine ratios estimated for 153 Aboriginal children from a New South Wales South Coast community in 1966-7 (Moodie and Galvin, unpublished) were corrected for height and weight and compared with a standard prepared for 'physically active young male adults'. On this basis, 22 per cent were classified as 'deficient', 55 per cent as 'low' and the remaining 23 per cent as 'acceptable'. The estimates were not made from 24-hour urine samples, and the interpretation is arguable because of this. Nevertheless, the results, taken overall, imply a low thiamine intake from the previous few meals. Most of the 'deficient' group were aged between 2 and 9 years.

Folic acid, another member of the B group of vitamins and an anti-anaemia factor, has been shown by Davis *et al.* (1965) to be at very low

levels in the blood of Aborigines from the Rawlinson Ranges of Western Australia, and by Jose and Welch (1970) to be at subnormal blood levels in Queensland Aboriginal children who were growth retarded. It is of interest that ascorbic acid is required to convert folic acid into a physiologically active form (Bell *et al.*, 1959). Another member of the group, Vitamin B_{12}, the anti-pernicious anaemia factor, has not been reported as deficient in Aborigines; Davis and Pitney (1957) found some abnormally high blood levels in Warburton Ranges Aborigines in Western Australia, which they attributed to increased levels of B_{12}-carrying protein in the blood.

'Tropical sprue', a malabsorption syndrome with fatty diarrhoea, megaloblastic anaemia, and wasting, of unknown aetiology but responsive to folic acid and therefore a disease which may be regarded tentatively as due to 'folic acid deficiency', has been reported by Douglas (1957) at relatively high prevalence among Aboriginal adults from missions and settlements on the east coast of north Queensland. It is not a common disease, but Douglas encountered ten cases among Aborigines as against three in whites during a period of three years. Douglas holds the view that infection may play a part in its 'cause' and attributes its relative frequency among Aborigines to their poor environment and high rate of intestinal infection. By 1963, Douglas had seen a further eighteen cases of 'tropical' sprue among Aborigines (R.A. Douglas, personal communication, 1966).

The only other deficiency of note which has been reported in the literature is calcium deficiency. The dietary calcium in Aboriginal communities in Western Australia and the Northern Territory was reported as deficient by Wilson (1951, 1953). There is little biochemical evidence of calcium deficiency in Jose's study in Queensland, however—serum levels in his 'growth retarded' group were just within the normal range.

ANAEMIA

The anaemia that is very common in Aboriginal populations may be regarded as a nutritional disorder, although it is not necessarily due to a dietary deficiency: failure to absorb or utilise specific anti-anaemia factors in the diet such as iron, protein or folic acid, or abnormal iron and protein losses such as may occur in heavy hookworm infestations are alternative mechanisms leading to deficiency. There is no evidence that Aborigines are affected by hereditary anaemias such as sickle-cell anaemia and thalassaemia, and tropical anaemias due to malaria or

kala-azar do not occur in the absence from Australia of the parasites responsible for these diseases.

The survey diagnosis of anaemia is based on a low level of haemoglobin in the blood, whether this is the result of insufficient haemoglobin in a normal number of red blood cells or of a reduced number of red cells. The criterion of abnormally low haemoglobin levels is subject to argument, however, and this creates some difficulty in sorting survey results into 'normal' and 'anaemic' groups. Results obtained from surveying a healthy population show a slow rise in the mean values for age from birth to adulthood, and a range of normal values above and below the mean which can be expressed in terms of the standard deviation or percentiles, as with height or weight. However, the 'low' normal values in early childhood can be raised to equivalence with the normal values of later childhood with supplementary iron included in the diet. It is not known whether infants whose haemoglobin levels have been artificially raised in this manner are at a health advantage when compared with their untreated but 'normal' contemporaries, although they would hold a temporary advantage if both groups were subsequently exposed to a deficient iron intake or an illness which decreased iron absorption or depressed the activity of blood-forming tissues. On this basis, it is a reasonable argument that Aboriginal children require a greater haemoglobin reserve than white children because of the additional dietary and communicable disease hazards that most of them must overcome during their growth to adulthood. Lovric *et al.* (1972) subscribe to the view that haemoglobin levels in children aged under 3 years must be below 10 grams per 100 millilitres of blood before morbidity results, although a mean of 12 grams or more can be produced with supplementary iron in healthy children. It has seemed reasonable, therefore, to accept a minimum 'safe' level of 11 grams for Aboriginal children up until the age where the standard mean-minus-two-standard-deviations limit climbs above 11 grams (around the age of 4 years), and thereafter use the latter value as the lower limit of normal.

Crotty (1958) investigated anaemia in Aboriginal children in the northern part of the Northern Territory, and showed that the mean haemoglobin values of 424 children were consistently at least 2 grams per 100 millilitres lower than standards based on New South Wales white children, at all ages from 6 months to 12 years. The deficiency was over 3 grams between the ages of 1 and 5 years, and the means were below 11 grams until the seventh year. Crotty observed no significant difference in mean haemoglobin levels between 155 children with hook-

worm and 129 children without hookworm, although he pointed out that this was no proof of the harmlessness of hookworm as worm load was not taken into account.

Crotty attributed the anaemia partly to 'lack of iron' and partly to protein deficiency, and he found clinical evidence (hair changes) suggestive of mild kwashiorkor in twenty-eight children studied in hospital.

Pitney (1962) found evidence of iron-deficiency anaemia (based on visible abnormalities in red cells examined microscopically) in 31 per cent of 187 Kimberley Aborigines of all ages over 10 years.

In Queensland, Jose and Welch (1970) found haemoglobin levels lower than 9 grams per 100 millilitres (a very strict criterion for anaemia) in 70 per cent of his growth-retarded group of Aboriginal children, as against a figure of 24 per cent for those 'normal' for height, or 33 per cent for those 'normal' for weight. Overall, no less than 47 per cent of 705 children had haemoglobin levels below 9 grams. On Mornington Island, 30 per cent of children aged between 1 and 3 years had haemoglobin levels below 7 grams. The authors noted that serum folate levels were also very low on Mornington Island, but did not state whether the anaemia was primarily macrocytic in type.

In metropolitan Sydney, Lickiss (1970) observed only one part-Aboriginal child out of forty-five aged 2 to 15 years who had a haemoglobin level below 11 grams per 100 millilitres, and only three blood films out of ninety-four showed more than slight hypochromia characteristic of iron-deficiency. In a New South Wales coastal rural-urban community, however, Hausfeld and Moodie (1967) reported that 35 per cent of children were anaemic (haemoglobin levels more than two standard deviations below the standard mean), and found that the prevalence of anaemia was lowest in the fringe-dwellers and highest at the Aboriginal settlements—an unexpected result. Data obtained by Dr T.L. Dunn during worm surveys in New South Wales North Coast Aboriginal settlements and reserves, in 1951, placed 70 per cent of children aged 5 to 16 years in the anaemic category. In a group of Aboriginal boys resident at a North Coast Aboriginal 'Boys' Home' only one of thirty was anaemic. Inmates at this home came from 'problem families' in New South Wales at various ages from about the age of 4 to 5 onwards, and many remained for long periods. It is of interest that whereas all but one had 'normal' haemoglobin values when surveyed by Dunn, they showed the same pattern of growth retardation as their contemporaries on North Coast settlements and reserves—an indication that improved diet and regular 'deworming', and possibly supplemental iron

prevented anaemia but did not restore lost growth commencing in early childhood.

It would seem from the figures available that anaemia in Aboriginal children, and possibly adults as well, is from ten to twenty times as common as in the white population, and is more often severe. In addition, lower mean values for Aboriginal groups imply that their haemoglobin 'reserves' against added nutritional or infectious stress are lower when they need to be higher than for white Australians as a whole. The impact of this widespread anaemia on general health, levels of physical activity and school performance, and capacity to survive serious infections in childhood must be enormous. The relative causal importance of dietary deficiency, malabsorption and associated biochemical abnormalities is conjectural, but all appear to play a part. 'Iron-deficiency' anaemia is probably the most important single disease entity in terms of numbers affected and in terms of overall effects on Aboriginal health. But, like the other specific deficiency diseases, it is only a part of the pattern of malnutrition and symptomatic of underlying socio-economic stresses, and it would be illogical to counter it with mass iron administration except as a short-term palliative measure.

In common with most other diseases in the Aboriginal population, the detection of the malnutrition problem does not in itself suggest the solution *in the specific context of Aboriginal society*. Finding the answer is a task in which the behavioural sciences must play the major part.

11 MENTAL HEALTH

The psychogenic disorders—the psychoses, psychoneuroses, psychosomatic disturbances and personality disorders—are negative aspects of 'mental health' which may be observed in a population and, if the criteria of diagnosis were unequivocal, might provide some measure of psychiatric morbidity which could be compared between populations. To obtain a complete picture of mental health, it would be necessary to include the effects of organic brain damage, intellectual handicapping due to hereditary and acquired causes, and psychological handicaps which may not easily be classifiable as psychiatric disorders and which may not be 'apparent' except to the trained outside observer.

A review of the whole field of mental health in the Aboriginal population is quite beyond the scope of the present study. Furthermore, it is evident that the diagnosis of many aspects of mental ill-health in a cross-cultural situation is so subjective as to make comparisons invalid; this applies nearly as much to the more clear-cut psychiatric syndromes as to the personality disorders, intelligence, and attitudes which underlie Aboriginal behaviour.

Probably in no health field other than mental health is the 'normal' range of attitudes and behaviour so determined by the 'abnormal' extremes. It is as if the extreme forms of behaviour acted like the yellow posts along each side of a roadway under deep snow—the middle-of-the-road 'normal' pathway cannot be located without them. If the signposts in other cultures or subcultures are placed differently, their 'normals' will also be different. It is not very difficult to argue that the eccentrics, extremists and possibly the psychoneurotics and psychotics are necessary

factors in social evolution and adaptation in the sense that experiments are necessary to demonstrate viable alternatives. The writer would subscribe to a sort of Darwinian theory of social evolution in which the 'abnormal' individuals may be regarded as mutants, while 'normal' values and beliefs, like genes, are generally acquired from one's parents and are restricted to those available from the community 'pool'. Most of the 'mutants' will be unsuccessful, but from among them will appear a few who contribute to the society in a lasting way, helping it to adapt to changing circumstances.

There are two important corollaries to this theory; firstly, it is both 'normal' and 'healthy' for a community to contain individuals who might be regarded individually as outside the normal range in terms of mental health; secondly, an Aboriginal who, for instance, is typically middle-class 'white' in his attitudes and behaviour is *ipso facto* 'abnormal'—an Aboriginal extremist.

In looking at the pattern of mental health in Aboriginal communities, the tendency has been to regard Aboriginal stereotyped behaviour patterns such as heavy drinking, 'apathy', Elkin's 'intelligent parasitism' or Christophers' 'institutional neurosis' as symptomatic of mental ill-health rather than as suitable adaptive mechanisms integrated with other more positive Aboriginal characteristics which do not draw attention. All this implies that it is extremely difficult, if not impossible, to reach valid conclusions with respect to Aboriginal mental health except, perhaps, in terms of 'assimilability' and white norms.

In looking at the incidence or prevalence of the more severe disorders in the psychiatric field, particularly the psychoses, the grounds for passing an opinion are firmer, and it can be assumed that the communities in which Aboriginal psychotics are found are fully aware of such individual's abnormal or extreme behaviour (while not necessarily being much perturbed by it).

Diagnosable mental illnesses and psychological handicaps in an Aboriginal community, particularly those which can be attributed to external stress, serve as signposts to factors in the social and psychological environment which may not be apparent from anthropological or sociological studies. However, inferences as to 'causes' of mental ill-health are bedevilled by complexities which are not present with most of the physical diseases—particularly the infective and nutritional disorders common among Aborigines, where at least the disease-producing agent is defined. Whereas it may be accepted that certain psychoses, neuroses, or personality disorders are produced by stresses, or by 'conditioning' to

stress-situations in the individual's past, it is by no means clear what the real stress is (or was). There are several schools of psychiatric thought with opposing views of causality in mental illness, upon which the writer is not competent to pass an opinion. It seems fairly clear, however, that most would accept that it is the net effect of the degree of stress and the individual's competence and conditioning to deal with the stress which determines the outcome. The simplistic view that all the mental ills of Aborigines are the outcome of the Aboriginal-white contact situation, for instance, ignores the contribution of Aboriginal (or part-Aboriginal) social conditioning in predetermining how the individual will react to this and many alternative situations he may otherwise meet in life.

This is not intended as an apologia for past and present white behaviour towards Aborigines, nor to dismiss the importance of white behaviour in creating deep-seated Aboriginal psychological handicaps which could have been avoided. It is a statement of the writer's conviction that Aborigines, as a group or as a society, must take positive steps themselves to reduce the stresses they find in the contact situation. The techniques needed to alleviate such stresses may well lie more appropriately in the fields of art and philosophy and other subtle forms of image-building rather than in direct social manipulation. It is unfortunate in more ways than one that the boosting of 'Aboriginality' as a counter to deculturation tends to conflict with policies of assimilation or integration as expressed by governments in the past. Such policies are still proclaimed by some to be the opposite of the apartheid approach. The real opposite to apartheid would seem to be freedom to choose, and this implies the removal of most (but not all) pressures upon Aborigines to conform to white Australian values. Even here there is no real escape, since freedom to choose is itself a cause of stress in those ill-equipped to make the choice, particularly for a choice which has such wide repercussions.

It has been noted earlier that the Aboriginal mortality data indicate a low suicide rate and a high homicide rate, when compared with Australia. It is difficult to interpret these findings in terms of mental health without knowledge of the prevalence of depressive and aggressive states in the Aboriginal population. A broad study of Aboriginal psychiatric status has been pursued by Cawte and his colleagues at the University of New South Wales, and this source provides the bulk of the psychiatric knowledge referred to below.

Cawte divides mental illness in the Aboriginal population into 'traditional' and 'transitional' groups, and has looked for evidence of the latter in records of Aborigines in mental hospitals (Cawte, 1965). Figures were

available from Western Australia and South Australia, during the ten-year period 1954 to 1963. Noting the difficulties imposed by the selection of cases and non-uniform diagnostic criteria, Cawte concluded that long-term severe psychiatric illnesses were more common in the rural Aboriginal population than in the white population; that city Aborigines appeared in the figures of the mental health services in a smaller proportion than expected from the proportion of Aborigines in the cities. The most commonly diagnosed clinical disorder was schizophrenia (41 per cent of cases), followed by mental deficiency (19 per cent), and manic-depressive psychosis (6 per cent). 'Endogenous depression is named only once' (0·5 per cent). Alcoholism was noted to be 'not rare', the figures showing it to be mentioned in the diagnosis of 14 per cent of long-term cases. The author noted that Reception House facilities (for acute illnesses) were less used than by the white population in proportion to the numbers of each, without implying that Aborigines are less prone to short-term mental illness.

With regard to depression, Cawte noted in an earlier study at Kalumburu Mission in the North Kimberley district of Western Australia (Cawte, 1964a:180):

> The endogenous variety of depression, with retardation and feelings of guilt, is very rare. More common is a degree of agitated misery, with blame transferred outside the person, usually to sorcery or sickness. The comparative absence of guilt feelings is in striking contrast with a Western population. Suicide is almost unheard of.

In the same population, Cawte (1964b) reported that no distinctive psychiatric syndromes were found in the Aboriginal population, although there were distinctive, culturally-regulated patterns of behaviour which influenced the symptoms of mental illness. He noted the rarity of acute and chronic brain disorders, the absence of alcoholism (in this mission environment), but thought that cerebral syphilis may have been an important cause of psychosis and death in the recent past. He found paranoid psychosis common, schizophrenia occurring but liable to be simulated by sorcery-beliefs, and manic-depressive psychosis exceedingly rare.

Neuroses presenting as phobias and hypochondria were stated to be 'widespread', together with personality disorders classified as passive-dependent, passive-aggressive and paranoid. Psychosomatic disorders were rare, and the author comments on the lack of support for the idea that past Aboriginal infertility was psychosomatic in origin.

Across the other side of north Australia, Cawte (1969) carried out a 'psychiatric morbidity census' of 276 adult Aborigines and 280 children

from Mornington Island in the Gulf of Carpentaria—again a full-blood community like that at Kalumburu. Among the adults he noted a psychiatric abnormality in 11·6 per cent, the most common being 'personality disorder' (4 per cent), depression (2·5 per cent) and neurosis (1·4 per cent). Among children, 15 per cent exhibited some psychiatric abnormality, particularly the 'tension-discharge' syndrome (about 4 per cent) and the 'anxious-inhibition' syndrome (around 3 per cent). The author noted that there was more mental illness among a group of Kaiadilt Aborigines who had been brought from nearby Bentinck Island in 1948 following a natural disaster, and who were more disintegrated socially and to some extent were an enclave in a hostile 'foreign land'; it was the Kaiadilt children who contributed most of the anxious-inhibition syndromes.

Cawte saw a 'vicious spiral' of social disintegration and mental illness in the Kaiadilt group, claiming that the abnormal behaviour and hypochondria of adults disrupted community life, and the adult depressives and the anxious-inhibition syndrome in children affected 'social cohesion and mastery', and that the childhood anxious-inhibition syndrome was 'related to the emotional climate of upbringing' in this group. There is a suggestion that the spiral of disintegration was operating before the population was contacted or transferred to Mornington Island (due to severe environmental stresses, recurrent famines, killings and vendettas) and was not simply the result of the stresses of transplantation into another locality and Aboriginal culture.

In the Walbiri tribe in Central Australia, Cawte and Kidson (1965) and Kidson (1967), recorded psychiatric disorders among thirty members in a Walbiri lineage—three generations—and observed different patterns in each generation: in the older group, aggressive personalities and paranoid states; in the middle group, organic disease was primarily responsible for mental disorders; in the younger group, a 'tendency towards schizophrenic disorders'. The authors concluded that there was an evolution of character and character disorders taking place as a result of the contact situation and a changing ecology. The overall prevalence of a history of psychiatric illness was 5·4 per cent.

Kidson and Jones (1968) studied the psychiatric disorders among Aborigines of the Western Desert and found functional psychoses, personality disorders, organic psychiatric syndromes and 'possession' but no dominant neuroses, psychosomatic illness or suicide.

Cawte and others have written extensively on many aspects of Aboriginal mental health, but a great deal of the writings is descriptive, and

cannot be summarised with any justice to their content. The following is so relevant to mental and general health in the contact situation that it has been extracted in full: (Cawte, 1969b).

Because good rapport is usually lacking, Europeans who contact Aborigines fail to perceive the extent of their problems and fail to appreciate that Aborigines in general do not regard themselves as fit and healthy. Aborigines communicate their complaints in a stereotyped fashion that invites under-diagnosis. A study in northern Queensland that went to the very considerable trouble of administering (in the vernacular) a modified Cornell Medical Inventory Health Questionnaire to a tribally oriented population produced striking results . . . There were five high peaks of complaints, concerned respectively with musculoskeletal pains such as backaches, respiratory troubles, insomnia, fatigue and anger. Musculoskeletal complaints were no surprise: possibly the Aborigines' build is better evolved for long distance walking than for labour with heavy objects. Respiratory complaints were predictable from the atmospheric conditions of high dust and smoke levels and extreme diurnal temperature variations. The insomnia—surprising to observers who see Aborigines asleep during the day—might reflect a crowded and disturbed sleeping environment and in many cases poor health, anxiety or depression. Fatigue came as no surprise since it was known that this population is subject to hookworm infestation and to dietary uncertainty. Suppressed anger was understandable in a people little experienced in high density group living.

What is perhaps surprising is the remarkable consistency of the complaint pattern in all the groups though the highest levels of complaints are found in the older age groups, the women, and the more socially disadvantaged people.

In outback and fringe Aborigines, a set of symptoms not adequately recorded by questionnaire methods, and not complained of by the subjects themselves, is the tendency to alcoholism and gambling. These tension-reducing devices give rise to further difficulties that contribute to the downward spiral of malfunctioning. Where alcohol is not available in more protected Aboriginal communities, petrol sniffing may be resorted to by stimulus-hungry youths . . . Although alcoholism is found in a number of Aboriginal people it is open to question whether it is more prevalent than in European Australians. What is clear is that Aborigines are less protected against its severe effects by their economy, by their social network and by the availability of medical services. In the crowded environment of slum, fringe or settlement even a small number of alcoholics has a disintegrative effect on the community in general.

Indeed of all the changes enforced upon Aborigines by the culture contact, perhaps none is more disruprive than high density camp life, . . . Many of the traits attributed to Aborigines may in fact be artifacts of camp life. Camps erupt in periodic brawls that often become riots involving most of the community until the police intervene to carry off combatants to the lock-up. That these riots are not merely due to drunkenness is obvious because they also occur on 'dry' missions and settlements. There they are put down to disputes over women. Whatever their origins, the riots are as disastrous for harmony in the camp as they are for the blackfellow's social image before the whitefellow.

. . . Characteristically the spark that ignites the riot may be a child. A child beats another, the defeated child's mother protests to the victor's and receives a blow with

the fist, the husband retaliates with a stick, the relations and inlaws join in. Coalitions crystallize around old grievances ... Equally characteristically, between riots the ethos of the community is one of tolerance and—especially—of evasion of confrontation. The Aboriginal 'ideal man' is restrained, generous, a peacemaker who never confronts another with his grievances until these become serious. Thus he becomes the victim of the carping wife, the dependent relation, the forgetful borrower and the stealthy manipulator. Europeans see Aborigines as indecisive and lacking initiative. Observations of riots suggest that this is related to evasion of confrontation and to the necessity to consult group opinion rather than take an individual stand and so run the risk of group conflict. It is an ethos that puts Aborigines at a disadvantage with Europeans, who expect communication of a more direct kind, with irritability expressed but controlled.

