THE INSTITUTE REPORT SERIES

A MATTER OF LIFE AND DEATH

CONTEMPORARY ABORIGINAL MORTALITY

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EDITED BY ALAN GRAY

Proceedings of a workshop of the
National Centre for Epidemiology
and Population Health
held at Kioloa, New South Wales, 10-12 July 1989

ABORIGINAL STUDIES PRESS
CANBERRA
1993

FIRST PUBLISHED IN 1990 BY

Aboriginal Studies Press for the
Australian Institute of Aboriginal and Torres Strait Islander Studies
GPO Box 553, Canberra, ACT 2601.

REPRINTED 1993

NATIONAL LIBRARY OF AUSTRALIA CATALOGUING-IN-PUBLICATION DATA:

A Matter of life and death.

Bibliography.
ISSN 1030 6544.
ISBN 0 85575 219 X
[1] Aborigines, Australian—Mortality—Congresses 2 Mortality—Australia—Congresses. [3]. Aborigines, Australian—Health and hygiene—Congresses 4 Australia—Statistics, Vital—Congresses I Gray, Alan. II Australian Institute of Aboriginal and Torres Strait Islander Studies. III. Australian National University National Centre for Epidemiology and Population Health (Series Institute report series)
304.640994

DESIGNED BY Denis French, Aboriginal Studies Press.
TYPESET IN Palatino 11 on 15 by Aboriginal Studies Press.
PRINTED IN AUSTRALIA BY Panther Publishing and Printing, Fyshwick ACT

FRONT COVER

Drawings courtesy of Rev Jim Downing from the Health Education series "Charlotte and Charley" - The Story of TB
AIATSIS Pictorial Collection

500 04 93

CONTENTS

ABBREVIATIONS

AHO	Aboriginal Health Organisation
AGA	Appropriate-for-gestational-age
ABS	Australian Bureau of Statistics
AIH	Australian Institute of Health
CDC	Centres for Disease Control
DEMOSS	Demographics Statistics System
ICD-9	International Classification of Diseases (Ninth Division)
NCEPH	National Centre for Epidemiology and Population Health
NCHS	National Centre for Health Statistics
NH&MRC	National Health and Medical Research Council of Australia
SGA	Small-for-gestational-age
WHO	World Health Organization

ACKNOWLEDGEMENTS

This volume could not have been produced without the contributions from those who participated in the workshop at Kioloa in July 1989. The authors of the papers (which were the basis of most of the chapters), and the other participants whose views are recorded in the discussions, have left a lively record of deliberation on a matter of great public health policy importance in Australia. In editing this volume, my main concern has been to ensure that the sense of urgency, which participants gave to the need to inform both government authorities and Aboriginal people themselves about contemporary Aboriginal health as evident in mortality rates, will be preserved in this written record.

Noor Khalidi undertook the administrative arrangements which enabled the workshop to take place, with characteristic efficiency. I express my deep gratitude to him and to the staff of the Executive Officer of the NCEPH for ensuring that the workshop took place, that participants could attend and be accommodated, and that everything ran as smoothly as it did. I also express my gratitude to the Edith and Joy London Foundation for providing the location for the workshop and accommodation for the participants.

The contributors wish to thank the following people and organisations for their assistance *Chapter 1*—Morteza Honari for assistance in the initial preparation of the figures and Robyne Zibar for the final presentation of figures and text; *Chapter 2*—the Aboriginal Health Program, Queensland State Health Department, through which the data for this study were collected, and the NCEPH which provided funding for the study; *Chapter 3*—The Australian National University, Canberra, and the NH&MRC, which provided financial support for this and related studies; *Chapter 4*—the NH&MRC and the NCEPH for funding, the Chairman of the Cherbourg Aboriginal Council, the Queensland Department of Community Services and Ethnic Affairs and the Queensland Department of Health for permission to use these data, and Marie Muller, Sister-in-Charge of the Infant Welfare Clinic and Jan Pratt, Nursing Superintendent of the Cherbourg Hospital, for their assistance; *Chapter 5*—the Queensland Health Department's Aboriginal Health Program staff for gathering the birth and anthropometric data on Aboriginal children throughout the State over the years since the Program began; *Chapter 6*—the Northern Territory Department of Health and Community Services for granting access to the data which have been used in the analysis; *Chapter 9*—the Registrar of Births, Deaths and Marriages in Darwin and his staff at the Alice Springs Registry, who granted the researcher access to the registration records, the Tangentyere Council for their support and providing office facilities in Alice Springs, and Dan Black and Bruce Illingworth from the Aboriginal Statistics Unit, ABS, who provided population data; *Chapter 12*—the Aboriginal Health Unit of the New South Wales Department of Health, in conjunction with whom the research was undertaken, Alan Gray for his constructive guidance, our informants, staff members of the New South Wales Department of Health and the Registrar-General, Aboriginal Health Services, and Geoffrey Attenborough, Wendy Cosford, Robert McNicoll and Christabel Young for their comments; *Chapter 15*—all Australian police forces and correctional administrations, for their full cooperation in