Cawte's conclusions are based on extensive observation and logical analysis. They provide a telling summary of many important factors in Aboriginal health and health-related behaviour. Although he speaks mainly of full-blood communities, what he says may be applied to part-Aboriginal communities in southern Australia if the 'feud' is substituted for the 'riot', and if a prolonged conditioning to high-density group living is substituted for 'little experience' of this type of existence. Uncontrolled aggressive behaviour—the 'explosion' of violence—may be seen as the principal 'cause' of the high Aboriginal homicide rate. The low suicide rate may be attributed to the low incidence of depression, and this in turn may be seen as being the result of a number of interrelated factors—the externalisation of causes of personal difficulties and failures; the lack of opportunities to withdraw and brood; the internal and external limits imposed on the achievement of high status so that a fall from status is not a long drop; and the involuntary 'group therapy' through close personal involvement in other people's problems and crises.

Nurcombe (1970) has written on psycho-social and socio-cultural deprivation with particular reference to rural-urban fringe Aborigines. He finds the fringe-dwellers suffering from 'the parapsychiatric disorders of alcoholism, petty crime, unstable impulsiveness and chronic obsession with physical (organic or psychosomatic) complaints', replacing the fears of sorcery, invasion by malevolent objects, possession and 'spirit-loss' exhibited by the partly detribalised Aborigines. But Nurcombe states that 'depression is common, especially among women. Depression ... may be masked by alcoholism, restless impulsiveness, apathetic resignation or chronic physical preoccupation'. The writer would argue with the definition of 'depression' implied in this last statement, attributed to Ross.

The field of 'parapsychiatric disorders', deviant behaviour in general,

and 'deviant' Aboriginal values and attitudes is of great relevance to Aboriginal mental and general health, but is of such complexity as to place it outside the scope of this study. Without entering into the polemics of causality, and the mechanisms whereby such disorders arise, one may impute the following important factors: the socio-cultural conditioning of Aborigines and part-Aborigines; the stresses of the contact situation (and the socio-cultural conditioning of white Australians); and the short- and long-term effects of physical disease. With regard to the latter, intellectual handicapping resulting from malnutrition and from infection —particularly infections of the brain but also from the toxic and biochemical effects of other infections—remains to be proved in the Aboriginal population, but the opportunities for the occurrence of intellectual handicapping due to physical causes appears to be sufficiently widespread to relegate 'racial' or genetic causes to a very minor place in the hierarchy of investigations needed. The current tendency to attribute poor Aboriginal performance in psychometric tests to defects in the test instrument may be hindering the identification of preventable physical causes for intellectual impairment.

Nurcombe (1970) believes that, in the part-Aboriginal situation, physical factors such as malnutrition are accompanied by the effects of psychosocial and socio-cultural deprivation to produce a cycle of intellectual 'retardation' which tends to be self-perpetuating. The reciprocal effects of mental and physical ill-health upon each other add great weight to the need for controlling and eradicating physical disease, especially infection and malnutrition, in the Aboriginal population as an important contribution to mental health. A more direct effect on Aboriginal mental health can be expected with the removal of any remaining discriminatory practices, including any practices in the field of health which single out Aborigines in a mixed community for special attention and which involve some personal distress or which necessitate hounding by the health authorities. In a mixed community, it can be seen as psychologically unfortunate that the physical diseases of greatest significance to Aboriginal health are of minor importance to the health of Australian whites of the same age-groups. Public health measures, therefore, have to be applied with the greatest tact and social skill if they are not to be rejected as discriminatory. It would appear to be unquestionable that efforts to improve the physical health of Aborigines must not add to their already heavy psychological burden, particularly because psychological handicaps are transmissible from one generation to the next.

To summarise the picture of psychiatric morbidity in the Aboriginal

population, the evidence available is that grave psychiatric disturbance—'insanity' in general terms—may be a little more prevalent than in the white population, but the distribution of syndromes favours schizophrenia and paranoia and disfavours depression in the Aboriginal society. The psychoneuroses manifest more as phobias, compulsions and hypochondria, and are said to be 'common'. Anxiety and anxiety-depression as manifested in white communities do not feature very much in the Aboriginal population. Personality disorders, and what have been termed 'para-psychiatric disorders' including alcoholism, apathy, restlessness and petty criminality, appear to be a major but as yet unmeasured component of Aboriginal mental ill-health. There is extensive but not particularly conclusive evidence for intellectual impairment (the results of psychometric testing are not reviewed in this study) which is likely to be contributed to by physical disease.

12 DISEASES OF THE NERVOUS SYSTEM AND SENSE ORGANS

Communicable infections of the nervous system and sense organs, and the likely contribution of malnutrition to intellectual impairment, have been referred to earlier. Psychiatric disorders, which are in a sense diseases of the nervous system, have been covered in the previous chapter. Cerebrovascular accidents ('strokes'), brain damage at birth, and brain damage resulting from injury are involved in the categories 'cardiovascular disease', 'neonatal disorders' and 'accidents'. Although there are a number of organic disorders of the central and peripheral nervous system remaining, few are documented in the data on Aboriginal health. Non-contagious diseases of the ear and eye occupy most of the space devoted to 'diseases of the nervous system and sense organs', and the question arises as to whether this is by default. Is organic non-contagious disease of the central or peripheral nervous system exceptionally rare in Aborigines, or is it under-diagnosed or under-reported?

In the writer's opinion, and from brief mentions in the literature, these diseases do occur in the Aboriginal population, but are not reported for several reasons: they are relatively rare in any population and usually occur in adults, so that doctors in contact with small Aboriginal communities will not see many cases; extensive neurological surveys, if carried out, have not been reported; and many of these diseases are insidious in onset and vague in their symptomatology, and may not be appreciated as a 'disease' by poorly-educated Aboriginal adults or indeed by many of those involved in the day-to-day medical care of sick Aborigines.

Thus the apparent absence of hereditary and familial diseases (such as

muscular dystrophy), and multiple sclerosis, paralysis agitans (Parkinson's disease), migraine, motor neurone disease and various cranial nerve disorders—to name a few 'missing' diseases—is unlikely to have the epidemiological or genetic significance it might appear to have. Such diseases attract more attention in the white population partly because of the different age-composition, but mostly because symptoms are more likely to lead to specialist investigation, diagnosis, and treatment. The same could probably be said for other diseases of insidious onset in adult life—a low prevalence in the Aboriginal population is more likely to be a socio-economic-diagnostic phenomenon, rather than an epidemiologic reality.

Two rare hereditary neurological diseases have been reported in the medical literature in recent years. Gollan *et al.* (1970) recorded the only case of hepatolenticular degeneration in a full-blood Aboriginal (from Wave Hill, Northern Territory). This disease is associated with a recessively-inherited disorder of copper metabolism,[1] and it is of no significance in the overall pattern of Aboriginal morbidity because of its rarity. In South Australia, Gale and Bennett (1969) have recorded the occurrence of a series of cases of Huntingdon's chorea in part-Aborigines originating from the Point McLeay area near the mouth of the Murray River. Huntingdon's chorea is a dominantly-inherited degenerative disorder of the central nervous system which usually appears late—during or after reproductive life—a fact which encourages its spread through successive generations although it is a progressive and eventually fatal condition. The authors do not state the incidence of manifest Huntingdon's chorea in the population studied, but from the genealogy ('pedigree') reproduced in their paper it appears that there are nine affected adults in approximately ninety-five living members of the affected lineage, and at least twenty-three who have probably inherited the disease but not yet developed signs. The authors point out that the large families and wide-spread emigration of Point McLeay Aborigines to other areas, coupled with sexual promiscuity and the late appearance of signs and symptoms, is encouraging the spread of this serious disorder throughout South Australia, Victoria, and New South Wales.

Some of the genetic implications of Aboriginal ecology relevant to all hereditary disease and abnormality are covered in Appendix II.

Epilepsy is another disease of the central nervous system which is

[1] The International Classification of Diseases places hepatolenticular degeneration among the metabolic diseases, but its predominantly neurological manifestations (tremors and rigidity) warrant its consideration here.

often familial, particularly in those cases who develop the disease early in life (in the absence of external causes such as head injury or organic brain disease). Cases have been recorded or mentioned in passing among many Aboriginal and part-Aboriginal populations, but there is no information on prevalence or incidence among large groupings. It is not known whether it is more or less common in the Aboriginal than in the white population, but where it does occur it will pose special problems for Aborigines because of the need for prolonged, regular medication to prevent attacks. Repeated fits lead to varying degrees of intellectual impairment and emotional changes, so that uncontrolled epileptics frequently end up in institutions. Cawte (1969) mentions one case of epilepsy with behaviour disorder in his survey of Mornington Island Aborigines, but records ten cases among Aborigines in Western Australian and South Australian mental hospitals—5 per cent of the group studied.

DISEASES OF THE EYE

The major cause of eye disease in the Aboriginal population is trachoma, mentioned in the chapter on infective and parasitic diseases. In the course of surveys for trachoma, other eye diseases have been noted and prevalence figures calculated. In desert Aborigines in the Warburton Range area of Western Australia, Mann (1957) observed that ocular health was good apart from the high prevalence of trachoma, and diseases common in the white population, such as styes and other lid infections, squint, mucopurulent conjunctivitis, glaucoma and colour blindness were not seen at all.

In the Kimberleys, Mann (1954a) noted a low prevalence of colour blindness (2·3 per cent) compared with whites (12·1 per cent)—both these figures referring to males only.[2] Mann offers no explanation for this marked difference. Even in the harsh environment of the desert, where normal colour vision may have been important in the search for food, it is not easy to see how selection or assortative mating could be a major factor (see Appendix II), although with thousands of years over which to operate, even the most minimal influence can exert very strong effects.

[2] Colour blindness is a sex-linked recessive inherited disorder. It can occur in females only when they are homozygous for the condition, whereas all male offspring of heterozygous 'carrier' females are affected. It is therefore usual to report the prevalence in males only. Affected males cannot pass it on to their sons, but their daughters will all be carriers, and if the mothers are also carriers, half the daughters of colour blind males will be colour blind homozygates.

In the rather kinder environment of the Kimberleys, such pressures on the gene for colour blindness seem less likely. If selection or assortative mating have been responsible, an increase in colour blindness would be anticipated under modern Aboriginal conditions, but the minor handicap under the new conditions will decrease in importance to the individual.

It is of interest that Mann, in her Kimberley survey, found colour perception to be particularly acute in so far as many Aborigines tested traced not only the 'normal' figures on the colour-perception charts (Ishihara plates) but also the superimposed figures which are usually perceived only by those with colour-blindness. There are explanations for this connected with literacy and powers of observation—the literate person tending to jump to conclusions when presented with a familiar pattern such as an Arabic numeral—but, as Mann points out, most of the Aborigines were familiar with the numerals because they are widely used in cattle-brands.

Although the findings with respect to colour blindness have little relevance to a morbidity study in themselves, they serve as a reminder that isolation in relatively small groups and the associated 'inbreeding' are not necessarily harmful to the genetic constitution of a population given time to reach stability. Very few other hereditary or congenital ocular abnormalities were noted in full-blood Aborigines. However, if positive aspects are to be found in the full-blood Aboriginal genetic constitution, an inescapable corollary is that negative aspects also exist, at least under conditions of 'new' stresses, or 'new' diseases. The latter is an old and somewhat tired argument, and currently unpopular with those who feel it implies 'racial inferiority' for Aborigines. It implies no such thing, of course, since inheritance is no more fixed than the stresses which influence it.

The only eye diseases in the Aboriginal group in the Kimberleys (other than trachoma) which occurred in significant numbers were pterygium (nearly always mild) and cataract. Neither were seen by Mann to constitute an eye-health problem in terms of numbers. Pterygium was less than one-tenth as common as trachoma, and cataract relatively rare.

Almost identical findings were made by Mann in the South-West (1956) and the Eastern Goldfields (1954b), with respect to the prevalence and relative importance of non-trachomatous eye diseases in Aborigines.

In all her surveys of Aborigines in Western Australia, Mann found no ocular evidence for a deficiency of vitamins A, B_1, B_2, or C.

Binocular visual impairment was nearly always due to the effects of trachoma, and monocular blindness to the results of injury. In the Kim-

berleys, Mann noted fifty-one cases of ocular leprosy, which corresponds with the high prevalence of leprosy in that area.

Defective vision due to refractive error was rare in full-blood Aboriginal groups, and Mann reports the incidence of myopia as being nil, compared to 4·8 per cent in white Australians (1961:271).

The prevalence or incidence of eye diseases and visual disturbance in part-Aboriginal communities is not well documented. A survey of part-Aboriginal schoolchildren from a New South Wales coastal community (Hausfeld and Moodie, 1967) showed six out of 136 children to have a visual defect (visual acuity 6/9 or worse)—a prevalence of 4·4 per cent. An additional 1·4 per cent had a loss of acuity in one eye only. Six per cent of male children were colour blind. These figures are not remarkable in any way.

In general, the ocular health and visual acuity of Aborigines does not suggest the existence of any particular handicap to schooling or employment, and if trachoma was eradicated the outback Aborigines would probably have better eye health than white Australians.

DISEASES OF THE EAR

The most important contribution to Aboriginal ill-health in this area arises from chronic middle ear infection—a common cause of deafness, and a common cause of temporary incapacity of varying degree when the condition flares up at intervals, as is usual. Although it is a common condition with permanent sequelae in a number of cases, it has not received much attention from otologists in Australia as an Aboriginal health problem. The underlying causal mechanism is the subject of some controversy, and preventive measures have not been generally agreed upon. Aborigines do not have the same access to skilled otological diagnosis and treatment as white Australians—for demographic and economic reasons—and appear to 'live with' this disease to the extent that, in many instances, no treatment at all is sought when a child develops an earache and subsequently a discharging ear when the ear-drum perforates.

Acute middle ear infections are usually diseases of childhood, and otologists point out that they were common in white children before the antibiotic era in medicine. Since the advent of antibiotics, a 'new' disease has risen to prominence among white children—the so-called 'glue-ear' in which the middle ear becomes filled with a sticky mucous exudate resulting from a low-grade or spontaneously regressing infection; spontaneous perforation does not occur; and surgical drainage is fre-

quently necessary to prevent hearing loss. There may be a number of factors operating together to bring 'glue-ear' disease to prominence—the frequent use of antibiotics, the reduction in relative importance of the acute suppurative infections, changes in the age-distribution of first attacks and changes in the general standards of health and immunity of white children. The pattern in Aboriginal children seems to remain that seen in the white population half a century or more ago.

Clements (1968) carried out an otological survey of 119 Aborigines at Allawah Grove settlement in metropolitan Perth, Western Australia, including audiometry. He found 23 per cent of 84 children under 15 with evidence of past or present middle ear disease, of whom half had active infection (chronic otitis media) at the time of examination. Among 59 children over 5 years, and 13 adults, some hearing handicap was present in 21 per cent, and a severe handicap in 7 per cent. The author compares these findings with those in Maoris (10 per cent with past or present middle ear disease), private schoolchildren in Perth aged 14-18 (5 per cent with evidence of past middle ear disease, none with active middle ear disease), and about 5 per cent of South Australian school-age children failing a simple screening test for hearing—many of these being found 'normal' with more sophisticated testing.

Clements noted that the Aboriginal children had 'a different sort of acute otitis media', with a minority going through the usual stages of pain, discharge through a small perforation and healing. More common, especially in children who had had a debilitating illness such as measles or gastroenteritis, was a relatively painless infection with sloughing of a large central area of the ear-drum. This fails to heal for many years, and a constant or recurring mucoid discharge follows with purulent exacerbations accompanying upper respiratory infections. He remarks that this type of perforation is relatively 'safe' in that it rarely causes intracranial complications (i.e. mastoiditis or abscess), but can result in severe deafness.

Clements attributes the high prevalence and early onset of necrotising otitis media in the Aboriginal children to poor nutrition and general health, and measles in infancy.

In a survey of 231 children from a New South Wales coastal population (Hausfeld and Moodie, 1967), 12 per cent of children had open perforations of one or both eardrums (5 per cent had both), and an additional 9 per cent had healed perforations. The total who had evidence of past or present middle ear disease was 21 per cent. These figures are nearly identical with those reported by Clements, and the large central perforations were also typical in the New South Wales group.

The high prevalence of middle ear disease and deafness is stated in almost every report on Aboriginal health, from every part of Australia. When noted—particularly during surveys—the disease is usually in the chronic stage, and it is evident that the first attacks commonly occur early in life, often in the first year. Those who escape are still subject to first attacks at any time up to early adult life. Whether or not the first attack is accompanied with pain before the perforation occurs, the occurrence of first attacks in infancy in a setting where medical advice is often not sought for crying infants—particularly when accompanied by an apparently minor respiratory infection—explains the frequency of perforation and the onset of the chronic phase and complications such as deafness. The writer has seen small part-Aboriginal children during surveys who have an acute otitis media unbeknown to their parents—even when the mother has been asked before the examination whether the child had had an earache 'at any time in the past four weeks'. Do Aboriginal children with earache complain less, or are their complaints ignored, or if not ignored, are their complaints not acted upon? Some Aboriginal mothers questioned by the writer have had sufficient experience of 'ear trouble' in their children to know that an earache usually ceases when the ear discharges (i.e. when the ear-drum eventually perforates) and seem to regard the appearance of the discharge with a similar view to that of the barber-surgeons to 'laudable pus'. But some of the children with red, bulging ear-drums were not distressed by earache, and others were too young to indicate a painful ear, although they were feverish and fretful to a degree which would have led the 'average' white mother to seek medical advice. Chronic suppurative otitis media seems to be favoured, therefore, by early age of onset of the acute infection, by a lack of awareness on the part of the Aboriginal mothers as to the significance and eventual sequel to untreated earache, and by the high background level of infection in early infancy. As Clements has suggested, the rapid, relatively painless course in many cases may be a consequence of poor nutrition and poor general health, presumably mediated through an effect upon the interaction between inflammation and immunity.

Maxwell (1969), summing up a seminar on Aboriginal health, stated that up to a third of Aboriginal school-age children may suffer deafness from this disease, and pointed out the gross handicap to education and educability.

The United States Bureau of Indian Health made otitis media a notifiable disease in American Indians in 1961. In 1962, the figures showed that otitis media was the second most common notifiable disease (annual

incidence rate 4 per cent for all ages, with over 60 per cent of cases occurring in those aged under 5 years, and 24 per cent of cases occurring in the first year of life). Gastroenteritis was slightly more common, with an incidence of 5 per cent per annum (*Indian Health Highlights*, 1964).

Phillips-Turner (1965) estimated the annual incidence of 'chronic suppurative otitis media' among Maoris to be up to 10 per cent in some areas. Aboriginal figures are not likely to be lower than those for American Indians or Maoris.

There is an apparent need for health education and related measures to combat otitis media and deafness among Aborigines, but there is a prior need for careful investigation and identification of common causal factors and mechanisms. Poor nasal toilet and unsatisfactory techniques of infant feeding may play a part, but unless it can be shown that changes in these aspects of behaviour reduce the incidence of otitis media materially they cannot be offered to the Aboriginal people as the solution. Early diagnosis and treatment of the inflamed middle ear is the obvious temporary solution, and this means that every sick Aboriginal infant should have an auroscopic examination regardless of other symptoms and 'obvious' diagnoses. This in turn means training medical auxiliaries at the lowest level to carry out this relatively simple procedure.

There is a danger that early treatment by non-specialists in the field of otology may convert the perforation and deafness problem into a 'glue-ear' and deafness problem, but there are organisational means of minimising this.

Once the ear-drum has perforated and the chronic stage has become established, effective treatment is difficult. Phillips-Turner (1965:36) states, 'A surgeon would think himself lucky if he could arrest chronic suppurative otitis media in three cases out of four treated, even in the most favourable conditions'. The 'glue-ear' problem is much easier to treat, and is therefore a lesser problem regardless of incidence.

Other ear diseases do not attract much attention in the literature on Aboriginal health. Inflammation of the external ear canal ('tropical ear', 'swimming-pool ear', etc.) is not infrequently encountered in coastal areas, but although it can be painful and sometimes difficult to treat effectively in Aboriginal communities it is of minor importance when compared with middle ear disease: it rarely leads to permanent deafness. Temporary (but sometimes prolonged) partial deafness may result from mechanical obstruction of the ear canal by the discharge in untreated cases, which may be an educational handicap.

13 DISEASES OF THE CIRCULATORY SYSTEM

A wide range of diseases of the heart and blood vessels has been reported in Australian Aborigines. While there is some evidence that Aborigines living under nomadic conditions suffered very little from the degenerative diseases associated with atherosclerosis and hypertension, modern Aborigines appear to be affected to the same extent as modern white Australians if they survive to and beyond middle age. A study of degenerative heart disease in Aborigines is relevant to world-wide research into causal and preventive factors, but not relevant to Aboriginal morbidity in particular.

If Aboriginal morbidity in terms of the *incidence* of degenerative heart and vascular disease is not exceptional (this view can be neither supported nor refuted from available statistics), it is not to say that Aborigines as a group do not suffer more than white Australians from its consequences. Treatment—or rather the control of symptoms—is a long-term affair requiring the active and informed co-operation of the patient and repeated evaluation by the physician. Ideal conditions for treatment along these lines are unlikely to exist for a large part of the Aboriginal population, so that severe disability and death for those affected is likely to occur at younger ages than in the white population. In common with other progressive conditions, age-specific death rates for Aborigines from degenerative heart disease can only reflect the balance between age-specific incidence, the rate of progress of the disease in the untreated case, and the halting or temporary reversal of this progress by medical treatment. Early death will lower morbidity prevalence rates but will not, of course, influence the incidence rate. There are no data for

incidence in respect to most chronic progressive diseases in the Aboriginal population.

It is not proposed to consider degenerative heart disease any further, but to turn instead to an important *preventable* cardio-vascular disorder in Aborigines—rheumatic heart disease.[1] The established disease in an individual is influenced by the same factors—age of onset, rate of progress and efficacy of medical intervention—but the peak incidence is in childhood and early adolescence, and is preceded by one or more attacks of rheumatic fever, which in turn are preceded by infections with haemolytic streptococci. The incidence of rheumatic heart disease is thus related to the incidence of streptococcal infections, but the principal measure to prevent rheumatic heart disease is the long-term prophylactic administration of antibiotics to children who have suffered one attack of rheumatic fever. More general measures to reduce the incidence of streptococcal infection, limit epidemics of this infection, and treat and observe carefully those children who still contract a streptococcal infection, are also important.

The extent of the problem is exemplified by the studies carried out among north Queensland Aboriginal children by Galbraith (1967) and Jose and Welch (1969). Their figures for the streptococcal carrier rate and streptococcal antibody pattern have been mentioned already (eighteen times the carrier rate and about seven times the high-titre antibody rate when compared with Brisbane children—see p. 155). Jose and Welch report the *incidence* of acute rheumatic fever to be around 80 per 10,000 per annum for Aboriginal children aged 5 to 15 years (from twelve Queensland settlements), compared with rates of 12 per 10,000 for Melbourne children of equivalent age *twenty years earlier* (in 1948, before antibiotic prophylaxis came into use). The authors also compare the all-ages incidence in the Aboriginal population of 27 per 10,000 with the notification rate of 0·9 per 10,000 for Queensland as a whole, in the same years.

Organic heart murmurs, indicating damaged heart valves mostly due to rheumatic fever, were reported by Jose and Welch (p. 216) to be ten times more common in the Aboriginal children than in Sydney schoolchildren. Of possibly greater significance was the evidence for a high prevalence of low-grade rheumatic carditis: three out of four malnourished Aboriginal children had abnormal levels of 'heart reactive

[1] 'Preventable' in the sense that prevention has been very nearly achieved in the white Australian community—not in the sense that there may be some diseases which are not preventable at all.

antibody', compared with about one in eight 'normal' Aboriginal children and one in thirty-three white Brisbane children. This finding, associated with hyper-immunity to streptococcal antigen and biochemical evidence of myocardial injury, implicated low-grade rheumatic carditis as a contributor to ill-health in half the Aboriginal children tested. Jose and Welch drew attention to a possibility that a proportion of these children may progress to rheumatic heart-valve disease without suffering a 'classical' attack of rheumatic fever. Such children will thus by-pass the clinical indications for commencement of antibiotic prophylaxis and treatment of the carditis in its early reversible stages. This suggests that a preventive measure of great value with well-nourished, generally healthy white children may not be generally applicable to Aboriginal children. Short of administering prophylactic antibiotics to all Aboriginal children from early infancy and at least postponing the first attacks of streptococcal sore throat, or continuously monitoring the serological and biochemical indicators of subclinical rheumatic carditis (both heroic measures with unwanted side-effects), what else can be done other than to radically change the general socio-economic, disease, and nutritional setting in which rheumatic fever and its sequelae are encouraged? The pattern of events outlined above suggests that rheumatic heart disease is probably not 'preventable' in the Aboriginal population by medical technology alone, as it is close to being in the urban white population. Even though the picture given may be more or less complex than reality, this disease is an excellent example of diseases with complex aetiological factors following an 'abnormal' pattern in the Aboriginal environment, and not necessarily amenable to the methods of control and prevention which are effective in other populations. It points to the need for continuing medical and social research beyond the mere identification of the presence and prevalence of disease.

The extent to which Jose's findings are applicable in other parts of Australia is unknown.

The following story is offered to illustrate another aspect of rheumatic heart disease—this time in a part-Aboriginal community. The writer was once asked to 'have a look at' a sick child while paying a courtesy call on her mother, a woman who had received some general nursing training and who had helped the writer during a previous medical survey. The child had obvious acute rheumatic fever with carditis and a valvular murmur. It turned out that the child had had at least two previous admissions to hospital for rheumatic fever; after the last admission the child was prescribed continuous prophylactic penicillin by

mouth and referred back to her general practitioner. When the tablets ran out, the mother neglected to obtain a further supply 'because - - - - seemed so well', and the child had been without medication for about three months.

But this was not simply ignorance or negligence on the mother's part. In further conversation the mother said that she had had a fierce argument with her general practitioner about which hospital the child was to be admitted into (with the previous attack of rheumatic fever), and desperately wished to avoid confronting him again. With a lot of moral and some logistic support an appointment was arranged with a 'new' general practitioner in the town and the child was referred to a city hospital, where she spent many months recovering from her illness. The mother's—and indirectly the child's—need in this case was a sympathetic informed 'organiser'—such as a community health nurse—to rescue her from a psychological situation with which she could not cope alone. Her brief nursing career may have been a handicap rather than a help with the problem. Her faith lay in a city hospital over a hundred miles away. If the child grows up to have rheumatic heart disease, what was the 'cause' in her case?

14 DISEASES OF THE RESPIRATORY SYSTEM

There is a little more to add to the picture of diseases of the respiratory system already reviewed in the section on infectious and parasitic diseases. The acute non-specific respiratory infections—'broncho-pneumonia' and 'pneumonia'—which figure so prominently in the Aboriginal mortality statistics are usually secondary to other respiratory infections, particularly viral infections, or secondary to any other debilitating illness including gastroenteritis, malnutrition, alcoholism, or exposure to cold. Pneumonitis caused by migrating *Ascaris* larvae is also secondary in this sense. The less common specific bacterial pneumonias (streptococcal, staphylococcal, Friedländer's pneumonia) may be assumed to share a place among the secondary pneumonias in the Aboriginal population, but data on their individual incidence are not available. A great number of other common diseases like measles or chickenpox often have a pneumonic component which may be serious or fatal. Primary pneumonias are uncommon in Western societies but are the most common cause of 'pneumonia' in otherwise healthy adults. Investigation of cases of so-called 'primary atypical pneumonia' will frequently identify a communicable viral agent, and an unidentified or undetected virus is assumed to be responsible in the remainder of cases. On circumstantial grounds, primary atypical pneumonias would be expected to be more common in Aborigines living under poor conditions, but the conditions tend to isolate sufferers from the virus laboratory facilities necessary to confirm or refute the expectation.

Although most pneumonias in the Aboriginal population are without doubt secondary to other diseases, they contribute greatly to respiratory

morbidity as well as mortality in that they convert what is often a trivial or minor or transient illness into a major illness, which may be followed by prolonged debility, or permanent sequelae.

An investigation of the individual incidences of the various types of pneumonia in the Aboriginal population may yield unexpected results, but is unlikely to contribute much to the solution of the 'pneumonia problem' any more than the identification of the individual causes of gastroenteritis can help solve the 'gastroenteritis problem'. Factors in the socio-economic, physical, and disease environment predisposing to all pneumonias can be seen as far outweighing factors likely to predispose to a specific infection. Under these conditions, 'pneumonia' is a health problem but hardly a medical problem at the community level.

The other important group of respiratory diseases are the chronic, often progressive, conditions that interfere with pulmonary function and thus with physical capacity—chronic bronchitis, emphysema, asthma. Such diseases assume greater importance for the unskilled and poorly-educated who rely on their physical capacity to earn an income. Many Aborigines are highly skilled as stockmen, timber-getters, fencers, shearers, or truck drivers, but even these occupations require a measure of physical strength and endurance often exceeding that required by a general labourer.

Added to the handicap of a reduced physical capacity, sufferers are more severely affected by intercurrent respiratory infections like 'colds' and 'flu'—to the extent that the latter may put them off work frequently enough to jeopardise employment even in 'light' jobs. Asthma affects all age-groups, emphysema is usually confined to older adults unless brought on earlier by severe asthma. Bronchitis tends to be 'recurrent' in the young and 'chronic' in older adults.

Gandevia (1967) used the prevalence of a loose cough and the presence of abnormal breath sounds to estimate the prevalence of chronic respiratory disease in Aborigines from settlements at Papunya and Warburton, in the central desert. He allowed that some false diagnoses would arise from the same signs being present in acute, transient infections, but the criteria have the advantage of being simple, and comparable between groups and populations. He found his criteria satisfied in about half the adults at Papunya and in about four-fifths of the adults at Warburton. Aborigines of the Pintubi tribe, particularly those recently arrived from a nomadic existence, contributed the bulk of the positive cases at Papunya, and all those studied at Warburton were recently-arrived Pintubi. Among those resident at Papunya for at least eighteen months, only one-third had

signs of chronic respiratory disease. Gandevia has advanced the theory that the higher prevalence in recent arrivals reflected first exposure to new infections in the change from the isolated, nomadic environment to the larger settled community, and this seems very likely.

For comparison, Gandevia estimated the prevalence of loose cough at less than 30 per cent for white Australian male adults (related mainly to smoking), and of adventitious sounds at less than 5 per cent.

The prevalence pattern in the Aboriginal children in these populations showed little difference between the 'new' Pintubi children and adults (both around 80 per cent), while in the remainder ('old' Pintubi and others) the peak prevalence was around 55 per cent in children in the 4–7 age-group, falling progressively thereafter to the adult level of one-third.

Maxwell *et al.* (1968) carried out a more detailed study of the children at Papunya, and concluded that there was 'a formidable health problem' caused by respiratory infections—including ear, nose and throat infections. A radiological survey showed lung abnormalities in about 30 per cent of children aged from birth to 15 years, the most common abnormalities being pulmonary consolidation (16 per cent of all those examined), accentuated basal shadows (3·6 per cent) and probable bronchiectasis (just over 3 per cent). The clinical examinations showed the presence of abnormalities (loose cough, adventitious sounds, etc.) in about 38 per cent of the children. One-quarter of the male children, and one-twelfth of female children (about one-sixth of children of both sexes) had signs such as marked chest deformity, impaired chest movement, or an acute lower respiratory tract infection such as bronchiolitis or bronchopneumonia. The respiratory disease was related to nutritional state but not to age.

Among Queensland Aboriginal children, Jose *et al.* (1969) recorded about 4 per cent with active chest infection (chest signs plus fever) but excluded those with 'chronic bronchitic cough'. They stated that 'chronic infection of the nasal sinuses, ears and lower respiratory tract was very common especially in the pre-school age group'.

In Sydney metropolitan Aboriginal children, Lickiss (1970) noted adventitious breath sounds in 12 per cent of 114 examined, but in a New South Wales coastal rural area, only 5 per cent of children were considered to have an abnormality (Hausfeld and Moodie, 1967).

The prevalence of such signs can vary greatly due to seasonal factors, in that a mid-winter survey would be expected to reveal more respiratory infections (both 'acute' and 'chronic') than a similar survey in mid-summer: the criteria of abnormality may also vary. It is not particularly informative, therefore, to persist with the review of the prevalence of

physical signs. It would be of greater value in determining respiratory morbidity to collect data on pulmonary function (expiratory volume and peak flow) along with the physical signs present in all age-groups. Although pulmonary function may be estimated with relatively simple portable equipment, no investigations of this nature have been reported. The end results of the high burden of respiratory infections in Aboriginal children and adults therefore remain undetermined.

Asthma may be a seriously incapacitating disease from early childhood or from any subsequent age, although many children, including Aboriginal children, 'grow out of it' without suffering permanent incapacity. This disorder has not been studied in detail in the Aboriginal population, although the fragmentary evidence suggests it is relatively common. A questionnaire administered to part-Aboriginal mothers in a New South Wales coastal community established a history of asthma in 3·4 per cent of 320 children. However, 'asthma' may be confused with 'bronchitis', particularly recurrent bronchitis. Very few of the children, when examined, showed evidence of asthma at the time or signs of severe asthma in the past, and the mothers often stated that their children had improved (author's unpublished data). In the same survey, one child in seven (14 per cent) was stated by the mother to have had 'pneumonia' at least once, and 2 per cent to have had 'rheumatic fever'. Asthma, at a prevalence established by the questionnaire, does not appear to be contributing overly to morbidity among children when compared with these two conditions.

Asthmatic symptoms are stated to accompany infestations with *Ascaris* and *Strongyloides*, and it is likely that at least some cases of episodic asthma in childhood—and possibly in adults—are related to the high prevalence of one or other of these worms in Aboriginal communities. Webb (1958:221) has observed that asthma in children in South India, where the exposure to infection and malnutrition in infancy and childhood parallels that of Aborigines to an extent, asthma occurs as acute episodes of bronchospasm and the picture of chronic asthma is very rarely seen. The writer has noted very few cases of asthma in the course of tropical practice in New Guinea and Sabah, and nearly all cases occurred in Chinese and Europeans.

Finally, in the category of diseases of the respiratory system, there is the ubiquitous 'runny nose'—a condition so common and so persistent among young Aboriginal children of the writer's experience that it tends to be ignored as part of the 'normal' scenery. Although such a common condition, its causes and effects do not appear to be well under-

stood. Frequent upper respiratory tract infections, such as 'colds', possibly initiate it, but its persistence between 'colds' suggests that other factors are involved—perhaps poor hygiene, perhaps nutritional deficiencies, perhaps allergy and the immune mechanisms may all play a part. While it appears to be associated with sinusitis and middle ear disease, there is no good evidence that it is a causal factor in itself, or that its treatment would reduce the prevalence of middle ear disease for instance. It is not, of course, a distinct 'disease entity' at a point in time in the life of an individual—it is its persistence in the individual that makes it an entity of note in the Aboriginal population. It is likely to play a major part in the dissemination of various pathogenic organisms (which may be present coincidentally) from child to child, and will be of social consequence to Aborigines in general in so far as it is a very visible disorder which detracts from the image of 'clean, healthy-looking children'.

15 DISEASES OF THE DIGESTIVE SYSTEM

As in the previous chapter, the most important contributors to Aboriginal morbidity in this field have been mentioned under the heading of infectious and parasitic diseases. There is another similarity in that transient but repeated infections, as well as chronic infections and infestations, may eventually produce morbidity through permanent damage to the organs involved. In this case the function of the intestine in digesting and absorbing nutriments from the food may be seriously impaired, while important organs like the liver are more likely to suffer from the repeated infective, toxic or nutritional insults (which are relatively rare in the white population). Intestinal function and liver function are not as easy to measure as respiratory function, and relatively little is known of the end-results of the pattern of frequent intestinal infection. Studies in this field, which have already been mentioned, are those by Elliott and his colleagues in Central Australia, by Jose and his colleagues in Queensland. While these studies have shown malabsorption to exist, they have not explained its origin with certainty. The malabsorption syndromes are becoming better understood with advances in investigational techniques such as closed intestinal biopsy and enzyme assay, but many of the procedures are difficult to carry out in the Aboriginal population and cannot be justified in routine surveys because they may give rise to serious complications in a proportion of individuals tested.

Harris *et al.* (1970) investigated thirteen New South Wales Aboriginal children hospitalised with a history of chronic diarrhoea. The majority of these children came from the 'Far West' of the State. The authors reported the findings on twenty-two biopsy specimens rather than on

individuals. Fourteen specimens out of nineteen showed lactase deficiency, and four showed a deficiency of other enzymes (sucrase, maltase). Of twenty-two biopsy specimens, all showed some degree of structural abnormality suggestive of atrophy, minimal in eight specimens. Intestinal parasites (*Giardia* and *Strongyloides*) were identified in only three children. The authors commented that the changes 'were probably due to a combination of causes, in which protein malnutrition and infection are most important'.

It is not known whether such children will recover normal function or remain with some degree of impairment, nor whether a persistent abnormality of an intestinal mucosal biopsy is necessarily significant to health throughout life.

Enlargement of the liver was noted in 11 per cent of Queensland Aboriginal children surveyed by Jose *et al.* (1967). No other significant liver abnormality was noted, and although liver enlargement was somewhat more common in growth retarded children, no cause was identified. Among Aborigines of the north-west of the Northern Territory and the north-east of Western Australia, Black (1950) noted enlarged livers in 14 per cent of children under 11 years, and rather higher proportions in older Aborigines—up to 22 per cent. The enlargement was suggested to have been of nutritional origin ('due to ... chronic subnutrition'). However, Black found enlarged livers in 12 per cent of white Australians in the same area, mostly adult males. In a survey of Aborigines from the Western Desert of Australia 'with no previous association with Europeans', Elphinstone (1971) noted four out of thirty-two (about 12 per cent) with enlarged livers—all from the same locality in the desert. Three had enlarged spleens as well (8 cms) and the fourth had a palpable spleen—uncommon findings among Aboriginal children. Elphinstone suspected an infective origin but was not able to confirm this. In Sydney metropolitan Aboriginal children, Lickiss (1971) noted an enlarged liver (2 cms or more) in six out of forty-one children (about 15 per cent). All children with enlarged livers were aged 1-3 years.

The significance of enlarged livers in Aboriginal children (and in children in other populations free of specific infections such as malaria or leishmaniasis which may cause liver enlargement) remains controversial. Crotty (1958), and Crotty and Webb (1960) noted liver enlargement to be 'almost universal' in Northern Territory Aboriginal children hospitalised for anaemia, and in performing autopsies on children dying from 'malnutrition and anaemia with diarrhoea or pneumonia' found excess fat accumulation typically present in those with enlarged livers.

While malnutrition, particularly protein-calorie deficiency, can be suggested as one cause, the prevalence of enlarged livers cannot be used as an accurate index of malnutrition. Clinical enlargement of a minor degree, in the absence of other abnormalities, may only reflect the relative sizes of the liver and the child, and is influenced by body build, the thickness and tone of the abdominal wall through which the liver may be felt, the technique of the examiner, and the co-operation of those examined. Liver function tests carried out by Jose *et al.* did not reveal any particular abnormality associated with liver enlargement. Various infections, migrating *Ascaris* larvae (both human and canine), and 'dietary indiscretions' have been advanced as causes of transient liver enlargements and transience of the enlargement is fairly common (Black, 1958), so that the survey prevalence of liver enlargements may vary with seasonal and epidemic factors as well as with diagnostic criteria, nutritional status, and 'general' variations in body type, development or muscle tone. Thus simple minor liver enlargement cannot be incorporated into the morbidity statistics of a population as a valid index of disease unless it can be carefully correlated with a pathological malfunction of the liver or other organs. If the pathological 'cause' is recognised, then this—rather than the enlargement—is the morbid condition.

There are a number of other disorders of the digestive system recorded as occurring among Aborigines—gastritis, appendicitis, hepatic cirrhosis, gall-stones, peptic ulcers—and none of these are unexpectedly present or absent. Their prevalence or incidence has not been reported, so that further comment is impossible.

16 DENTAL DISEASE AND DISEASES OF THE ORAL CAVITY AND JAWS

Dental pathology (and disease of the mouth in general) is an important component of morbidity. By historical accident, perhaps, or because dentistry was concerned with an easily accessible and treatable 'organ' long before medicine was put on a scientific basis (where it could successfully diagnose and treat diseases of internal organs and physiological 'systems'), medicine and dentistry have evolved as separate professions in Western society. Thus, although it is possible to view dentistry as a specialty within the field of modern medicine and health, dental diseases, their causes, effects and treatment are not subjects with which the average medical practitioner or public health scientist are well acquainted. The writer is diffident about attempting to examine and analyse dental morbidity in the Aboriginal population, but it cannot be omitted as if it was not relevant to this study.

Quite apart from the general effects on health of dental disease, or the dental or oral manifestations of more generalised disease, disorders of the teeth and mouth contribute directly to ill-health, suffering, loss of income, inconvenience, and to some extent may influence social acceptability both within and between groups of people. Severe dental disease can result in rejection for employment or training in certain categories of work. If dental ill-health is to be remedied, as well as prevented, the costs to the community in money and time can be enormous, and these costs will have to be considered along with other costs in establishing priorities for health action. The costs of private dental treatment, particularly restorative dentistry, can place dental health for the family in the category of a luxury for the 'average' part-Aboriginal household, par-

ticularly for those without the free dental treatment available to pensioners and their dependants. A free dental service has been available to the inhabitants of supervised missions and settlements in various States, but generally speaking this service can only be periodic, coinciding with the visits of travelling dental teams; between visits, dental care may not be available, or may depend on the unskilled efforts of medical or paramedical personnel and the occasional evacuation of sufferers to distant centres for emergency treatment.

The dental scientists were at first interested in the full-blood communities of northern and Central Australia for the same reasons as the physical anthropologists—the elucidation of the physical characteristics of a unique ethnic group. The dental and facio-maxillary characteristics of Central Australian Aborigines have been studied over many years, and in the course of these studies a number of observations have been made, more or less in passing, on the dental health of these groups. The traditional Aboriginal nomads appear to have possessed excellent dental health. The coarse 'natural' foods not only encouraged the development of characteristically strong jaws and well-spaced, even teeth, but discouraged dental caries and the early onset of gingival disease and degeneration. The only ill-effect, if it can be called that, was a pronounced wearing away of the opposing surfaces of the teeth—a process which does not appear to have caused a significant dental or health handicap.

As nomadic Aborigines turned to a 'civilised' diet of flour, sugar, and tea, with beef or mutton where these were produced and were freely available, both the Aboriginal dentition and dental health appear to have suffered. All dental surveys reported support this view. Later additions to the basic ration, and a more balanced and nutritious diet evolved after dietary studies, have not recreated the dental health conferred by the natural diet in nomadic times—nor is this possible now or in the foreseeable future. Part-Aborigines, and many full-bloods, who have passed through the 'tea-and-damper' era in outback Australia, have acquired other dietary habits which have had a profound influence on the dental health of the children—with sequelae for the adults. Sweet biscuits, cake, white bread and jam, 'lollies' and artificially-sweetened carbonated drinks—all the things which make the preventive-minded dentist shudder—have been added as normal components of the everyday diet, and often make up entire meals. Fresh salad vegetables and fruits, and 'chewable' meat to counterbalance the effects of the prepared high-carbohydrate foods, do not seem to have been added to the same extent—one can advance availability, cost, storage problems, convenience, and custom as likely explanations.