the research project and for supplying the basic information on all Aboriginal and non-Aboriginal deaths that had occurred in police or prison custody over the nine year period, and for supplementing or clarifying the information supplied, Joseph Reser of the School of Behavioural Sciences at the James Cook University of North Queensland, who was a visiting scholar attached to the Research Unit of the Royal Commission on Aboriginal Deaths in Custody for a three month period in early 1989 and made numerous valuable comments on the early drafts of this chapter, and Stella Pantelidis, who handled the layout of the text and statistical tables with care and skill

Pre-publication production of this volume was undertaken by the NCEPH Publications Officer, Stefanie Pearce, and I thank her deeply for the thoughtful assistance and cheerful energy which she lent to the task The substantial job of transcription of audio tapes of discussions was undertaken by Maureen Lawrentin, Maria Morton, Dianne Archer and Noor Khalidi. Processing of all of the final draft was done by Claire Hinton. For all their work I thank them warmly Finally, I thank NCEPH's Director, Bob Douglas, for the strength of the support which he has given to this project

ALAN GRAY
NCEPH
MAY 1990

FOREWORD
Opening Address

Some of the participants in this workshop also attended the 1973 seminar held at Monash University with the theme 'Better Health for Aborigines?' I think John Stuart and Gordon Briscoe were at that workshop. Sixteen years later we know that there have been improvements in some aspects of Aboriginal health, particularly in those aspects of maternal and child health where it was possible to place effective programs of medical intervention. In the early 1970s, infant mortality rates of 100 or more per 1,000 live births were not uncommon—now we see rates of maybe 25 to 35 per 1,000, still three times as high as in the rest of the Australian population, but a vast improvement nevertheless. There have also been some very effective public health interventions, such as the National Trachoma and Eye Health Program which began in the 1970s and lasted well into the 1980s.

There have been massive improvements in the extent to which Aboriginal people have been involved in their own health care, and it is sad to reflect that these improvements had to be fought for all along the way—and they still have to be fought for, despite the strength and vigour which the Aboriginal health service movement has picked up as it has matured. We are pleased that Aboriginal people from organisations involved in community-based services or government health services are present here.

Despite the improvements of the last 16 years, looking back at what was said and what was proposed at that landmark conference, it is remarkable that so many of the same hazards to the health development of Aboriginal people persist with so little change after so many good intentions have been expressed over the years. Indeed, with new forms of substance abuse—petrol sniffing in the centre and narcotics in the southeast, and the phenomenon of Aboriginal deaths in custody, it could even seem that the impediments to Aboriginal health development are multiplying.

Certainly, there was optimism in 1973, when the Australian Government adopted an official policy of improving the standard of Aboriginal health to the standard of other Australians within 10 years. As 1983 came and went, change in the level of optimism could be measured by the way in which the target for Aboriginal health equality was put further away in the future—to the year 2000. The recent report of the National Aboriginal Health Strategy Working Party puts some targets further away again, up to 25 years in the future.

Is this the evidence of optimism fading away? Or is it simply realism setting in? While most of us would prefer to think of it as setting targets in a realistic way, targets which we can hope to attain if we do not lose our optimism, our enthusiasm and our commitment to improving Aboriginal health, we should be very aware that for many public officials a goal 25 years away might as well be 250 years away.