The major weapon against the harmful items of diet—the toothbrush—does not appear to feature very much in the personal hygiene of part-Aborigines to judge from the writer's experience. In a New South Wales coastal community, mothers or their children were asked if each child 'owned' a toothbrush; except for families living in town the answer, even for children in their teens, was usually 'no'. Those who owned a toothbrush or shared one with brothers and sisters were usually infrequent users. It is interesting that there were two exceptions—the rare toothbrush-conscious family, where every child was stated to own a toothbrush and used it twice a day; and the occasional individual child who, for some reason unknown to his mother, cleaned his teeth regularly even though he was the only person in the household who did so, and where there appeared to be little parental pressure upon any of the children to brush their teeth. The sort of remark that stays in the writer's memory is this: 'Young Billy, now, he's always brushing his teeth—can't keep the toothpaste up to him—my other kids never bother', said with an air of puzzlement as to what motivates 'Billy' and not the others. Perhaps 'Billy' was receptive to a dental health 'message' at school or on television. In a home environment where parental pressures to follow the principles of dental hygiene are not very evident, there is an interesting field of study in the 'Billys' who appear to have acquired the dental health message from some external source and followed it on their own initiative.

The very poor dental health of many part-Aboriginal children—visible to the unskilled observer as a high proportion of children with teeth decayed to the gum-line and spongy, bleeding gums—may be attributed to the combined effects of early malnutrition, unsuitable foods from a dental viewpoint, and poor or absent oral hygiene. To this may be added, at present, the generally rural distribution of Aborigines and the poor access to fluoridated water supplies now being extended to the urban and metropolitan white majority. But there is another important factor—the great majority of part-Aboriginal children receive little or no early treatment for dental caries, and do not pay routine visits to a dentist for a check-up. Some Aboriginal children obtain free service of this kind through school dental programs. Nearly all of the 40 per cent of children who had been to a dentist in the New South Wales coastal community surveyed by Hausfeld and Moodie (1967) had gone only to have a persistently aching tooth extracted. Routine dental care by private dentists was non-existent in families in this population, who were battling on the settlements in the area or who were battling on the rural-urban fringe. If cost was not the only factor, it was a major one—not merely a

matter of a few dollars for two or three children with a few cavities each, but probably hundreds of dollars for five or more children with hardly a sound tooth between them.

The Far West Scheme in New South Wales helps Aboriginal families with children suffering from severe dental disease, and children who require extensive dental treatment are accommodated in Sydney for this purpose. There may be similar opportunities for Aboriginal children in other States. But this valuable service can contribute little in the preventive field, or in the maintenance of dental health for those treated once they have returned home. Along with dental health education and other preventive measures including fluoridation of water supplies, there is a need for continuous access for Aboriginal children to free or low-cost dental care in the areas in which they live. Outside of settlements and missions, free or low-cost dental care cannot fairly be offered to Aboriginal and part-Aboriginal children without offering the same service to children in the general population. A dramatic improvement in Aboriginal dental health is therefore unlikely until a free dental health scheme is generally available, at least for children. Aborigines in impoverished families are not likely to benefit relative to more affluent Australians from a partly-subsidised or voluntary insurance scheme for dental treatment, for the same reasons that they are at a relative disadvantage with respect to medical and hospital insurance.

Adequate dental care for Aborigines living on settlements and missions, and in other isolated situations, cannot be provided through visits from dental teams unless the visits are very frequent. There is a case here for the training and employment of Aboriginal dental 'hygienists' or 'assistants', capable of performing simple fillings and extractions as well as the ordinary duties of a dental hygienist working with a qualified dentist. As with medical auxiliaries, the conflict between governmental and professional demands for high standards and high qualifications on the one hand, and community needs for easy accessibility and continuous cover at low cost on the other hand, will have to be resolved. The initiation of a dental auxiliary scheme will meet less resistance from professional groups if it occurs *before* dental care by private practitioners is subsidised by government or covered by insurance, so that there is some urgency in the matter. (The boat has already been missed in relation to medical auxiliaries who may find themselves in competition with private practitioners, whose 'bread-and-butter' income is derived ultimately from a voluntary or subsidised medical insurance fund.)

Notwithstanding the importance of dental health, the communicable

diseases and malnutrition are such important causes of mortality and morbidity in the Aboriginal population that adequate dental care cannot be given the high priority it deserves in the white population. However, there are reasons other than those of medical priority for including adequate dental care in community health programs for the benefit of Aborigines.

17 OTHER CATEGORIES OF DISEASE AND DISABILITY

This is a 'dust-bin' category covering a multitude of defined and ill-defined illnesses recorded in the Aboriginal population. The medical literature contains references to neoplastic diseases; diseases of the genito-urinary system; complications of pregnancy, childbirth and the puerperium; diseases of skin and subcutaneous tissue; diseases of the musculo-skeletal system; congenital anomalies; certain causes of perinatal morbidity and mortality; and symptoms and ill-defined conditions. But although references to these categories are often vague, restricted, too few or too out-of-date to warrant separate consideration, it is not implied that they are of little significance to Aboriginal morbidity. Some of them can cause life-long handicapping from birth or early infancy, and some of them are major contributors to infirmity in later adult life and in the aged. Morbidity arising out of accidents and injuries is not very well documented, although these causes occupy an important place in the pattern of mortality.

To understand the apparent neglect of these disabilities in current medical literature, it is necessary to review briefly the history of medical interest in the Aboriginal population—that is, medical interest as revealed in published material. There is little doubt that the medical profession in contact with Aborigines has always been interested in Aborigines as patients, in the health problems of Aboriginal communities, and in the factors which influence Aboriginal health.

Much of the early writing on Aborigines by medical men was concerned with physical anthropology—with Aboriginal 'racial' characteristics—to which subject the writers were attracted by their training

in anatomy and physiology. This led to surveys and measurements of full-blood groups in the most remote and inaccessible places, and treatises on Aboriginal and Tasmanian bones, blood groups, and other characteristics. During such surveys, diseases were often noted and commented upon, but rather as an aside or a by-product of an expedition. The three occurrences which were most likely to draw comment were—'an unusual case of----', 'an unusual prevalence of----' or 'an unusual absence of----', in that order. It seems certain that 'unusuality' is very much conditioned by the times, and what is usual in the Aboriginal population and unusual in the white population today was often the expected finding in the white and Aboriginal population a century or more ago, when a great number of white Australians lived in poor economic and environmental situations, and when the control of communicable diseases had advanced very little beyond knowledge of the general principles of sanitation and hygiene.

The part-Aborigines, having lost their racial 'purity', were of no particular interest to the physical anthropologist-physician—at least in terms of the announcement of discoveries.

Writers such as Basedow (1932) and Cleland (1928, 1962) have patiently reviewed and recorded the occurrences of almost every known common disease in the Aboriginal population, and have noted the prevalence of some of the more unusual conditions in the full-blood communities they surveyed. Their findings, taken as a whole, tend to dispel any idea that Aborigines reacted uniquely to old or new diseases, but have done little to raise the diseases mentioned earlier in this chapter from the 'dust-bin' category. It is possible that the 'dust-bin' is where they belong if it could be shown that they are neither unusual in character nor important in prevalence, but the writer suspects that this is not the case—particularly with reference to diseases causing long-term physical handicapping of adults. The identification of the nature and extent of the Aborigines' problems in this area can occur only through large-scale and broadly-based morbidity surveys or the recording and compilation of nationwide —or at least State-wide—morbidity statistics. The identification of hitherto unsuspected health problems in the Aboriginal population can be expected to awaken the interest of medical (and, one hopes, social) researchers, who will want to find out why. In the process, as with any research, a considerable spin-off of knowledge in other fields can be expected.

The identification of chronic or obscure handicaps, and the estimation of their impact on human ecology, is a rather specialised field, but the

broad picture of morbidity, including morbidity from the 'dust-bin' diseases, requires no more than the careful compilation of data which are being acquired daily by doctors, nurses, hospitals, and clinics throughout Australia: that is, the recording of data whether they appear unusual or otherwise.

DISEASES OF THE GENITO-URINARY SYSTEM

Acute glomerulonephritis and its sequelae share the same aetiological agent as rheumatic fever—the ß-haemolytic Group A streptococci—although some other infections can also lead to nephritis. Because of the common aetiology, however, it would be expected that acute and chronic nephritis would be relatively common in the Aboriginal population. Jose *et al.* (1969) diagnosed glomerulonephritis from urine examination in nineteen children from a random sample of Queensland missions and settlements. The authors do not state the number of urine specimens tested: it was unlikely to be more than 500, which yields a prevalence of 4 per cent with evidence of nephritis (subacute, resolving or chronic). From tests on 1,005 urine samples from Aborigines of all ages at Arakun and Doomadgee, along the Gulf of Carpentaria's eastern and southern shores, 2 per cent were diagnosed as suffering from glomerulonephritis. In a coastal New South Wales community, Hausfeld and Moodie (1967) reported only one child out of 176 tested as having albuminuria to a degree consistent with nephritis—0·6 per cent. A repeat survey four years later showed three out of 193 children (1·6 per cent) with significant albuminuria (unpublished data).

These few figures suggest that nephritis and its sequelae *may* be an important contributor to Aboriginal morbidity, particularly in northern communities, and it warrants further evaluation. Persistent albuminuria in adult life, even in otherwise 'healthy' individuals, is a common cause of rejection for permanent employment in the Public Service, and for training in such fields as nursing, teaching, or in the armed services. If this condition turns out to be a handicap to permanent employment or specialised training for a proportion of Aborigines ten or twenty years from now, it is important now because its origin will lie largely in the unidentified and untreated acute episodes among Aboriginal children of the present day. Like rheumatic heart disease, however, prevention may prove to be exceptionally difficult in the outback Aboriginal environment.

The gynaecological disorders are another group within the category

which would be expected to feature prominently in the Aboriginal population—particularly those disorders arising from repeated childbirth. The almost complete absence of such conditions from the literature on Aboriginal health can be attributed partly to the reluctance of Aboriginal women to seek advice or treatment for gynaecological ailments, or to have periodic post-natal 'check-ups' and 'cancer checks', and partly to the extreme difficulties in carrying out surveys for gynaecological disease.[1]

COMPLICATIONS OF PREGNANCY, CHILDBIRTH, AND THE PUERPERIUM

These days, the great majority of Aborigines—both full-blood and part-Aborigines—give birth to their babies in hospitals or in delivery-rooms under the care of doctors or trained nurses. A large proportion attend ante-natal clinics at least once, particularly with their first pregnancy. From routine records kept in relation to these events, there is a wealth of data covering not only the problems of pregnancy and childbirth but also the general health of Aboriginal women in the child-bearing age-group. With one exception, there has been no attempt to collate and publish any of this material. The publication of sundry unusual cases has merely indicated again that Aborigines are subject to the same sort of unusual obstetrical complications as any group of fertile women without conveying anything of the impact of obstetric morbidity in the Aboriginal population.

The exception referred to above is thus particularly valuable, if limited as to its relevance throughout Australia. Morrison (1968), published an analysis of 100 consecutive deliveries of Aboriginal mothers coming from urban surroundings in and around Perth in Western Australia. The author compared some of his findings with those for white women from the lower socio-economic class (social class 4) admitted to the same obstetric unit—a group who were known to be at high risk. The findings, which were published in considerable detail, can only be summarised here. Forty-three of the Aboriginal patients were 'unbooked'—i.e., had not attended the hospital's pre-natal clinics. The proportion of primiparae and multiparae 'unbooked' was identical.

[1] The writer has had experience of this difficulty during a survey throughout the Northern Territory in 1968. The idea of an external examination of the genitalia caused embarrassment to a majority of Aboriginal women when asked if they had any objection, even when the examination was performed by a nurse. An internal pelvic examination would have been more strongly resisted, and any attempt to include these procedures in a survey would have resulted in massive non-co-operation—quite understandably.

From the histories obtained on admission, Morrison noted that the 'previous still-birth rate was 35'—presumably 35 per 1,000 total births. The figure calculated from the data supplied by the author for stillbirths and live births is 25 per 1,000 births, and it is possible that the figure of 35 is a misprint or an error in calculation. The author gives the perinatal mortality rate as 81 per 1,000 full-term previous pregnancies and the abortion rate (i.e. miscarriages) 65 per 1,000 pregnancies. With complications occurring prior to delivery, the author compares the following percentage prevalence figures with those for the non-Aboriginal patients (in parentheses)—moniliasis 32 per cent (17 per cent); trichomoniasis 25 per cent (6 per cent); anaemia 18 per cent (9 per cent) and urinary tract infection 19 per cent (10 per cent). Pre-eclamptic toxaemia, together with essential hypertension and unspecified toxaemias of pregnancy, were present equally in both groups at about 32 per cent, although the disorder in the Aboriginal group was more often 'unspecified' because the mothers were not seen prior to the onset of labour. The prevalence of heart disease was 3 per cent (0·5 per cent), and of diabetes 2 per cent (0·6 per cent).

With complications of labour, it was noteworthy that disproportion occurred with 10 per cent of the Aborigines and 4 per cent of the whites.[2] Prolonged labour, breech delivery and other abnormal presentations, Caesarean section and puerperal pyrexia occurred twice as often in the Aboriginal group. All other complications noted were a little more frequent in the Aboriginal group. The author does not give the overall incidence of complications of labour and puerperium for the 100 Aboriginal mothers; the figures show that 105 complications occurred in the 100 deliveries, including 15 artificial ruptures of the membranes. For non-Aboriginal mothers, Morrison's figures indicate 76 complications occurred in each 100 deliveries, including 21 artificial ruptures of the membranes.

There were 103 babies born, including three sets of twins. Of these, 12 weighed less than 2·5 kilograms (5½ lbs), and only one of these 'immature' babies was from among the three multiple pregnancies.

Foetal distress was diagnosed in twenty-two cases during labour. Two of the underweight babies and three others were stillborn, and there were two neonatal deaths. Three babies were stated to have had major congenital abnormalities, none of which appear to have been responsible for stillbirth or neonatal death. The author calculates the perinatal death

[2] The higher frequency of disproportion probably reflects a higher frequency of small pelves, but the author does not comment on the cause of disproportion in his study.

rate for this series of pregnancies at around 68 per 1,000 births, and comments that this was twice that found in social class 4 in the white population.

Although this is a small series, it is a valuable pointer to likely obstetrical problems in the Aboriginal population. Such studies may be easily replicated in other Aboriginal and part-Aboriginal communities and should provide a sensitive index of the health of Aboriginal women in the childbearing age-group at the same time as they will show the risks associated with pregnancy and childbirth.

DISEASES OF THE SKIN AND SUBCUTANEOUS TISSUE

This category includes a number of non-specific infections of the skin—'sores', 'impetigo', and diseases such as eczema, dermatitis, chronic ulceration, etc. Beyond the recording of the occurrence of many of these conditions by Cleland (1928, 1962), Basedow (1932) and others, very little is known of their current prevalence in the Aboriginal population. In a survey of New South Wales coastal Aboriginal children, 20 per cent were found to have some abnormality such as sores, infected cuts and scratches, or a non-specific 'ringworm' (Hausfeld and Moodie, 1967). Less than 3 per cent of white schoolchildren were 'notified' as having skin diseases in the report on school medical examinations in New South Wales as a whole (1965 Report of the Director General of Public Health), but the figures cannot be compared. The majority of skin diseases noted in the Aboriginal children were of little direct significance to morbidity, but being highly visible afflictions they are likely to influence social acceptance of Aboriginal children in mixed communities and may interfere with school attendance. Aboriginal mothers in poorer circumstances, particularly those with numerous children, do not appear to pay much attention to festering sores, impetigo, or ringworm among their children until their attention is drawn to these conditions by outsiders—school teachers, community health nurses, and so on. From questioning mothers the writer has gained the impression that an Aboriginal household rarely contains an adequate 'first-aid' kit—even of the 'iodine and bandaids' variety—to handle these minor complaints. This is another fruitful field for community health nurses and health auxiliaries to provide a service and impart a little health knowledge in the course of their rounds, and in the process gain the necessary confidence of the children and their parents; confidence which will be invaluable when a serious illness occurs.

DISEASES OF THE MUSCULOSKELETAL SYSTEM AND CONNECTIVE TISSUE

A 'normal' range of the bone, joint, and ligamentous diseases in this category has been observed in the Aboriginal population without data on prevalence or on the effects of such diseases on the Aboriginal communities. The one disease of bone which has attracted a deal of attention over the years is 'boomerang leg'—a disease characterised by anterior bowing of the bones of the lower leg, with the tibia in particular assuming a curved, somewhat flattened shape rather like a returning boomerang. In life, the muscles and tissues of the lower leg, which are not usually heavily developed in northern Aborigines, appear as if strung across the bow between the back of the knee and the ankle. The calf muscles are reduced in lateral diameter while increased in the antero-posterior diameter, so that the lower leg appears extraordinarily thin and blade-like when viewed from the front and quarter-moon shaped when seen from the side. The two ends of the shaft of the tibia may be out of alignment by as much as 70° in severe cases; both legs are usually affected to an identical degree, and the sufferer is compelled to stand with slightly flexed hips to maintain his balance comfortably.

Hackett (1936a) reports that this condition was first recorded by Stirling in Central Australia in 1894, and he states that the condition was not only common throughout the Northern Territory and Central Australia, but has been observed affecting Aboriginal bones found in south-eastern Australia. 'Boomerang leg' of a pronounced degree is still fairly common throughout the Northern Territory, and minor degrees of anterior bowing, barely detectable unless tested for with a straight-edge, is even more common. Of over 1,500 Aborigines of all ages, examined during a Territory-wide survey in 1968, 4·7 per cent showed some degree of anterior bowing, while the prevalence in the over-44 age-group exceeded 20 per cent (Moodie, unpublished, Moodie *et al.*, 1970). The condition was as common in the northern division of the Territory as it was in Central Australia (Moodie, unpublished). Thus 'boomerang leg' is far from being merely a medical curiosity of a bygone era. The likelihood of the minor degrees—which are little or no physical handicap—progressing to the more marked deformity is unknown.

The cause of this disorder also remains obscure. Hackett (1936) believed the deformity was a sequel to yaws, but if this is so it is strange that 'boomerang leg' in its classical severe form has not been widely reported from among the millions of people affected by yaws throughout the

tropical world. 'Sabre-tibia' is a known sequel to yaws and syphilitic infection, and some have implied that 'boomerang leg' is a variation in which the usual periostitis and blade-like bony thickening along the anterior edge of the tibia is replaced by or masked by the bending of the shaft of the bone and the mechanical-structural consequences of this bending. In the Northern Territory survey, one-quarter of those with detectable anterior bowing of the tibia were sero-negative to the TPI test, which is a reliable indicator of past long-term yaws or syphilis infection. Several of the relatively few with classical 'boomerang leg' were also sero-negative. In some cases of 'boomerang leg' examined, the slightly irregular ridging suspicious of periostitis was noted, while in others the anterior edge of the tibia was smooth, regular and rather 'blunt', although with a marked anterior bow. It seems very likely that 'boomerang leg' is a disease of complex aetiology, in which treponemal infection, nutrition, and the architecture and mechanics of the lower leg may each play a variable part. It is common enough in a minor degree, and serious enough as a deformity in some instances, to deserve further study.

CONGENITAL ANOMALIES

This section excludes the congenital metabolic disorders, the hereditary haemolytic anaemias, and the hereditary and familial diseases of the nervous system, which have been considered earlier. Apart from a small number of reported cases of congenital abnormality, deformity, or monstrosity, there are no data from which to estimate Aboriginal morbidity from these disorders.[3] It has been stated earlier that mortality from congenital defects (hereditary or otherwise) appears to be very little higher than in the white population of Australia when mortality is corrected for age-distribution and it has also been suggested that the higher mortality may be accounted for by less efficient diagnosis and treatment of congenital defects in the Aboriginal population, without implying that the incidence of the defects is any higher (based on births).

[3] 'Congenital' means 'existing at birth'. Congenital disorders may be inherited (hereditary. familial); may be due to faulty early embryonic development (without any evidence for or against an inheritable factor—often ascribed loosely to 'defective germ plasm'); or may arise from damage to a 'normal' embryo or foetus by external influences, such as rubella during pregnancy, drugs, or factors not yet recognised. The terms 'hereditary' and 'familial' are almost synonymous; hereditary disorders may be detected in several generations according to a recognised pattern of inheritance, while familial disorders are sometimes defined as those which appear in several members of the same sibship—they appear to be inherited, although the mechanisms of inheritance may be obscure.

The writer's opinions on the relationship between Aboriginal ecology and the occurrence of hereditary abnormalities are stated in Appendix II.

CERTAIN CAUSES OF PERINATAL MORBIDITY AND MORTALITY

The high perinatal *mortality* in Aboriginal populations has been referred to on several occasions earlier in this study. Perinatal *morbidity*, on the other hand, is very poorly documented in the reports and literature relevant to Aboriginal health. As with obstetric morbidity, the data are collected routinely in obstetric hospitals or wards—and on remote settlements and missions—and all that is required to produce a reasonably accurate picture is that these data be collected in an organised (standardised) manner, be collated and tabulated, and published.

The most obvious condition is immaturity (prematurity), as evidenced by low birth weights in Aboriginal populations. The risks associated with immaturity—in terms of survival after birth, or ill-effects persisting throughout infancy and childhood and perhaps into adult life—cannot be predicted from birthweight alone; the circumstances under which the infant is reared—child care, nutrition, exposure to infection, access to skilled medical advice, and motivation and ability to follow such advice will have a greater bearing on the outcome than birth weight alone. Nevertheless, it may be assumed that immaturity, in giving an Aboriginal infant a nutritional and physiological, and probably an immunological handicap at birth, is a greater hazard to Aboriginal than white infants. It is difficult to judge the extent to which this handicap may be reduced by skilled nursing and medical attention in the neonatal period. The writer is aware of another difficulty in the management of immature Aboriginal babies—particularly with 'borderline' cases: this is caused by the reluctance of many Aboriginal and part-Aboriginal mothers to remain in hospital for the usual period after confinement, let alone prolong this period for the benefit of the infant. Another adverse feature in the setting of the standard health services provided in Australia is the reluctance of Aboriginal mothers in rural areas to attend post-natal and baby clinics.

A recently developed concept in relation to immaturity is the division of low-birth-weight babies into two groups—a 'premature' group and a 'small-for-dates' group. The latter, if undersized at full term, are unlikely to have the same disadvantage as a baby of the same weight born one or more months prematurely, but if small size is the outcome of poor maternal-foetal nutrition it may still be a grave hazard to an Aboriginal

infant. The writer is not aware of any published studies on this differentiation of low-birth-weight Aboriginal infants, or the causes and effects of 'small-for-dates' Aboriginal births. It has obvious relevance to both the prevention and the treatment of immaturity. The diagnosis of 'small-for-dates' Aboriginal babies cannot often be established from the dates alone, as is possible with the average white Australian mother who is date-oriented with respect to her menstrual history and the possibility of conception, but there are alternative criteria for true prematurity which can be applied in the Aboriginal population.

Another important cause of neonatal morbidity, with sequelae throughout life in a proportion of cases, is birth injury—a frequent accompaniment to a difficult labour. If difficulties due to disproportion are more common amongst urban Aboriginal mothers (as suggested by Morrison's findings in Perth), birth injuries should show an increased incidence at least to the same degree. The rural distribution and isolation of many Aboriginal communities from skilled obstetrical services is likely further to increase the incidence of birth injury, at least in those births where disproportion occurs. Again there are no data.

ACCIDENTS, POISONINGS, AND VIOLENCE

The mortality figures given earlier indicate that injuries contribute significantly to Aboriginal mortality at all ages, but particularly in childhood. Serious injuries, and most of the severe injuries, result in the victim or his parent or guardian seeking medical advice. The lack of data on Aboriginal morbidity from these causes, or the incidence of serious injury, can be attributed—again—to a failure to report and collate the information which is collected.

There are two major consequences of a non-fatal injury at any age—firstly, the acute domestic crisis that the majority of severe accidents or poisonings may produce, together with the expense of emergency transport and treatment, the loss of time from school, work, or household duties; secondly, the late and sometimes permanent sequelae affecting employment, mobility, and ordinary functioning as a member of a family and a community.

Although statistics on the incidence (by cause and result) of accidents and injuries in the Aboriginal population would provide a great deal of useful information relevant to risks and preventive measures, the assessment of long-term effects depends on prevalence surveys for residual handicaps, and on the analysis of worker's compensation and disability

pension data. No such data are available at the present time. The costs to the Aboriginal and to the general Australian community of disablement following accidents and violence cannot even be guessed without this information.

Lickiss (1971:85), in 112 Aboriginal children living in poor circumstances in metropolitan Sydney, recorded 15 per cent as having been admitted to hospital at least once for an injury, including burns and poisoning. Injury appeared to be the third commonest cause of hospitalisation after respiratory infection (29 per cent) and gastrointestinal infection (21 per cent). After observing these families for nine months, she noted that fourteen out of 109 children had been in hospital at least once during the period (for any reason), and six of these after being hit by a motor vehicle.

Hausfeld and Moodie (1967) recorded that 16 per cent of 130 part-Aboriginal children (average age about 8 years) in a New South Wales coastal community had experienced an injury serious enough to warrant hospitalisation or medical treatment. Data collected throughout rural New South Wales in 1965 (Rowley, unpublished) showed that a little over 2 per cent of 540 children had been hospitalised because of injury within the previous year. This can be taken as equivalent to an *incidence* of serious injury in children under 15 of 2 per cent per annum.

What do these figures mean? The numbers are too small to analyse them by age, sex, and the nature or cause of the injury, and without comparable data such figures are relatively meaningless. It is probable that the incidence, cause, and effect of injury varies widely between different communities. The high proportion of children knocked down and injured by vehicles in metropolitan inner Sydney, in Lickiss's study, is unlikely to be found in rural areas. In the coastal rural-urban community studied by Hausfeld and Moodie, a few children had been seriously injured as passengers in vehicles involved in collisions, but a more common serious type of 'accident' was poisoning through drinking kerosene or battery acid, or swallowing a household member's 'pills'. These events are not unexpected in any metropolitan or rural-urban population, and it is not known whether the Aboriginal experience with them is exceptional in any way.

Aboriginal adult males face occupational hazards in the cattle and mining industries, in working with heavy machinery (as in road maintenance, earth moving, and agriculture), in the timber-felling and sawmilling industry, and in many other outdoor cocupations. With relatively few Aborigines employed in sedentary jobs, it would be expected that

occupational injuries may contribute to adult morbidity to a much greater extent than they do in the Australian population as a whole, but it is perhaps more important to find out if the incidence of serious injury is greater than it is in white Australians following the same occupations. There is no published material on morbidity from occupational injury in the Aboriginal population, although it is probable that the data—in at least their raw form—may exist in records maintained by social welfare and labour organisations, particularly where Aborigines are on Worker's Compensation or disability pensions.

18 SOME FURTHER OBSERVATIONS ON ABORIGINAL MORBIDITY

The foregoing chapters show up some of the defects in knowledge of the causes and nature of Aboriginal morbidity, particularly in the adult population. The most significant hiatus in knowledge, however, is the almost complete absence of studies of total morbidity—even within small discrete communities. Such studies are carried out to show not only the prevalence of all significant diseases and disabilities in all age-groups, but also to show the relationships between the diseases themselves, the relationships between sickness in childhood and sickness in adult life, the direct and indirect sequelae of morbid conditions, and so on.

Total morbidity studies of this kind require, first of all, a team approach. The necessary medical, diagnostic, and epidemiological skills are rarely found together in a single individual these days. If the findings are to have more than a merely descriptive or clinical import, the team must include members from other than medical disciplines—social scientists, psychologists, nutritionists, dentists, ecologists, statisticians, laboratory technicians, etc.

Secondly, such studies require careful organisation and planning, logistic support, equipment, and plenty of time. They can be very difficult to organise when team members, equipment, and transport are plucked temporarily from other tasks. Broadly based surveys which attempt to gain a depth of insight into problems (after first identifying them) also take time to reach a level of efficient operation in the field, and teams constituted overnight and subsequently disbanded may never reach the level of competence that the skills of its component members

would imply it should. These factors combine to make total morbidity studies expensive to mount and to maintain.

Thirdly, total morbidity studies require an exceptional degree of willing co-operation from the subjects. This co-operation cannot be had for the asking—it requires patient explanation, consideration of personal feelings and idiosyncrasies, and some evidence of good faith such as a fairly tangible and quick reward for co-operation. In the writer's experience this sort of co-operation is most difficult to obtain from part-Aborigines in settled areas—for reasons which are too obvious to require further explanation here. One cannot read Rowley's *The Destruction of Aboriginal Society* and wonder at the difficulties. In full-blood Aboriginal communities—the settlements, missions, and pastoral properties—a degree of amiable co-operation can be expected in most instances, provided that the approach is made gently and with explanation.

These handicaps can be overcome if the goal is valued sufficiently. The trouble and expense of mounting such detailed studies are easily justified if they provide the sort of data which, on analysis, show clearly the basic causes and indicate the immediate actions which are compatible with long-term answers to problems. The piece-meal study of disease entities, and the piece-meal studies of associated social, economic, and environmental factors—virtually all that has been achieved so far in the field of Aboriginal health ecology—is like acquiring sundry pieces of various jig-saw puzzles and having them lie loose in a number of boxes. Some pieces can be fitted together, but the puzzle cannot be solved. Often one cannot be sure what the puzzle is.

The analysis of such studies is a complex task requiring many specialised skills. The results may be expected to contribute not only to the well-being of Aborigines: there are likely to be lessons to be learnt which have universal application. The general dearth of multidisciplinary 'total' studies of Aboriginal health ecology is as good a reason as any for Aborgines being seen usually as recipients but not sources of knowledge of how man can live successfully on this planet. This remark is not limited to considering the success of the pre-contract nomadic Aborigines in preserving their society in an unchanged environment for many thousands of years. There are other indicators of successful adaptations of full-blood and part-Aborigines to the enormous stresses imposed by the European invasion, and the accelerating pace of change in twentieth-century Australia.[1]

[1] Features of Aboriginal society, where adopted by the emerging white society, are likely to be seen as having been 'invented' by the latter. This is hinted at by Rowley (1970: 183)

The tasks of a total morbidity survey team might be reduced and simplified if Aboriginal morbidity data were available from nationally-collected statistics—a theme of an earlier chapter. The task of analysing such data and correlating them with socio-economic data, and the mounting of special studies to make the details of morbidity clear, will still remain. The United States has both the team (as represented by the Division of Indian Health and specialised study-groups) and the statistics (as collected routinely with American Indian subjects so identified). New Zealand also has the statistics for its Maori population and can create (and has created) teams within and outside its national health service. Australia has neither the teams nor the statistics. This study indicates quite clearly the need for both. That Australian governments have been obliged to call in professional management consultants with contracts to evaluate 'the Aboriginal problem' is an indictment of our national attitudes toward continuing research into 'the Aboriginal problem'.

The next chapter will examine some aspects of the interrelationships between Aboriginal health status and the health and medical services in Australia, from the data available. It is appropriate at this point to attempt to summarise Aboriginal ecology-mortality-morbidity patterns.

Taking 'Aborigines' as meaning all those of Aboriginal descent, including only those part-Aborigines who identify as such, and considering the mortality and morbidity data reviewed, it is possible to see a typical pattern of mortality and morbidity, even though there seems to be no such thing as a 'typical Aboriginal' or, more importantly, a 'typical Aboriginal household', a 'typical Aboriginal family' or a 'typical Aboriginal community'. Nor is there a 'typical full-blood Aboriginal' and a 'typical part-Aboriginal' component in the larger population.

Is this 'typical pattern of mortality and morbidity' a valid stereotype? It cannot be entirely so, allowing for the selection of communities studied according to accessibility and identifiability, and for the eclecticism (from the negative aspect) resulting from the study of the most prominent causes of morbidity and mortality in different communities without at the same time documenting those diseases which may or may not be absent, or at very low prevalence.

The mortality patterns in the various States are remarkably similar—the similarity being to some extent emphasised by the consistent differ-

who sees the possibility that such Australian tension-relieving mechanisms as 'mateship', egalitarianism, and anti-authoritarianism have been acquired in part from contact with Aborigines.

ences from Australian figures for age-specific and cause-specific death rates. More than any other evidence, this suggests that the patterns of morbidity from potentially fatal diseases and disease combinations are also similar. It does not suggest, however, that the morbidity status of the total Aboriginal population—prevalence and incidence rates for all diseases—can be estimated by generalising from incomplete studies in small communities.

But if the total morbidity load borne by the Aboriginal population cannot be stated from the available data, there is a useful function to be served by drawing a qualitative picture of Aboriginal health, emphasising the important health problems at various ages for those Aborigines exposed to identifiably high risks (if not for all Aborigines). The picture arrived at is one of possibility as far as the individual is concerned, but a degree of certainty as far as the community—sometimes the population—is concerned. Because the ecologies—really the ecosystems—of full-bloods in 'Empty Australia' and part-Aborigines in 'Economic Australia' show clear-cut differences in many respects and imply a need for different approaches and long-term solutions to health problems, I have attempted below to construct a separate generalisation for each group.

ABORIGINAL HEALTH STATUS IN 'EMPTY AUSTRALIA': THE 'TYPICAL' CASE

A baby is born under some medical or nursing supervision, to a full-blood mother who has already had several pregnancies. The mother has come from an economically-poor and unhygienic environment to which she will return within a matter of a few days with her new-born baby. The baby is of low birth weight and has less-than-optimal stores of nutriments due to maternal malnutrition and possibly prematurity at birth. Alternatively, if the baby is large, it may still have low stores of some nutriments and will have undergone physiological stress and possibly injury as a result of a difficult labour. During the first month the baby will gain weight rapidly as a result of adequate breast feeding unless it is one of the 6 per cent who die during this period from a combination of pre- and post-natal nutritional deficiency, birth, and infective stresses. The baby will continue to grow rapidly over the next few months, and reach one of his health pinnacles at around four months. Thereafter his growth rate will decline, due to a partial failure of the quality and quantity of his mother's breast-milk supply, and the onset of a long period during which he will acquire repeated respiratory and gastro-

intestinal infections from his environment, particularly from his siblings and other children. If his mother's milk supply fails, he may acquire *additional* infections via prepared milk feeds and may, theoretically at any rate, be deprived of specific substances in breast-milk aiding specific immunity. Another 4 per cent of babies will die before the end of the first year. Termination of lactation, whether through infant death or failure of lactation, will hasten the subsequent pregnancy which will replace the child lost, or supplant him in his mother's care with another infant likely to face even greater nutritional stresses.

In the second year another 3 to 4 per cent of children will die from the accumulating infective and nutritional stresses. If he survives, the child will become anaemic, underweight, and his capacity to digest lactose will be impaired. As the child explores his domestic environment, he will start acquiring the local bowel parasites which will chronically infest him at least until his late childhood. He will be bitten by mosquitoes transmitting a variety of virus infections, some of which will make him sick, and sometimes leave a permanent handicap—physical or intellectual. Flies, fingers, and close contact with others will bring him trachoma, gastroenteritis and respiratory infections. Repeated infections will greatly tax his immunity-producing mechanisms. He will roll into fires, fall onto sharp objects, and risk drowning if he lives near water, and learn by personal experience how to survive the physical hazards of his environment.

If he survives to school age, his chances of further survival are good, but during middle and late childhood he will remain below optimal health due to anaemia, worm infestations, recurrent infections, sub-optimal nutrition, and risks permanent disability from deafness, or severe injury. Exposure to tuberculosis, leprosy, yaws, or rheumatic fever may result in a subclinical infection at this age with the possibility of developing the disease in later childhood or in adult life. Up until the age of puberty his mental health and personality development will benefit from a warm, generally permissive family atmosphere but there may be some conflicts generated by his contact with white Australian cultural values (at school) which he cannot resolve at home.

Much more than any white child he must learn to lead a 'double life' according to different concepts and values at home and at school. Although he may become adept at changing gear mentally, each set of values will undermine the systematic interrelationship of the other set. If he chooses eclectically from both sets of values (to gain maximum advantage from each set), the result will be a collection rather than a

system. The psychological stresses built in at this stage may not become manifest until his values are put to the test—at the stage when he seeks socialisation in the wider community and starts to acquire adult roles.

In later childhood and early adult life his physical health will improve as parasite loads wane, and he eventually benefits from the broad pattern of immunity to virus and some bacterial infections acquired in infancy, and is able to fend for himself to a degree in meeting his nutritional needs. He has reached his second pinnacle of physical health, but only after a prolonged 'survival-of-the-fittest' battle which will have left him with some residual defects—retarded growth, permanently damaged organs (heart, lungs, kidneys, intestinal mucous membrane, ears, eyes, teeth, bones and joints, or cerebral cortex). Nevertheless, he will probably feel healthy, and apart from the infrequently visible scars of past illnesses he will appear healthy. The invisible scars, physical and psychological, and if he is unlucky in the chronic infection lottery, the effects of tuberculosis or leprosy, will be added to the degenerative diseases and occupational injuries, and the cumulative effects of alcohol and tobacco, to hasten severe incapacity or early death. The fact, revealed in the mortality statistics, that the survivors of the severe health hazards of infancy and childhood still have higher age-specific death rates than Australian whites at all ages thereafter does not suggest that the 'fittest' are physically fit. However, some allowance must be made for the quantity and quality of medical care available to adult Aborigines in the north—care which in these circumstances may not postpone the sequelae of diseases and disorders acquired in childhood or commencing in adulthood.

For Aboriginal women the pattern of health throughout life is likely to be much the same except that occupational hazards are replaced by the equal, if not greater, hazards of repeated pregnancy and childbirth. Jones (1970:33) estimated the age-specific mortality rate for each sex in the Northern Territory full-blood population for 1958-60. His figures show higher female than male death rates between the ages of 15 and 30 years, and again after the age of 65 years—the reverse of the situation in the Australian population. In the 20-24 age-group, Jones estimates a female death rate of around 6 per 1,000 per annum, which is twice that for Aboriginal males and ten times that for Australian females.

Cause of death data provided by the Bureau of Census and Statistics, probably reasonably complete for adult deaths, show that in 1964-5 four out of fourteen Northern Territory Aboriginal female deaths in the 20-29 age-group (28 per cent) were due to complications of pregnancy and childbirth. The numbers are too small to analyse in detail and do not

carry much weight statistically, but they suggest a maternal mortality rate for these young Aboriginal women of around 1·3 per 1,000 females in the 20-29 age-group each year, as against the Australian figure for the same age-group of about 0·043 per 1,000.

A more realistic way of estimating the risks is to base the maternal mortality on pregnancies rather than on age-group populations. For Australia, in 1964–6, Townsend *et al.* (1969) estimated that there were 0·41 maternal deaths per 1,000 pregnancies at all ages, including deaths due to abortions and ectopic pregnancies. This is equivalent to one maternal death per 2,400 pregnancies. It was nearly twice the rate for England and Wales in the same period, and the authors classified over half the Australian maternal deaths as preventable. The total number of pregnancies (including abortions and miscarriages) cannot be estimated for the Aboriginal population. If the Australian maternal deaths in the study quoted (202) are related to live births in the period (674,600) the risk for Australia is one maternal death per 3,340 live births.

In the Northern Territory Aboriginal population in 1964–5, the six recorded maternal deaths accompanied 1,350 live births, yielding a risk of one maternal death per 225 live births—fifteen times higher. The excess may be attributed to the combined effects of poor maternal health, less pre-natal care (for various reasons within and beyond the control of the health service), and a lower overall quality of obstetric care. It is not evident whether the maternal deaths were more or less often related to first pregnancies, but the observation that no maternal deaths occurred before age 20 in this small series, together with the estimation by Jones (1963:88) that the average of first pregnancy in 1961 was 18½ years in the Northern Territory Aboriginal population suggests that most of the maternal deaths may have been associated with second or later pregnancies. Further elucidation of the maternal mortality (and morbidity) situation in the Aboriginal population of northern Australia must await the publication of long-term surveys.

ABORIGINAL HEALTH STATUS IN 'ECONOMIC AUSTRALIA': THE 'TYPICAL' CASE

The picture drawn here is of the part-Aboriginal living in 'poor circumstances'—relative economic poverty, a poor domestic and immediate physical environment—the low-status battler within or on the fringe of white Australian society. More so than the northern Aboriginal he fits into a 'culture of poverty' pattern, but unlike Oscar Lewis's 'culture

of poverty' among Puerto Ricans, the setting is rural-urban, the enclave is smaller and less self-contained, and there is no sustaining *imported* culture or value-system as with the Puerto Ricans, Mexicans, or Negroes in the United States. The part-Aboriginal subculture has arisen *in situ*, and the successors to the source-cultures still inhabit its fringes, but changing faster, perhaps, than the 'society' they combined to produce. A 'culture of marginality' and an ecosystem determined by marginality and poverty, perhaps.

The part-Aboriginal baby is almost invariably born in a hospital, but into a larger and more closely-spaced family than his counterpart in northern Australia. He will be of 'average' birth weight, and maintain a 'normal' weight gain for the first six to twelve months, when he will start to fall behind white Australian standards—not gradually, but stepwise. He is more likely to be bottle-fed, on a mixture made from unsuitable ingredients, his mother either getting or taking little expert advice in the matter. After a minimum period in hospital, he will be taken home to a house which will be an improvement on a humpy, but likely to be lacking many basic facilities and services, and overcrowded with children. Here he will be exposed to the same pattern of diseases and dangers as his northern counterpart, except for hookworm (in the far south), arboviruses, leprosy, leptospirosis, trachoma and a few others. As he explores his environment, particularly in his backyard, he is almost certain to acquire heavy loads of roundworm and whipworm, however, which he will maintain until at least his middle childhood. He will be more exposed to seasonal influences on infections like colds and gastroenteritis, and in particular he will risk accidental injury and death in early childhood from motor vehicles (as pedestrian or passenger), and from household 'accidents' (swallowing toxic substances like kerosene or battery acid, or burns and scalds). Like his northern counterpart, in his early childhood he will be emotionally well-supported by his mother and siblings, but the quality of this aspect of his upbringing will not be quite as warm or as stress-free or as constant. By the time he goes to school he will be more conscious of interpersonal friction or breakdown in his immediate family, and he will be more nervous when in contact with unfamiliar white people like doctors and nurses. He will be sexually precocious but also guilty or ashamed along with his knowledge.

His nutrition, particularly with respect to calories, will be better than that of the northern Aboriginal child, but his bouts of infection will be as severe and as frequent. His immunisation status will be lower, his dental health poorer. The episodes of illness will attract less attention outside

his family and will be less likely to be treated. If treatment is sought by his parents, continuity and follow-up will not usually be at the standard which can be achieved in a supervised community with a resident nurse, although initial diagnoses and treatment may be technically better.

Otherwise, the pattern of illness throughout life, for southern part-Aborigines living on reserves or in a rural-urban fringe situation, will not be very different from the pattern in northern settlements and missions and rural-urban fringes, although rates will be lower in most cases. There will be the same cumulative effect of illnesses in childhood adding to the degenerative disorders of later adult life, and risks of severe occupational injury.

The women will face greater risks in pregnancy and childbirth than white Australians among whom they live. Mortality data collected by the writer in New South Wales shows that, during the period 1950-64, 13·4 per cent of Aboriginal female deaths during the child-bearing period (20-40 years) were due to complications of pregnancy, as against only 4·7 per cent of deaths in the Australian population in the same age and sex group.