They will certainly not be around to be held to account when the target date finally arrives. In a sense it puts a framework of unreality into programs. It disguises

COMMUNIQUE

A group of experts in Aboriginal mortality from around Australia met at Kioloa on the New South Wales south coast from 10-12 July 1989 and expressed concern that despite progress in some areas, Aboriginal mortality remains unacceptably high throughout Australia, and is alarmingly high in the 25 to 50 years age group. The meeting, convened by the NCEPH at The Australian National University, compared information from studies in different parts of Australia and found that a similar pattern of Aboriginal death rates exists in all States.

Pattern of death rates The meeting heard evidence that in some age groups Aboriginal death rates were up to 10 times higher than those for the rest of the Australian population. Compared with the total Australian population, Aboriginal people have extremely high death rates in early to middle adulthood, that is from approximately age 25 to approximately age 50. A large proportion of this excess mortality is due to preventable causes, such as diseases of the circulatory system, including ischaemic heart disease and cerebrovascular disease. Accidents, violence, respiratory system disease, and communicable diseases also contribute, across all ages, to higher Aboriginal death rates. While male rates are consistently higher than female rates, both are much worse than those for the non-Aboriginal population. For both sexes life expectancy is 15 to 20 years less than for the total Australian population.

The epidemiological pattern of Aboriginal mortality in the adult years is unique and possibly contributes to the pattern of Aboriginal deaths in custody. In the opinion of the meeting, the interaction between the underlying mortality patterns and deaths in custody means that the judicial enquiry into these matters must extend its gaze well beyond prison and police walls to explain what is happening within them, and seek to understand the reasons for this extraordinary racial inequity in community death rates.

Need for Aborigines to understand the nature and magnitude of the problem Awareness of the problem of high mortality rates among Aboriginal infants and children is already widespread. A first step in addressing the problems of adult mortality, however, must be to ensure that the Australian Aboriginal community is adequately informed about the nature and magnitude of adult mortality levels. The group believes that this is a responsibility that the research community has not yet met.

Need for preventive programs The consensus of the group, which included representatives of health research institutions, government agencies and Aboriginal organisations, was that the information before it was sufficient to demonstrate the extent of health disadvantage suffered by Aborigines, and to point to some of the areas where preventive programs should be initiated.

The group recognised that the fundamental source of Aboriginal ill-health is the position of Aboriginal people in Australian society. The group recognised also that mechanisms exist for setting priorities in Aboriginal health programs which

recognise the pre-eminent right of Aboriginal people to determine their futures. Nevertheless, the meeting was able to suggest many specific areas in which basic health programs, if introduced as a matter of urgency, would have immediate impact.

One area where recent significant improvements were obvious was in perinatal and early childhood mortality. The expansion of basic maternal and child health services had resulted in a rapid reduction of mortality during the 1970s and slower improvements since then. Even here continuing problems are evident. Aboriginal stillbirth rates have not fallen much and perinatal risk levels are apparently associated with lack of adequate and acceptable antenatal care. The meeting also heard evidence that Aboriginal children who begin life nutritionally deprived do not appear ever to 'catch-up'. The meeting called for redoubling of efforts to achieve universal, adequate antenatal care for Aboriginal mothers-to-be through social (or cultural) strategies aimed at improving Aboriginal access to health services.

The group noted that the National Aboriginal Health Strategy Working Party had recently called for:

1. investigation of means of decreasing lifestyle induced diseases by reduction of substance abuse and improvement of nutrition; and

2. effective prevention and treatment programs for various conditions including diabetes, hypertension, vascular disease, respiratory disease, chlamydial diseases, cervical cancer, liver disease, kidney disease and sexually transmitted diseases (including pelvic inflammatory disease and HIV infection).

In endorsing these recommendations, the group supports the Working Party's emphasis on action programs.

Coherent research strategy needed The group emphasised that research should be undertaken within a coherent strategy and urged the NH&MRC to continue to consult with Aboriginal organisations in order to develop research priorities in Aboriginal mortality and its social context and to commission specific studies in the priority areas.

Research into Aboriginal mortality and its prevention has rightly been assigned a high priority by the NH&MRC. The development of practicable intervention strategies however, involves very complex issues which defy simplistic approaches and are closely linked to alienation, powerlessness and unemployment. Efforts should be directed to systematic research into the impact of intervention strategies in a large prospective cohort study of Aboriginal communities. The group believes that there are difficulties with intermittent cross-sectional studies which a longitudinal cohort approach can overcome.

Need for improved data systems The meeting called for improvements to the systems now available for data on Aboriginal health, particularly mortality. Much of the information available came from only a few States leading to serious problems of

coverage of Aboriginal deaths in all existing systems. Careful research by groups working independently in different States had overcome deficiencies in some of the existing data. Now, comparing the results at the workshop, all participants expressed dismay at the close similarity of the patterns and high levels of Aboriginal death rates which existed throughout the country.

The meeting called on the ABS and the AIH to pursue an active program of producing regular, accurate statistics on Aboriginal mortality with which to assess the impact of existing and future health intervention programs. State Governments have the responsibility to provide registration data identifying Aboriginal deaths and the group called on the Queensland Government to give urgent attention to this issue as it is the only State which does not identify Aboriginal deaths on death notification forms used for registration purposes. The group also felt that there was an immediate need for the ABS to conduct a comprehensive national survey of Aboriginal health and social conditions. The statistical problems in this area however will only be solved ultimately through a sustained and coordinated effort by all relevant State and Federal authorities. This effort should be orchestrated by the Australian Health Ministers Conference in cooperation with the Australian Aboriginal Affairs Council.

The health components of existing Aboriginal community data collections maintained by State, Territory and Commonwealth authorities need to be improved and the results made public. Also, the capacity of Aboriginal communities to collect data to document their own health should be strengthened. Members of the group offered to provide advice and assistance. The members of the group stressed that action needed on health problems identified in their studies did not depend on better statistics. The statistics were needed to assess progress on Aboriginal health development.

Section One

Infant and Child Mortality

1 TRENDS IN ABORIGINAL INFANT MORTALITY

Neil Thomson

The infant mortality rate (deaths in the first year of life per 1,000 live births) has been the focus of considerable attention since the documentation of rates of almost 150 infant deaths per 1,000 live births for Aborigines in the Northern Territory in the 1960s. During the 1970s there was a steady and statistically significant decline in the Aboriginal infant mortality rate (Thomson 1983).

Since 1972-74, the infant mortality rates have declined substantially for Aborigines living in the Northern Territory and in the Queensland communities, from levels around 80 infant deaths per 1,000 live births (see Table 1 and Figure 1). The best estimate for Aborigines in Western Australia around this time, produced by a special survey in 1971, was 76 infant deaths per 1,000 live births. For each State and Territory, the major decline occurred during the 1970s, and further improvements in the 1980s have been less impressive. For the South Australian Aboriginal communities of Amata, Aparawatatja, Ernabella, Indulkana-Mimili and Yalata in the northwest and west of the State, the infant mortality for 1980-82 has been estimated at 56 infant deaths per 1,000 live births (Thomson 1985a). For South Australia as a whole, in 1981 there were eight infant deaths and 274 live births, giving an infant mortality rate of 29, more similar to the rates shown in Table 1 and Figure 1. The only reliable figure relating to the other States is for selected country regions of New South Wales in 1980-81, for which the infant mortality rate was estimated as 25 infant deaths per 1,000 live births (Thomson and Smith 1985).

As shown in Figure 1, the infant mortality rate ratios have declined substantially from the initial levels, but except for the Queensland communities, the ratios of the Aboriginal rates to the rates for the total Australian population have remained fairly static since the late 1970s. For the most recent triennium, 1984-86, the rates are still between 2.3 times (Queensland communities) and 3.5 times (Northern Territory) as high as the overall Australian rates for the period.

Perinatal mortality

The key indicator of foetal outcome is the perinatal mortality rate, defined as late foetal deaths plus deaths of live-born infants within the first 28 days of life per 1,000 total births. Some caution needs to be used in comparing the figures quoted here with international figures, some of which relate only to foetal deaths plus deaths of live-born infants within the first seven days of life. Although Aboriginal perinatal mortality rates have declined substantially since the early 1970s, they have only declined at the same rate as that of the total population (see Table 2 and Figure 2).