Assuming a crude death rate for New South Wales Aborigines of 10 per 1,000 in 1950-64, and a crude birth rate of 50 per 1,000 (both assumptions made in the direction which will minimise the rates derived from them), and accepting the age-sex distribution for New South Wales Aborigines as given by Jones (1970:10) from 1961 Census data as applying for the whole period, estimates of New South Wales Aboriginal maternal mortality may be made from the mortality data referred to above.

These estimates are 0·25 per 1,000 aged 20-29, and 0·69 per 1,000 aged 30-39. The equivalent figures for the Australian population averaged over the same period are 0·08 and 0·09 per 1,000.

Assuming approximately 11,000 part-Aboriginal births over the fifteen-year period, the 13 maternal deaths recorded yield a ratio of one maternal death to 850 live births. This risk is about one-third that estimated for Northern Territory Aborigines in the last two years of the same period. The ratio in New South Wales (total population) for 1955-9 (the middle five years of the period) was one maternal death in 1,380 live births, and this rate, being higher than in 1964-6, draws attention to the fact that maternal mortality in Australia has declined from around 14 per 1,000 to around 4 per 1,000 since 1950. There is no way of estimating whether there has been a similar decline among part-Aborigines. There is also no way of estimating the degree to which Australian maternal mortality rates have been disproportionally inflated

by the inclusion of part-Aboriginal deaths in Australian statistics: the Australian figures do not match 'optimum' figures from overseas, and the contribution of Australia's part-Aboriginal population, in certain age or cause groups, may be partly responsible for this.

19 CONTACT BETWEEN ABORIGINES AND THE HEALTH/MEDICAL SERVICES

It is currently fashionable to speak of the 'delivery' of health and medical services to communities and populations. The concept embraces not only the provision of public medical and health facilities and personnel, and an organisation behind these; it also includes private medical and para-medical personnel, pharmacists, dentists, etc., who may be non-organised (perhaps 'disorganised' in terms of meeting community needs) and various voluntary organisations and individual volunteers, both professional and non-professional. The way in which these combine and interact to influence health is the current 'health and medical care delivery system'. Attempts to improve the system, remove its shortcomings, or radically change it to meet new circumstances and concepts of health, is a major area of current concern and discussion with governments and health professionals. It is through the 'health and medical care delivery system' that health becomes a major political issue—and at times a very sensitive issue. And yet the effectiveness of the whole system, whether highly organised or not, ultimately hinges upon the point of contact between the public and the service ('primary' health and medical care): the most careful and detailed planning and organisation of the 'system' will not produce the desired results unless, at the periphery, the system offers close contact with the community *and the community maintains close contact with the system*. Thus acceptability is as important as accessibility, and a most important part of health planning is concerned with optimising these two factors. The planning of higher-echelon services—hospitals, specialist services, rehabilitation, even continued

domiciliary care—is directed towards handling the events subsequent to this first contact between the sick person and the outermost periphery of the health 'system'.

Traditionally, Australian and many other 'developed nation' health services have relied on the individual delivering himself to the system—and largely delivering himself *up* to the system. With increasing technology in medicine (including many facets of preventive and social medicine) the patient with a non-trivial complaint tends to be swallowed up by the medical machine, processed somewhat impersonally, and finally returned to his family stamped 'cured', 'incurable' or 'come back in three months'. Neither the patient nor his family is always fully aware of what happened to him or what the implications are for his future, and it is very difficult for him to find these things out later because the contact-point is not in his immediate environment. Individuals who have a personal contact with a doctor, a nurse, a health professional—especially through family connections or neighbours—are at an advantage over those who have no such contact. Generally speaking, Aborigines and part-Aborigines fall into the latter class. The disadvantage is not limited to a lack of understanding of personal or family health matters—it would appear likely to influence health values in the family and in the wider community.

The points at which part-Aborigines are likely to make contact with the health 'system' (including the non-systematised sectors of health and medical care) are relatively few. Many sectors are restricted to certain age or sex groups. There are the general practitioners, the hospital outpatient's clinics, the prenatal and baby clinics, the School Medical Services, immunisation and chest X-ray 'clinics', and there are people on the medical fringe such as the suburban chemists, chiropractors, osteopaths and so on who may assume some of the roles of general practitioners—with varying degrees of skill. In addition, there have been the semi-skilled or unskilled lay administrators of southern settlements and missions (or their wives) who are not properly within the 'system', and in the larger rural-urban communities in recent years the vanguard of the 'community health nurses' or 'public health nurses'—representatives of a concept of health care which is still relatively undeveloped in Australia. In the northern and central communities of 'Empty Australia' most of the above are represented only by the resident trained nurse and the infrequently visiting public health nurse, the Flying Doctor or Flying Dentist or his land-transported equivalent.

The close and continuous point of contact resides only in the resident

nurse (in the north) or the full-time local community health nurse where she exists in settled Australia. Where such personnel are absent, first contact may be made with the local settlement 'administrator' or his wife, who may act only as a further contact with health or medical professionals at a distance. For Aborigines in an urban environment, first contact with a general practitioner or hospital does not have the same quality—it is episodic contact, lasting only as long as the illness which precipitates it. The need for further explanation, sometimes repeated clarification of misunderstandings and misconceptions to patients and their families, has been strongly indicated by the writer's personal experience with family and friends, and with Aboriginal mothers and patients. In particular this applies to the preventive or 'follow-up' aspects in the management of an illness, and reassurance or advice about the future; many of the worries and questions arise only after treatment of an illness has been completed—after close contact with the hospital or general practitioner has been lost.

These aspects of the delivery of health and medical care can only be explored through the eyes of the recipient—by surveying for opinion and experience in the community. They cannot be assessed through the operational statistics of the 'system' itself, and the observations of personnel within the system can only apply to the segment of the population which the 'system' itself has selected.

There are no published studies in depth dealing with the pattern of contact between Aborigines and the health/medical care system, but some relevant data have been obtained in other studies of Aboriginal communities. Under the direction of Professor C.D. Rowley, the Social Science Research Council's Aborigines Project carried out an extensive survey of part-Aboriginal households in rural-urban New South Wales and in the Eyre Peninsula region of South Australia. The social, economic, and environmental data retrieved in this survey have been presented by Rowley in *Outcasts in White Australia*. Professor Rowley has kindly made the data relevant to health and medical services for this Aboriginal 'sample' available to the writer for analysis.[1]

The sample of New South Wales part-Aboriginal households is large enough to allow an examination of some differences in the different age and sex groups. The numbers from the Eyre Peninsula part-Aboriginal

[1] The writer was involved in the processing of the raw data from Professor Rowley's survey and takes full responsibility for deficiencies in the analysis. Unfortunately there has been no opportunity to reprocess the original data in the light of questions raised by the foregoing analysis of mortality and morbidity.

sample, and from a white control group in the same area[2] are too small to permit a detailed analysis, although the experiences of the white group are particularly valuable because the data was obtained in the same way and organised according to the identical criteria.

There were four questions directed towards establishing the health *experience* of the population studied: the occurrence of 'illness' in the past four weeks; the occurrence of hospitalisation in the past twelve months, the presence of a chronic handicap, and the results of pregnancies among the women. Although the answers were subjective in that the criteria for the presence or absence of illness or handicap were those of the persons questioned, the data give some indication of the needs of the population for health and medical care. Further questions were asked as to the type of medical advice obtained for illnesses experienced, the attendance at pre-natal and infant welfare clinics, and the persons covered by medical benefits insurance.

Table 36: *Percentage claiming illness in the previous four weeks, by age-group, both sexes*

Age-group	Percentage ill, and total in group*		
	N.S.W. Aborigines	S.A. Aborigines	S.A. whites
0-4	21 (262)	14 (85)	37 (19)
5-9	13 (222)	15 (65)	12 (17)
10-14	10 (179)	10 (49)	9 (11)
15-29	9 (269)	4 (69)	10 (31)
30-44	27 (143)	7 (41)	8 (13)
45-59	24 (75)	11 (28)	14 (7)
60 and over	39 (36)	8 (19)	33 (6)
All ages	17 (1,215)	8 (362)	16 (106)

* The figure 21 (262) represents 21 per cent of 262 persons.

In Table 36 the numbers in the two South Australian groups are noted to be considerably smaller than the number in New South Wales. The major points of interest in the figures are the relatively large proportion stated to be ill in the South Australian white children under 5, compared with the two Aboriginal groups; the high proportion of New South Wales Aboriginal adults stated to have been ill in the age-groups from age 30 onwards, and the overall low figures for the South Australian Aboriginal group. It is not possible here to enlarge upon apparent differences: the figures are given primarily as the background pattern of

[2] The white group comprised 24 families living next door or adjacent to Aboriginal households interviewed.

Table 37: *Percentage claiming permanent physical handicap, by age-group, both sexes*

Age-group	Percentage handicapped, and total in group*		
	N.S.W. Aborigines	S.A. Aborigines	S.A. whites
0–4	1·6 (257)	1·2 (83)	— (21)
5–9	1·8 (218)	— (65)	— (17)
10–14	4·5 (176)	— (47)	— (10)
15–29	4·6 (261)	1·5 (66)	— (30)
30–44	10·4 (134)	5·0 (40)	— (13)
45–59	14·5 (76)	11·5 (26)	20 (5)
60 and over	18·4 (38)	15·4 (13)	33 (6)
All ages	5·3 (1,160)	2·7 (340)	3 (102)

* 1·6 (257) represents 1·6 per cent of 257 persons.

perceived 'illness' from which to look at the other variables studied. The figures may have little or no relation to morbidity measured objectively.

Table 37 indicates that a greater proportion of New South Wales part-Aborigines perceive themselves (or their children) as having a physical handicap, and all ages are 'affected'. Although an attempt was made to establish, roughly, the nature of the physical handicap in each case, the answers showed no particular pattern. The figures exclude cases judged to have only minor handicaps not affecting employability or activity.

Table 38: *Percentage hospitalised in the previous twelve months, by age-group, both sexes*

Age-group	Percentage hospitalised, and total in group*		
	N.S.W. Aborigines	S.A. Aborigines	S.A. whites
0–4	28·2 (227)	26 (81)	24 (17)
5–9	8·1 (173)	5 (65)	13 (16)
10–14	7·9 (140)	·8 (49)	— (11)
15–29†	12·2/21·1 (213)	8/29 (72)	3/14 (29)
30–44†	18·2/34·5 (110)	8/13 (40)	—/— (13)
45–59	17·2 (58)	11 (28)	28 (7)
60 and over	33·3 (33)	12 (17)	17 (6)
All ages†	16·3/21·3 (954)	12·5/17 (352)	10/13 (99)

* 28·2 (227) means 28·2 per cent of 227 persons.
† The figure following the oblique stroke includes hospital admissions related to pregnancy and childbirth.

Adams *et al.* (1971:510), in a sample survey of families in Sydney's western suburbs, found that 14·8 per cent of persons had been hospitalised over the past two years for reasons other than childbirth: this is equivalent to at least 7·4 per cent in twelve months, a little less than half the hospitalisation rate for the New South Wales Aboriginal sample. In all age-groups

except among those aged 10-14, the hospitalisation rate for New South Wales Aborigines exceeded that for South Australian Aborigines, particularly among adults. Admissions for pregnancy and childbirth (10 per cent of all female admissions), when added to admissions for other reasons, nearly double the Aboriginal hospitalisation rates between the ages of 15 and 45 (both sexes). The admission rates for males and females in this age-group were very similar when admissions for childbirth were excluded. About 7 per cent of all admissions (excluding confinements) at all ages were for infections. About 3 per cent of all male admissions and 1·6 per cent of all female admissions (excluding pregnancy) were for injuries.

Table 39: *Type of medical advice obtained for illnesses in past month, as percentage of persons ill*

Type of medical service obtained	N.S.W. Aborigines	S.A. Aborigines	S.A. whites
Number in group	187	26	16
	%	%	%
Doctor	50	69	56
Chemist	1·5	7	31
Hospital	19·5	—	6
Nurse, station matron or free clinic	2·5	3	—
Advice indicated but not obtained	26·2	21	6
	99·7	100	99

The prominent features of the figures in Table 39 are that half the New South Wales and two-thirds the South Australian Aboriginal illnesses resulted in a consultation with a doctor—a figure not very different from that for the South Australian white control group; and that in the New South Wales group, one-quarter received no advice from any 'medical' source, and one-fifth went straight to a hospital for advice or treatment. Advice from a chemist was not popular with either Aboriginal group, but was sought by nearly a third of the white group, and left only 6 per cent as not having received any advice for an illness.

Of perhaps greater significance is the calculation that 8 per cent of the total New South Wales Aboriginal group had seen a doctor in connection with illness in the previous four weeks, as against 15 per cent of the South Australian white group. Adams *et al.* (1971:509) recorded 41 per cent of persons in their western Sydney survey as having consulted

a doctor over a period of two months—the proportion doing so in one month would thus exceed 20 per cent. Was the lower consultation rate in the New South Wales Aboriginal group due to cost factors, poor access to medical opinion, a high motivational threshold, or better health? Both objectively and subjectively the latter factor does not appear to feature (see earlier tables). With regard to cost, does membership of a medical benefit fund play a part?

Table 40: *Percentage of sample covered by medical insurance, 1965*

	N.S.W. Aborigines	S.A. Aborigines	S.A. whites
Number in group	1,195	352	103
	%	%	%
Covered by medical insurance	27·3	66	91
Not covered	62·7	34	9

The western Sydney survey by Adams *et al.* showed that 73·5 per cent were medically insured in 1970. On the surface, the figures suggest that the lower proportion covered by medical insurance in the New South Wales Aboriginal group may have led to the lower consultation rate. However, when medical insurance coverage is cross-tabulated with the proportion obtaining advice from doctors for illnesses, the figures are as follows:

	Total	Number ill	Percentage of total	Saw doctor for illness	Percentage of those ill
Covered by insurance	326	58	17·8	29	50
Not covered	869	121	13·9	57	47

showing no significant difference in the proportion seeing a doctor for an illness. Lack of medical benefit insurance was no apparent barrier to obtaining medical advice. The hidden costs of obtaining medical opinion —fares, difference between fee and sum recovered, cost of medicines— and accessibility and motivation are more likely to explain the low consultation rate for illness in the New South Wales Aboriginal group as these costs are borne by the insured and the uninsured. Unwillingness to confront 'white society' as represented by doctors, their receptionists, and other patients in the waiting room probably reinforces financial and accessibility handicaps in seeking medical advice. Because of such

handicaps, it is rather surprising that the consultation rate for New South Wales Aborigines was as high as 8 per cent per month in 1965. For all ages, the doctor (general practitioner) appears to be the major point of contact between the New South Wales rural part-Aboriginal and the health and medical system. This is followed by the hospital. Other contacts appear to have played a minor role. The pattern is likely to have changed markedly in those communities now served by a 'community health nurse' in daily contact.

The pattern in other States is unknown, but in 'Empty Australia' it may be safely assumed that first contact is usually through a nurse.

The major contribution of childbirth as a cause of contact of New South Wales part-Aboriginal women with hospitals has been noted. Between the ages of 15 and 45, 64 per cent of female Aboriginal hospital admissions were for a confinement, and around 20 per cent of all women in this age-group were confined in hospital in one year. What of the contact with non-hospital services for the same reason?

Table 41: *Proportion of women attending pre-natal clinics at least once for the last pregnancy previous to interview*

Age-group	Percentage attending, and total in group*		
	N.S.W. Aborigines	S.A. Aborigines	S.A. whites
15-19	89 (9)	100 (2)	100 (1)
20-29	90 (51)	90 (28)	90 (10)
30-39	85 (53)	73 (15)	75 (4)
40-49	67 (36)	50 (6)	75 (4)
50 and over	14 (24)	60 (15)	100 (3)
All ages	69 (187)†	76 (68)†	86 (22)

* The figure in parentheses is the total number of parous women in the age-group.
† Includes persons of unstated age.

The figures in Table 41 show that the majority of the younger Aboriginal mothers had made contact with the health system through pre-natal clinics. The contact achieved by the South Australian white group appeared to have been no greater, but there is little certainty in the small numbers included in this group. The older Aboriginal mothers (aged 50 and over) in the New South Wales group had had very little contact mediated by pregnancy—probably because of lack of opportunity.

Table 42 shows that all groups made less contact after the birth of their babies than before the birth occurred, but the contact (or rather the maintenance of contact established through ante-natal visits or upon

Table 42: *Attendance at baby clinic following last live birth, where infant survived at least one month*

Age-group	Percentage attending, and total in group*		
	N.S.W. Aborigines	S.A. Aborigines	S.A. whites
15-19	33 (6)	100 (1)	100 (1)
20-29	36 (47)	54 (28)	56 (9)
30-39	38 (53)	47 (15)	75 (4)
40-49	17 (35)	67 (6)	75 (4)
50 and over	8 (25)	13 (16)	67 (3)
All ages	29 (178)†	44 (68)†	67 (21)

* The figure in parentheses is the total number of women in the group.
† Includes persons of unstated age.

childbirth itself) fell dramatically in the New South Wales Aboriginal group; even among the young mothers only a third had any contact through the baby clinic situation. Because the presence of a pre-natal clinic (or opportunities for a pre-natal check-up by the local general practitioner or at the local hospital) should in most cases be associated with equal opportunity to have the baby's health checked during infancy, the difference cannot be seen as resulting from a difference in accessibility. If accessibility depends on the ability to attend a distant clinic, however, the presence of a young baby may have partly immobilised mothers relative to their mobility before confinement. Otherwise, attitudes to the need for advice and help in management may be the principal factor.

Twenty-six out of 178 New South Wales part-Aboriginal mothers had had at least one live-born baby die in infancy, and twenty-one had also had a child die between the ages of 1 and 4 years. The survey data do not permit the cross-tabulation of these experiences with attendance at baby clinics: it is unlikely that an association could be proved or disproved with the small numbers in the individual age-groups even if an association existed. The New South Wales Aboriginal ecology, and overseas experiences in similar communities (McDermott *et al.*, 1970), suggest that baby clinics may have little or no impact on infant or childhood mortality in 'poor' communities—contrary to the expectations of infant welfare services—because the common causes of high mortality (diarrhoea-pneumonia-malnutrition) are not amenable to reduction through the clinic as a point of contact. This is one aspect of what McDermott refers to as 'technological misfit in the application of biomedical technology'—a very important concept which will be considered in the concluding chapter.

In another survey into health in a New South Wales coastal community (Hausfeld and Moodie, 1967; Moodie, unpublished), where the questions related to children only, 75 per cent were stated to have had an 'illness' in the previous four weeks. The criteria here were different from those applied in the Social Science Research Council survey discussed above—by persistent questioning, children who had been described by their parent or guardian to have been 'in perfect health' for the whole month were considered to have been well, and all others to have been 'ill', irrespective of the nature of the 'illness'. The difference between the figure above and a figure of about 15 per cent for the same age-group in the Social Science Research Council survey are therefore not comparable. The figure of 75 per cent does suggest, however, a high level of awareness of the presence of suboptimal health among a group of Aboriginal mothers when pressed to commit themselves. This is against the hypothesis that a high level of 'background' illness makes illness inapparent: it does not go against the hypothesis that it makes illness 'insignificant' or 'of minor importance'.

In the same community, 12·3 per cent of 211 children had been seen by a doctor in the previous four weeks. In the Social Science Research Council survey, only 5·2 per cent of children in the same 0-14 age-group had seen a doctor in the previous four weeks. The difference may be explained on further knowledge of the coastal community's situation: the sample included a large number of children resident at a settlement which was visited regularly (weekly) by a government doctor who saw patients at no charge; in this group, 16 per cent of the children had 'seen a doctor'. In a smaller group of 'fringe dwellers' only 7 per cent had 'seen a doctor'—a figure close to that from Rowley's survey. School medical examinations were excluded from the figures for having 'seen a doctor'. The figure for a small group living in a town in relative affluence, close to doctor's consulting rooms and the hospital, was an intermediate 10 per cent.

Here were three different types of ecology in the one larger community —settlement, 'fringe', and town-dwellers. When questioned as to the past occurrence (since birth)[8] of an illness serious enough to require medical attention (GP or hospital), the town group led slightly (43 per cent) from the others (around 37 per cent each): serious injuries had been equally frequent in the histories of all three groups (around 16 per cent) but the 'fringe' group led the town group on history of ever having

[8] The average and median ages of the three groups of children were similar, around 7½ years.

been hospitalised (63 as against 47 per cent). A history of a surgical operation was equally common in all three groups (around 12 per cent). The general pattern suggested that serious illnesses and injuries were *treated* as frequently in all groups, but the 'fringe' group had a higher threshold to seeking medical consultation, but on the other hand its children were more often hospitalised. The possible interactions of social, economic, and accessibility factors to explain the differences observed are too complex to go into here, but the pattern described was not unexpected.

A most significant finding was the proportion of children who had received attention from medical specialists—the definition of 'specialist' being left to the subject's parent or guardian. The percentage of children 'ever treated by a specialist' was nil for the settlement group, 10 per cent for the fringe group, and 27 per cent for the town group. The settlement sample in this analysis was very small, but when combined with the fringe group, the town group appeared to have a significantly higher access to specialist medical care ($0{\cdot}05 < P < 0{\cdot}025$).

Remembering that the occurrence of a serious illness or accident was about the same in both group's histories, there appears to have been an economic or 'charity' barrier which only the relatively affluent and more 'assimilated' town-dwellers were able to pass. While the contact of the fringe group with the 'system' was adequate at the hospital level, they do not seem to have gained contact at the specialist level to a degree which their equal morbidity would indicate as being necessary. Specialist consultations and follow-up usually follow general practitioner referral rather than hospital admission as a public patient. While general practitioners may be willing to see Aboriginal children for no return, it is unlikely that they would as readily refer the same patients on to a specialist colleague. The argument is advanced that Aborigines in poor circumstances also face difficulties in entering the medical delivery system beyond the point of primary contact—mainly for economic reasons. This may not have been merely an inability to meet fees, because the town in question had few resident medical specialists, and consultations in many cases would have involved a journey to a city centre, the nearest being 50 miles away.

In metropolitan Sydney, Lickiss (1971:62) produced the following table relevant to contact with medical services for part-Aboriginal children in the late 1960s.

Lickiss says that the mothers saw baby health clinics as being useful for premature or 'weak' babies but not for 'normal' babies.

On discussing the low rate of attendance at baby health clinics, Lickiss

Aboriginal children in Sydney—Contact with medical services

	Percentage of children
Ante-natal supervision of mother during pregnancy preceding birth	92 (n = 112)
Immunisation (at least one dose of an immunising agent)	86 (n = 103)
Seen by private doctor at least once	71 (n = 100)
Attended at baby health centre at least once	38 (n = 103)
Hospital admission three times or more in first five years of life (children 5 years of age or more)	33 (n = 69)
Hospital admission five times or more in first five years of life (children 5 years of age or more)	17 (n = 69)

observed that 'underlying most of the discussion appeared to be the mother's fear of being found to be at fault or inadequate by the clinic sister in a cross-cultural situation' and 'mothers did not . . . fear hospital staff as much as they feared the clinic staff'. Lickiss also noted that children from pensioner families, not charged for medical attention, were frequently seen by private practitioners.

It would seem that Aborigines in the northern communities, once primary contact is made, gain access to medical attention when required by being transported to distant centres by road or air at no cost to themselves. Part-Aborigines in the south must compete with the more affluent white community to obtain the same service, and this places them at a disadvantage. This disadvantage is not likely to be removed through membership of a medical insurance scheme, which does not cover all the costs involved.

In the southern part-Aboriginal communities, the most serious defect in the health and medical care delivery system appears to lie in the frequent failure to establish and maintain contact on behalf of an infant. The part-Aboriginal infant is at high risk in health and remains so throughout early childhood. The baby health clinic, even if it was utilised by all mothers for all their children, is unsuited to dealing with infant and child populations experiencing high morbidity. Its primary role in the white community is 'well-baby care' —charting progress, giving advice on infant feeding difficulties, and dealing with minor complaints. The sick child cannot be treated adequately in the clinic situation, and if referral to a doctor or a hospital is necessary, one can understand that Aboriginal mothers may be as aware of this need as the clinic nurse, and by-pass the clinic accordingly. If a child is really 'well', the clinic may serve a purpose

in reassuring the mother that this is so, but it is an expensive (for the State) and cumbersome (for the mother) method of fulfilling this purpose, and will have little or no impact on morbidity from illness of significance to Aboriginal children.

If the concept of the baby health clinic fails to provide the sort of primary health care required for part-Aboriginal children, it also appears to have failed to be accepted as 'well-baby care' by the majority of part-Aboriginal mothers, to judge from the figures given above.

In the northern communities that have a resident nurse, the baby clinic is a different concept: the clinic is physically *within* the community, the nurse also delivers babies, and undertakes treatment of quite major illnesses. She is a familiar figure who is the equivalent of a 'doctor next door'. Provided that the nurse provides a 'domiciliary' as well as a 'clinic' service, and is familiar with households and families and not just with mothers and their youngest children, she is the community health nurse in the proper sense, and can be the ideal point of primary contact between the community and the medical/health system. Whether or not the ideal is fully realised will depend on the nurse's work load, personality, her acceptance by the community, and her training in and awareness of the cross-cultural values and behaviour patterns involved in her contacts.

There will always be a need for teams of visiting experts in the health and medical field in the remote areas of Australia—not only for Aboriginal or part-Aboriginal populations. 'Remote areas', in this context, may include towns well served by general practitioners and a local hospital, lacking only immediate access to personnel with specialised medical, health, or sociological skills. If the findings of such teams or their therapeutic or preventive capabilities are to be put to their most effective use, the 'system' will need an 'agent' in the community. Similarly, the community will need an 'agent' in the system. These two roles can be performed by the one individual, but only if the individual is *within* the community—and thus has a foot in both camps. This is the basis of the argument that the 'agent's' qualifications as a community member (in the sociological sense of the term) are no less important than his technical or professional qualifications as a health worker, and are probably more important. It is far easier to graft the technical qualifications onto a qualified community member than the other way around, particularly if community membership is determined by kinship, upbringing, and skin colour. The obvious step is to train an established member of the community for the health role. If such a step is considered impractical

because of community attitudes or the refusal of members to accept the dual role, the service needs to rethink its tasks of 'social engineering'. If it is considered impractical because of low educational standards in the community, the service may be reassured that this is of little importance and warned that the impasse it creates will tend to be self-perpetuating.

20 THE PROGNOSIS OF THE ABORIGINAL HEALTH 'PROBLEM'

In this concluding chapter an attempt is made to predict some future health problems for the Aboriginal population. It is recognised that such a prognosis is not very securely based on knowledge of past trends and present health status, and that it may be changed for better or for worse by unforeseen changes in Aboriginal or Australian ecology. Nevertheless, it is immediately apparent that it is the *prognosis* of Aboriginal ill-health, rather than its current manifestations and symptoms, that should principally determine priorities for remedial action—and determine to a large extent the general nature of remedial action.

In the clinical analogy—the sick individual—the prognosis arises from the diagnosis, and is based on a sound knowledge of the natural history of the illness and the known benefits of therapeutic intervention. The untoward effects of therapy, and the minor or major risks of intervention, are included among the factors determining the prognosis. The physician or surgeon is expected to weigh these factors and present them to his patient in such a way that the latter can assess the risks and benefits (and the expense) and make his choice. If the condition needs no treatment (or is shown by investigation not to be present), or if the condition is incurable or 'hopeless' there is no choice. If the disease is simply remedied, once and for all, by short-term treatment or minor surgery, the choice is simple. It is only when the disease is serious, but potentially curable, and there are therapeutic risks of a high order, that the estimation of relative risks becomes a difficult task for both physician and patient.

In looking at the health of a community or a population, prognosis is complicated by the 'immortality' of most communities, the 'chronic'

nature of their disabilities, the complex interaction of community health status with all other aspects of existence, and by the fact that a society may change profoundly and at a faster rate than it can be influenced by 'treatment'. The health planner may find himself in the situation of a veterinary surgeon whose sick dog metamorphoses into a horse. The anatomy, physiology, and pathology will have changed during treatment.

For Aborigines, a most important influence upon their health, past and present, has been the evolution in Australia of non-Aboriginal society, and one must assume this will be the single most important external factor in the foreseeable future. It is hardly possible to prognosticate in detail upon Aboriginal health problems, therefore, without taking into account changes in the health status, attitudes, social and economic conditions, and demographic situation of the whole of Australia at least, and similar changes in the rest of the world as these influence Australia.

In the first chapter reference was made to the apparent time-lag difference in the health status of Aborigines and white Australians—with Aborigines in the Northern Territory, and probably equally so in north Queensland and the north of Western Australia having mortality and morbidity statistics generally similar to those of white Australians at the turn of the century. In so far as Aboriginal health problems are partly determined by environment or economic status, this gap might be expected to be closing as Aboriginal housing improves and more Aborigines and part-Aborigines work for award wages, but there is little evidence to suggest that this improvement is occurring any faster than a similar improvement (from a higher take-off point) in housing, wages, and health for white Australians. Furthermore, the increasing rate of Aboriginal 'natural increase'—with larger families and increased number of dependent children—can only counteract the benefits of improved housing and lower the *per capita* income of Aboriginal households. At the same time, inflation is reducing the potential benefits of more widely available welfare assistance and pensions, and the cost of medical attention (and preventive services) is rising with the salaries of health professionals, the fees of private practitioners, and the increasing capital outlay required for advanced medical technology. These health handicaps are not confined to Aborigines—all Australians in lower-income brackets are experiencing them, but from a lower level of need in terms of the morbidity burden.

Of no lesser importance are the cultural handicaps to good health for Aborigines and part-Aborigines on the fringes of white Australian culture.

These handicaps arise from aspects of belief systems, value systems, behaviour patterns that act as barriers to preventive or therapeutic medicine by blocking contact with the health care delivery system. The failure of Aborigines to acquire behaviour patterns suited to healthy high-density living, or the urge and skills to modify their domestic environment (poverty notwithstanding) to minimise the transfer of communicable diseases, can be seen as a basic reason for the exceptionally high morbidity and mortality from infections and malnutrition.

A particular handicap of this kind arises from the lack of early personal contact of Aboriginal and part-Aboriginal women with the domestic skills and 'household values' of urban society—skills and values which have been 'traditional' among white women of all social classes since long before Australia was settled. The writer contends that there is a sort of white female domestic culture which can only be acquired and fully incorporated into a woman's value system through the mother-daughter relationship. This aspect of culture includes housekeeping, cooking, household management, and child-rearing, and the striving to 'keep a good house' or at least 'keep up appearances'—a major force for improvement of domestic hygiene. Those who feel that part-Aborigines, after many generations of contact and admixture with white or Chinese Australians, should have acquired the household arts may be forgetting one very important fact: very few part-Aboriginal mothers today have a white or Chinese female ancestor. At no point in the lineages of these part-Aboriginal women has there thus been much opportunity for even one to acquire Western household skills and values at her mother's knee. The techniques and attitudes which non-Aboriginal male ancestors could have introduced are likely to have been limited to those of the frontier bachelor. Part-Aboriginal mothers have been influenced by household 'technology' through school, magazines, TV programs, and for some the 'girls' homes' and domestic training schemes on settlements and missions. This is rather like a white Australian woman learning Japanese cooking and flower arrangement—however adept she becomes, she will not run her house like a Japanese housewife, nor regard the running of it with the same deeply-held values.

While the above may apply to the majority of fringe-dwellers, and inhabitants of settlements and missions, there are many exceptions even among those facing severe economic restrictions. The employment of Aboriginal women in homes, offices, hospitals, and factories, and the powerful influence of communication media such as television, may be expected to have a marked impact on the skills and values of Aboriginal

children and parents in coping with urban domestic life. Thus the long-term prognosis for the communicable and nutritional diseases is for a steady decline in incidence where Aborigines participate in Australian society. Where they do not participate, the prognosis is gloomy—particularly where segregated communities are increasing rapidly in numbers, density, and youthfulness.

To what extent can medical technology now and in the future influence the pattern of Aboriginal morbidity and mortality? This is a most interesting and important question; an answer is suggested by a study in the United States of a situation which parallels that in Northern and Central Australia in many ways. McDermott *et al.* (1970) have reported in detail on the results of an attempt to modify, through what they call 'biomedical technology', the disease pattern in a group of Navajo Indians —the Many Farms-Rough Rock community living in the centre of the Navajo Reservation in northern Arizona. The society studied, comprising some 2,000 non-English-speaking and tribally oriented people isolated from south-western American society by large tracts of the surrounding Navajo Reserve, had some similarities but many ecological differences from the nearest equivalent in Australia—the Aboriginal population in the government settlements and adjacent reserves in western Central Australia. In both cases there is a 'tribal society coexisting with a skeletal modern infrastructure . . . introduced, and . . . still largely managed from outside' (p. 12). However, the economy of the Navajo community was based on dry farming, sheep herding, rug weaving, silver work, and occasional labouring jobs away from the area. Housing and water supplies were primitive and latrines non-existent, but the Navajo tribal culture and social system was apparently better maintained than that of Aborigines in Central Australian settlements, and the Tribal Council was involved in day-to-day administration and decision making, and legislation, and had revenue to distribute. Into this community, a physician-based medical and health care service was introduced where none existed before.[1] The initial disease pattern was similar to that which has been described in Aboriginal communities. The five most common diseases were diarrhoea, otitis media, impetigo, pneumonia, and burns, in that order, in the two-thirds of the population who sought medical care each year from the introduced service. Important chronic diseases were trachoma, tuberculosis, and congenital hip disease.

McDermott *et al.* comment on diseases which they expected but were

[1] Members of the Many Farms community had to be transported or find their own way out of the reservation to the nearest hospital or Western-trained physician.

missing from the pattern—viral hepatitis, neonatal tetanus, mental disorders. Diphtheria and some other infections were assumed to have been prevented by the 'cordon sanitaire' of immunised citizens outside the reserve.

The service introduced was a well-equipped health centre, with a satellite facility, and several radio-equipped vehicles for home visiting. These were staffed by personnel that usually consisted of two physicians, two nurses, four Navajo auxiliary health workers and a Navajo teacher *for each 1,000 population*. The resident staff were continually guided by resident social scientists and maintained consultation with various disciplines at the parent university supporting the project (Cornell University in New York). This was an extremely 'powerful' team in terms of personnel, equipment, and support, and maintained high standards of diagnosis and treatment. It was also supported financially, practically, and 'morally' by the Navajo Tribal Council, and it may be assumed that the service available was not rejected out of hand by the target population. Care was taken not to set up in opposition to the traditional medicine-men who, it appears from the story, maintained positions of secular and religious leadership during the period of the study.

After a period of six years with the service functioning, the authors noted a definite reduction in the transmission of tuberculous infection and in the incidence of otitis media. *There was no reduction in the incidence of pneumonia and diarrhoea, or trachoma.* The infant mortality remained as before at three times the national average. Although their figures showed a slight reduction in crude mortality rates, there was 'no clear-cut evidence' that lives were saved through the provision of the service. Nor was there evidence that the community had benefited from the therapeutic 'curtailment of otherwise self-limited illnesses'.

The authors discuss the implications of these results in detail. The mechanisms whereby the powerful instrument of medical and health technology was able to control some infections (tuberculosis, otitis media) but not others (trachoma, pneumonia, and diarrhoea) cannot be easily summarised here. The pneumonia-diarrhoea complex continued to cause high infant and child mortality. The basic reason for the persistence of the latter diseases unabated was the inability of the technology, *particularly primary medical care delivered by a physician*, to exert an effect at the domiciliary level. Tuberculosis and otitis media were reduced because neither achievement required an 'associated change in the habits or structure of the home'. The failures were in spite of considerable efforts to change habits, using Navajo field auxiliaries, home visiting,

home demonstrations, and training of ante-natal and post-natal subjects in infant care.

The authors concluded that the health centre or field clinic was not capable, even with domiciliary visiting associated, of changing the pattern of events in the home, and if this had been realised at the beginning of the project 'the domiciliary practices would have been the central program to which the primary health care at the field clinic would have been the adjunct' (McDermott *et al.*, 1970:39). This is a lesson of enormous importance and relevance to the similar Aboriginal health situation throughout Australia—including the situation in part-Aboriginal communities in settled Australia.

Thus the likelihood of improvement in Aboriginal health, over a short period of, say, five years, by a great expansion of highly skilled and motivated personnel and the best of 'bricks and mortar' facilities for medical technology, is small. This is not to say that such a service would not be greatly appreciated by Aborigines, nor that it is entirely unnecessary in pursuing long-term goals for Aboriginal health. McDermott expresses the view that what was achieved at Many Farms could also have been achieved without having clinical physicians resident at the exceptionally high ratio of one per 500 inhabitants, and without having a resident physician *at all.* As McDermott also points out (p. 48), when the conventional Western medical system is once set up, '*it cannot feasibly be subjected to major change*' because 'given a choice today, the Many Farms community would opt for what was actually introduced there, rather than for a system with a much better technological "fit", *if the latter meant no physician in residence*' (my italics). The authors' final words about the failure of the Many Farms project to make its expected short-term impact on community health are all too applicable to the Aboriginal health 'problem':

> efforts to organise systematic studies to obtain this basic information [on rational systems for the application of medical technology in a cross-cultural situation] are constantly met with the rejoinder, 'Why do any more studies?—the problem is to apply what we know right now.' And the thorough application of 'what we know right now' was exactly what was done at Many Farms (McDermott *et al.*, 1970:52).

The alternative in Australia is a health and medical care delivery system mediated through resident community health nurses, assisted by indigenous (Aboriginal and part-Aboriginal) auxiliaries, supervised and supported from outside by physicians and hospitals, and *integrated* with social, economic, and educational welfare and betterment programs. No spectacularly rapid results can be forecast, but failure to initiate such a

system will certainly retard advances in Aboriginal 'quality of life' and ensure that an increasing number (although not necessarily proportion) of Aborigines remain handicapped physically, intellectually, and psychologically. All but a few favoured Aboriginal communities have a long way to go before they enjoy the health status of the tribal American Indians studied in the Many Farms project before the program there began.

The system outlined above is feasible in 'Empty Australia' without drastic reorientation of the existing health and medical care delivery system, merely by interposing a domiciliary primary health care system between the communities and the hospitals, clinics, and itinerant community health professionals already in the field. Although various State and Territory health services are already implementing these ideas in a small way, there would appear to be a real danger that they will be overtaken and 'killed' by a rapidly expanding medical service devoted to treating the seriously ill in hospitals. Systems for the delivery of medical (therapeutic) technology like the Royal Flying Doctor Service, and the Aerial Medical Service in the Northern Territory, create a greater technical misfit than most in solving the Aboriginal health 'problem'. They also face a great danger of creating a demand for an inappropriate system which, like the resident physician in Many Farms, cannot be withdrawn once it has become highly appreciated. It is not too difficult to imagine that efforts to improve the coverage of 'flying doctors' (in area or in frequency of visits) may soon lead to the situation where it is more economic in personnel and hardware to replace them with resident physicians on the ground, even for relatively small Aboriginal communities. If the Many Farms experience is relevant, this will only replace one technological misfit with another.

In the more closely settled areas of Australia, the interposition of auxiliaries (to undertake primary health care) between the communities and the existing system may appear likely to be resisted by the private sector of the medical profession. Community health nurses, and many of the auxiliaries they would supervise, are quite capable of performing vaccinations, diagnosing and treating a wide range of minor and not-so-minor illnesses, carrying out haemoglobin estimations and so on. Furthermore it is essential that they undertake these tasks if they are to be accepted as multi-purpose community nurses, influence health in the non-therapeutic areas, and remain strongly motivated and happy in their jobs. To restrict such personnel to the roles of adviser in trivial matters and maker of appointments to see doctors and social workers in all other matters would make the job not only unattractive and unrewarding, but

also render it ineffective in the preventive role and waste resources. A fully effective primary health care system will therefore inevitably draw off a proportion of the traditional 'bread-and-butter' income of private medical practitioners, but in the case of the Aboriginal communities in southern Australia the income derived from treating Aborigines is not great, and it is fairly certain that the system would increase rather than decrease the presently minimal contact between Aborigines and doctors in private practice once the overall health delivery system becomes properly integrated, and liaison with private practitioners is facilitated. The same may be less true of private practices serving non-Aboriginal communities.

The argument is therefore advanced that the initiation of primary health care at the domiciliary level, in Aboriginal communities anywhere in Australia, is unlikely to be resisted by Aborigines or by the medical profession if it is initiated *now*. There may be political and social repercussions if the non-Aboriginal population sees such a move as discriminating in favour of Aborigines, but it may be pointed out that the initiation of domiciliary health care in the way suggested is a valid, valuable, and ethical social experiment which is certain to bring benefits, in time, to the Australian population as a whole. A prolonged delay in establishing such a system on a rational footing is believed by the writer to be a factor which may eventually create a worse Aboriginal health situation than now exists—partly because of the non-implementation of programs which can counter both the effects and the extent of the population explosion among impoverished Aboriginal communities, and partly because the delay will encourage attempts to solve Aboriginal community health problems with sophisticated, expensive but basically ineffective therapeutic technology. Meanwhile, the upsurge in Aboriginal numbers, morale, and pride of culture, if not carefully balanced by an increase in opportunities for Aborigines and tolerance by non-Aborigines, may threaten Australian society with a real 'culture of poverty' deeply entrenched in large Aboriginal rural enclaves and metropolitan ghettoes. The enormous difficulties in solving any sort of community health problem in the ghetto situation is attested by experiences in the United States.

In an earlier chapter, the serious and wide reaching consequences of unrestrained Aboriginal fertility were considered. The consequences appear to have been chiefly visited upon the Aborigines themselves. The present Aboriginal population is still a small minority in Australia. It will still be a small minority at the end of this century even if the high natural increase rate relative to non-Aborigines is maintained—it will be some-

thing like 2 per cent, double the present figure of around 1 per cent. But if Aboriginal morbidity and mortality levels remain substantially unchanged relative to those for Australia, or change only in the relative importance of individual causes, the deaths and diseases contributed by Aborigines will be twice as common in the total population. The estimates will become, for instance, that Aborigines will contribute 4 per cent of the births, 20 per cent of the infant deaths, over 50 per cent of deaths in the second year of life, and around 20 per cent of deaths in the remaining years of early childhood. It is likely that all the medical technology that could be brought to bear now, or may be developed in the next decade or so, will be powerless in itself to prevent these figures eventuating.

Medical and immunisation techniques are not to be discounted as powerful weapons for bringing about improvements in certain specified and important disease problems responsible for considerable morbidity, provided that the techniques can be brought into contact with the affected individuals. Tuberculosis, leprosy, worm infestations, trachoma, venereal disease and many other diseases are susceptible to a variable degree to the mass campaign techniques in disease control, and even when such diseases cannot be eradicated with drugs—as with trachoma or worm infestations —mass campaigns may be expected to reduce the prevalence, and the incapacity and permanent sequelae resulting. However, the mass campaign approach requires a 'captive' population, or at least a population which is fairly easily defined geographically, in groups large enough to justify the mounting of diagnostic, immunising or therapeutic teams. It also requires ready co-operation from the target population. Mass campaigns are an easy matter in the institutionalised segment of the full-blood population of the north and centre of Australia, less easy but still not exceptionally difficult in the part-Aboriginal settlements and reserves and some easily-definable fringe communities. They become much more difficult among the more mobile and less 'visible' part-Aboriginal populations in the cities, and in any situation where Aborigines are scattered within non-Aboriginal populations or living as isolated families in rural areas. Increasing mobility of Aboriginal individuals and families, and emigration of Aborigines away from the supervised communities may be expected in the next few decades, increasing the difficulties of tracing newly diagnosed cases and their contacts. High mobility itself may also help to spread some diseases as fast or faster than new cases can be detected. This problem is, of course, another argument favouring the development of a health care system in which primary health care personnel can remain in close personal contact with community members and, when

necessary, co-ordinate activities in health programs across district and State boundaries.

The other major problem which needs to be solved if the Aboriginal health situation is to be improved is that of data acquisition. In an earlier chapter the case was made for an examination of the system of acquiring data through the identification of Aborigines and part-Aborigines—any self-identifying Aborigines—in official records of birth, death, illness, hospitalisation, etc. Although administratively simple, this may prove to be politically impossible if there is any objection on the part of Aborigines to identifying themselves as such in official records. The alternative is to employ the primary health care system as the source of health data—a method which is used extensively in many non-Western countries with an organised health service. It depends on the community health worker, or surveillance agent, or whatever the person in direct contact with the community is called, maintaining sufficiently close contact with all households in his or her allotted sector of the community to be able to detect and record all births, deaths, and significant illnesses as they occur. At first sight it might be thought that the quality of health data may suffer if the diagnosis (a cause of death, a cause of illness or injury, or their results) is obtained from a householder rather than from the medical attendant; this would certainly pose some problems in evaluating the impact of the more obscure diseases, or in cases where fine distinctions need to be drawn between two similar illnesses which cannot be distinguished by the layman. However, while there may be a loss of technical quality in this way, the loss would be compensated for by a gain in social quality—partly because of better coverage, partly because the data obtained relate to health and illness as it is perceived or experienced by the community, in the community's own terms. In countries where a highly-developed domiciliary or community-based primary health system operates—as in many parts of India—the demographic parameters established are usually more accurate and comprehensive than those obtained in national censuses. This is particularly so where the primary health care system has evolved from programs to control or eradicate specific diseases such as malaria, smallpox, tuberculosis, or leprosy, and where accurate surveillance has been essential to epidemiological and operational control of a program. As the disease which was the primary target of the program is brought under control, the field personnel enlarge the scope of their activities to progressively include diseases with lower priorities, but maintain the same sort of close contact at the domiciliary level.

The point is simply that the problems in Aboriginal health, and the associated demographic, social, and economic problems, cannot be treated effectively unless they are monitored, and they cannot be monitored effectively without a continuous feed-back of relevant data. Both control and prognosis for Aboriginal health may be expected to improve markedly as soon as an efficient monitoring system is functioning. There is also a side-effect of a monitoring system which is likely to benefit all parties—Aborigines, health services, non-Aboriginal society, and governments: this is the morale-boosting effect of simply knowing where one stands, and being able to observe even quite minor improvements or gains. It could be alleged that Australian authorities at all levels who are responsible for Aboriginal affairs are—and have been—excessively 'cagey' about 'the Aboriginal problem'. The writer would argue that this 'caginess' is partly due to embarrassment at not being able to give an accurate picture of present conditions, and partly due to fear that they will be unable to show any benefit from past or present endeavours. Thus, while there is little doubt that the overall picture of Aboriginal health is improving slowly in most areas, the parties who could gain some credit for the improvements are unable to prove their claims. By contrast, the authorities in the United States and New Zealand are prepared to publish openly the details of the worst aspects of Indian or Maori health, but at the same time are able to show gains made and the effectiveness of measures formulated. Even when things go badly in certain areas of health, the successes make it impossible to condemn the authorities out of hand for being ineffective. Unfortunately, and unfairly, this is not so in Australia. So another important requirement for the future is that Aboriginal health statistics and other observations should not only be collected by the responsible authorities or government departments, but should be published regularly and openly. To do this, a political decision must be made, rather than a departmental decision. This study has been published with the approval of the Commonwealth Department of Health, and much of the data have been made available to the author by various Commonwealth and State Departments and published with their approval. That a study which contains much material that could be used for political ammunition has been published with the consent of potential political targets may be seen as a source of great hope for the future for Aboriginal health.

There is an urgency for mounting a carefully planned, co-ordinated and radically conceived program for the improvement of Aboriginal health. The urgency is not to produce quick and spectacular results, for

these are not possible for a problem which has widespread roots deep in two very different cultures. The urgency is to bring the situation under control, even to a limited degree, while the population with the problems remains relatively small and accessible. The aim is to reduce and then to remove the physical and mental handicaps to physical well-being, educability, employability and sociability either within or outside white Australian society—the choice should be open—and in doing so restore something of the quality of life which one feels was appreciated by Aborigines in their traditional environment. The traditional environment has gone for ever.

APPENDIX I

GROWTH INDICES

For children of a given age and sex, growth indices such as height and weight are distributed above and below the average (mean) for the group. The frequency with which the individual values fall at increasing distances above or below the mean closely follows the 'normal' frequency distribution curve—both for a population being surveyed and for the population on which a 'standard' is based. The range of normal variation above and below the mean may be expressed in terms of the 'standard deviation'[1] or in terms of 'centiles'. The standard deviation provides that, if the values are 'normally' distributed, 68·3 per cent of observations will lie within a distance of one standard deviation above or below the mean, and 95·5 per cent of values will lie within a distance of two standard deviations of the mean, etc. About one normal value in forty-four (2·25 per cent) would be expected to be less than the mean minus two standard deviations. Both the means and the standard deviations increase with age—as height and weight increase, the normal range of values becomes wider. The increase is not a straight line rise, being steeper in the first two years for weight and height, and again from about nine to after puberty for weight.

This variation may also be described in terms of 'percentiles' (also called 'centiles'), and because true percentile values are not based on an assumed 'normal' distribution (in which there are equal numbers and ranges above and below the mean) they would be preferred in circumstances where the distribution of a standard population's growth parameters are skewed—say with high values further above the mean than low values are below it.

'Percentile' values in some height/weight standards—for example, Tanner's English height standards—have been calculated from the standard deviation assuming a 'normal' distribution, rather than being determined directly from observations, Tanner arguing that height standards in centiles are more accurately represented this way. Tables of the areas under the 'normal' curve show that the 3rd centile falls at 1·881 standard deviations below the mean, and Tanner's 3rd centile for height (by age and sex) lies at 1·881 standard deviations below the mean, and not the observed 3rd centile below the median. Weights being more noticeably skewed, Tanner's centile values for standard weights are along the smoothed centile values (3rd, 10th, 25th, etc.) determined from the raw data.

[1] A statistical term describing the average variation or 'dispersion' about the mean or average. It is the square root of the average squared difference from the mean of all the values.

A distribution described in terms of percentiles or centiles is divided into 100 layers; each layer contains 1 per cent of the population of the particular age and sex group, and is separated from the adjacent layers by corresponding percentiles. Although there are 99 such percentile values, only a selected few are usually incorporated into growth charts—typically the 3rd and 97th, the 10th and 90th, and perhaps the 25th and 75th, as well as the 50th, which is the median.

As the percentiles are limiting values for each layer, it follows that in the standard population of a given age and sex, 3 per cent of *normal* individuals will fall below the 3rd percentile, 10 per cent below the 10th percentile, and so on. The 3rd percentile is sometimes used as the lower limit of normal growth, as is the mean minus two standard deviations, but it is important to note that these are not absolute limits of normality in the individual case.

An individual can only be stated with certainty to be 'abnormally' short or light for his age if his height or weight falls outside the extreme limits—i.e. below the mean minus three standard deviations, or below the first centile, for his age and sex. However, if in a large group all or most values are below the mean or median 'standard', the *group* is abnormal. It is not possible to identify the abnormal individuals in the group, however, unless their heights or weights are below the lowest values observed in a healthy population. Generally speaking, the presence and causes of minor degrees of growth retardation can only be studied in groups of individuals, and the effects of improved nutrition or reduced infection can only be observed in groups. The statistical implications of normal variability must be appreciated before proper conclusions can be drawn from such observations.

From the foregoing, it follows that a growth parameter for a group of children of varying ages and both sexes cannot be expressed in units of weight or height. One approach is to express a growth deficit as the proportion of individuals who fall below a selected percentile value or a value expressed in 'standard deviations' from the mean, but this does not take into account the extent of the shortfall in weight or height of those identified as being below the limit set. A second approach is to average the growth deficit in terms of standard deviations or centile deviations from a standard mean or median curve. This allows the most accurate comparison between groups, but suffers from the disadvantage that the units which describe the difference are meaningless to many people, and if the average centile deficit is to be calculated one needs a table of all the standard percentile values for each sex at all ages—a table which would have at least 3,000 values. The results are more appropriately entered on a standard weight or height curve for age and sex, and interpreted visually. A graphic analysis has the additional advantage that the different growth performances at different ages may be appreciated. However, results from different surveys can only be compared if the raw height and weight data are published with the survey.

A third approach, which has been used by Jose in Queensland, is to calculate a 'growth index' which expresses the deficit in height or weight as a ratio of the age by which a 'normal' individual would have reached the observed value to the

chronological age of the individual observed. Thus a child aged 4 of 94 centimetres should have reached this height, if his growth rate was average for the standard population, by the age of 2 years. His height index is $\frac{2}{4}$ or 50 per cent. While such an index is attractive because of its simplicity and its 'understandability', it should not be used for comparisons in weight. A child following the 10th centile for weight, for instance, has a weight index of about 75 per cent at age 1, about 65 per cent at age 2, and about 80 per cent at age 11. The index based on height-age is more consistent, so that heights at the tenth centile consistently provide a height index of between 80 and 85 per cent from age 1 through to age 11.

There have been various attempts to produce an index which combines height with weight but because of the asynchronous increase in height and weight during growth such indices are not suitable for averaging children of varying age. The 'Wetzel Grid' (Wetzel, 1941) is a chart for measuring the growth of individuals in which the co-ordinates are height and weight. The grid was designed to evaluate 'physical fitness' rather than growth *per se*, and 'channels' are superimposed upon it along which children of various 'physiques' should progress in ideal circumstances. Cross-lines (at right angles to the 'channels') provide a reference scale to annual progress. The grid's main function of measuring height-weight ratios detracts somewhat from its usefulness in measuring the rate of growth as an index of nutrition, but it is a useful tool for the simultaneous visual assessment of the height-weight progress of an individual or the height-weight status of a population. It has not been used much in public health practice, probably because the original grid was copyrighted by Wetzel—a step which has made its routine use expensive and legally complicated.

For research purposes, and for monitoring trends in *groups* of growth-retarded children, the writer favours what might be called a '3rd centile index', based on the interval between the median and the third centile value for the same age and sex in a standard population (e.g. 'Tanner' or 'Harvard' standards). Populations with different age-compositions, or single age-groups may be compared and their progress followed over time with greater accuracy than is possible with indices based on the median alone (such as the percentage median weight, or the chronological/development age ratio).

The index may be calculated as follows: plot the child's weight (or height, etc.) at his exact (fractional) age on a chart showing the standard median and 3rd centile curves; with a pair of dividers (preferably) measure the plotted value's difference from the standard median for his age in the units of measurement (e.g. kilograms), assigning a minus value if it is below the standard median; also measure the median-3rd centile interval in the same units at the same fractional age (as a plus value) and divide this into the first value x 100. The result is a percentage variation above or below the median in terms of that variation beyond which growth retardation may be assumed. The same calculation may be performed on a computer programmed with the median and 3rd centile standards for age and sex as reference data, using subroutines which calculate decimal age from

dates of birth and examination, and which interpolate standard values for fractional age.

A zero result occurs if the observed weight or height is at the median. Positive percentages indicate above-median growth, negative percentages below-median growth. A result of −100 per cent is returned by a value at the 3rd centile *at any age*. Grossly retarded growth will be reflected by indices such as −140 per cent, and obesity by weight indices such as +230 per cent.

In a group of growth-retarded children followed from early to late childhood, a rise or fall in the averaged '3rd centile index' will indicate real improvement or deterioration in growth achieved, particularly with weight. The index based on age/developmental age can show an 'improvement' when in fact average weights have fallen to lower percentile levels. The index based on weight as a percentage of the median standard can 'fall' when in fact average weights have risen to higher percentile levels; the same index will exaggerate a real deterioration in achieved growth.

APPENDIX II

GENETIC DISEASES: IMPLICATIONS IN ABORIGINAL ECOLOGY

In the traditional Aboriginal society, serious dominant defects manifesting early in life would have been rapidly eliminated when and if they appeared through selection pressures operating in the absence of advanced medical and surgical technology. According to Abbie (1960) even minor visible malformations would have been eliminated because 'Aborigines destroy babies with any evidence of malformation'. A dominant defect manifesting later in life, such as Huntingdon's chorea, could have been perpetuated, but there is no evidence for its occurrence in Aborigines of 'pure' descent—the cases from Point McLeay appear to have descended from a white ancestor. Serious dominant defects may be expected to appear with increasing frequency among Aborigines as a result of successful treatment of affected parents (Aboriginal or white) and dominant defects arising from mutations may be added. However, the morbidity load from individual serious simple dominant defects is never likely to be a major contributor to Aboriginal ill-health until therapeutic correction of such abnormalities is possible on a wide basis and has continued for many generations—by which time it may be assumed that Western populations will be even more severely affected by the consequences! Huntingdon's chorea is probably the only serious exception to this prediction because it is particularly favoured by Aboriginal attitudes, behaviour, and high fertility early in life.

For recessively-inherited defects, the position is rather different. Because the majority of carriers of defective recessive genes are 'normal' (heterozygous), and the defective genes are relatively rare, selection operates very slowly and minimally against such disorders, and gene frequencies for rare recessive abnormalities remain remarkably constant in large populations. There is no reason to believe that full-blood Aborigines have not been carriers of a range of the relatively rare recessive genes which can produce serious abnormalities when inherited from both parents (i.e. in the homozygous individual), although the Aborigines' prolonged genetic isolation, and possibly small beginnings in terms of the number of 'founders', may have restricted this range when compared with the range to which the world population in general is exposed. Consanguinity of the parents increases the chances of homozygosity and thus the occurrence of a recessive abnormality in a given individual. In the traditional Aboriginal population, consanguinous marriages were the inevitable outcome of small community size or isolation of

communities, and in some tribes a moderate degree of consanguinity was predetermined by the traditional marriage system. In some modern Aboriginal communities the degree and chances of consanguinity may be further increased by a breakdown in the traditional marriage system, by promiscuity, and by increased fertility in small communities (larger but fewer families, with excess population migrating away as family groups rather than as randomly-motivated individuals). Modern Aborigines are also exposed to 'new' recessive genes introduced from non-Aboriginal populations. In other modern Aboriginal communities, the natural increase in numbers, increased mobility and the addition of immigrants from distant places will operate to decrease the likelihood of consanguinity.

It is well known that the rare recessive abnormalities tend to occur more often in remote, isolated communities. It is not so well known that it is not the frequency of closely-consanguinous marriages that is primarily responsible, but the size of the population 'isolate'. Dahlberg (1948) has demonstrated mathematically that, for very rare conditions, a prohibition on cousin-marriages can reduce the frequency of affected individuals by only 15 to 30 per cent, but doubling the size of the isolate by merging it with outsiders will reduce the frequency by 50 per cent. The isolate is not equivalent to a single community, but to the effective group within which marriages are contracted. It is not possible to say whether the average isolate in these terms was larger or smaller in the pre-contact Aboriginal population. On the average, it was probably larger, for many modern communities are very small indeed.

It is also apparent that very rare recessive genes will be entirely absent from some small isolates, and relatively common in others. Where a rare gene is present, small community size and inbreeding will produce marked fluctuations in local gene frequency, so that there will be a tendency for particular recessive genes and affected offspring to become sequestered in particular isolates, and temporarily within lineages within those isolates. Thus the expected pattern for serious recessive defects in the Aboriginal population is for them to recur in particular small isolates, and as 'epidemics' in particular lineages. As the isolates are broken by intermarriage, the effects of disseminating the defective gene will be more than counterbalanced by a reduction in the frequency of homozygous individuals in the 'new' isolate. Very small isolates, where fluctuations in gene frequency are more marked, have an excellent chance of losing a defective gene altogether, so they have some advantages in the long term although they will periodically suffer a higher incidence of recessive defects in their children.

The Mendelinan laws of inheritance, at the community level, operate to cancel out the genetic advantages and disadvantages arising from ecological, behavioural, and demographic changes. This is a long-term process because genetic stabilising influences can only operate in steps a generation apart. Genetic stability depends ultimately on the laws of probability—like the inevitable pattern of results obtained from repeated coin-tossing or tossing a large number of coins

at the same 'throw'. There are only two factors that can alter the frequency of harmful recessive genes in a large population—selection,[1] which reduces the frequency, and mutation, which raises it. The balance between mutation and the effects of selection determines the gene frequency. Mutation is evidently a rare event, and selection operates minimally against rare disorders, as it does against the milder abnormalities even if common.

The genetic constitution of the Aboriginal population is not as relevant to inherited disease as the genetic constitution of Aboriginal population isolates. Two additional factors can influence the genetic constitution of these isolates—random fluctuations from generation to generation arising from small numbers (genetic drift), and introduction of 'new' genes from outside (genetic diffusion). The first can be likened to the effects of limiting the number of coins tossed and the second to the occasional introduction of coins with a different design on one side.

With recessive abnormalities, the frequency of affected (homozygous) individuals in a population is determined by the gene frequency *and* by the size of the isolate, the frequency of consanguinity, and assortative (or preferential) mating. The distinction between selection and preferential mating is a fine one as it separates biological and behavioural factors which tend to have the same result. The important difference is that selection occurs independently of the influence of attitudes and values, as a biological phenomenon beyond human control, while preferential mating is very much influenced by attitudes and values, and preferences can change with the passage of time or the influence of another culture. It is apparent that individuals with severe hereditary defects may fail to reproduce their 'quota' of offspring either because they have reduced fertility, or because they are rejected as mates, or for both reasons together.

Aborigines, then, because they tend to live as relatively small population isolates, can be predicted to display a relatively high proportion of recessive defects, but specific defects will be limited to certain isolates and will fluctuate in incidence in these communities. The range of specific defects will be limited. As isolate-breaking continues, with increased mobility and migration, the incidence of serious defects within isolates should fall but the range of specific defects should increase—'new' disorders will appear in communities which have never experienced them before. Those communities continuing as small isolates, particularly where family size is large but the number of families is limited, should produce the majority of recessive defects but such occurrences will still be rare in the total population, and in the small isolates will still be rare in terms of time.

Recessive disorders which are complexly inherited (polyallelic) would be expected to follow the same trends under conditions of changing ecology.

In a sociological context, the point should be made that traditional and present-

[1] Selection operates through the effect on fertility produced by a genetic handicap (or advantage). Selection is total if a defect is lethal to the individual before he or she reaches reproductive age, partial if it reduces but does not preclude reproduction.

day full-blood populations have not produced any evidence of serious recessive defects (under 'inbreeding' conditions which test for their presence) which are not already present in the white Australian population. Aboriginal-white miscegenation does not therefore constitute a 'genetic disease' threat to the white Australian population—the position is the reverse, if anything, with whites benefiting and Aborigines suffering (as with Huntingdon's chorea in the Point McLeay part-Aboriginal community) from a genetic interchange.

Again in a sociological context it is relevant to examine some possible effects of genetic admixture on the Aboriginal physical type—an inheritable 'condition' which is no physical disadvantage although it has social consequences. If part-Aborigines preferentially 'marry white' (and there is evidence for this in several studies of part-Aboriginal communities), there is an unexpected possible outcome of major significance. The assortative or preferential mating involved in 'marrying white' will operate to eliminate or at least reduce the Aboriginal physical characteristics from that component of the part-Aboriginal population, with the reverse effect upon the highly fertile remainder. Incomplete tolerance on the part of whites to mixed marriages, where the threshold is determined by the degree of colour or features, may be the basic determinant of the part-Aboriginal values involved, but the effect may be the re-emergence of a darker-skinned, more 'Aboriginal'-featured minority. Conditional mixed marriage is not isolate-breaking for the whole group if it but forms new, smaller isolates based on 'wanted' and 'unwanted' physical characteristics. There are many unpredictable influences which may alter or condition this trend, but those who expect the part-Aborigines and the 'Aboriginal problem' to disappear through miscegenation are unlikely to see their expectations fulfilled until there are no marriage preferences held by *any* group—full-blood Aboriginal, part-Aboriginal, or white.

APPENDIX III

CRITERIA FOR IDENTIFICATION OF NEW SOUTH WALES ABORIGINES IN REGISTERS OF DEATH

(a) *RESIDENTS OF ABORIGINAL STATIONS*

(i) Station given as place of residence of deceased,

OR (ii) Station given as address of closely related informant (i.e. a parent in the case of a neo-natal death).

(b) *RESIDENTS OF RESERVES, TOWN COMMONS, OTHER LOCALITIES*

(i) Place of residence of deceased given as '- - - - Aboriginal Reserve',

OR (ii) Place of residence of parent of deceased, or informant in cases of neo-natal death, given as '- - - - Aboriginal Reserve'.

OR (iii) Place of residence of deceased, or closely related informant in the case of a child, given as a place known to be a reserve or locality occupied by Aborigines according to local terminology,

AND (iv) Name of deceased, *and* his/her parents, *and* his/her spouse if applicable are consistent with names of Aborigines in the area,

AND (v) Birthplace of deceased is consistent with his/her Aboriginal origins; also place of marriage if applicable.

OR (vi) Place of burial is an 'Aboriginal cemetery'.

Notwithstanding the above criteria being fulfilled, a person will be excluded if:

(a) His/her occupation or his/her father's occupation implies ownership of property or a business requiring substantial capital.

(b) He/she was born overseas or in a place inconsistent with Aboriginal social identity.

(c) He/she was married overseas or at a place inconsistent with Aboriginal social identity.

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Designed by Philippa Walker
Text set in 11 *point Bembo one point leaded and*
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