Table 1 Infant mortality rates[1,2] by triennium for Aborigines and total Australian population, 1972-86.

Triennium	Aborigines		Total population	
	Queensland3 communities	Western Australia	Northern Territory	Australia
1972-74	78.4 (4.8)	-	74.3 (4.5)	16.5
1975-77	58.3 (4.8)	-	59.0 (4.9)	12.1
1978-80	33.9 (3.0)	27.6 (2.4)	43.0 (3.8)	11.4
1981-83	29.4 (2.9)	23.1 (2.3)	35.4 (3.5)	10.0
1984-86	21.3 (2.3)	25.0 (2.7)	32.5 (3.5)	9.3

NOTES: 1. Rates are infant deaths per 1,000 live births.
2. Numbers in parentheses are the Aboriginal:total population rate ratios.
3. The Queensland data apply to a variable number of Aboriginal reserve communities, including Aurukun, Bamaga, Bloomfield River, Cherbourg, Doomadgee, Edward River, Hopevale, Kowanyama, Lockhart River, Mornington Island, Palm Island, Weipa South, Woorabinda and Yarrabah, and recently, Camooweal, Cowal Creek, Thursday Island and Weipa North.

SOURCE: Thomson, Honari and d'Espaignet in press.

Figure 1 Infant mortality rates by trienniuum for Aborigines and total Australian population, 1972-86.

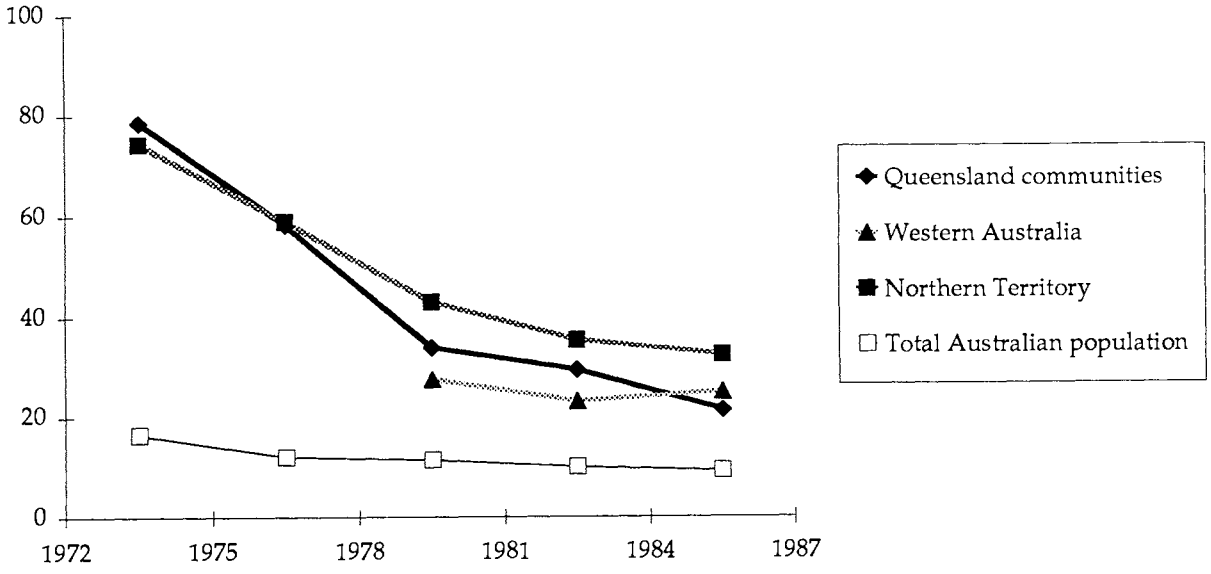


Table 2 Perinatal mortality rates[1,2] by triennium for Aborigines and total Australian population, 1972-86.

Triennium	Aborigines		Total population	
	Queensland3 communities	Western Australia	Northern Territory	Australia
1972-74	63.3 (2.7)	-	60.3 (2.6)	23.3
1975-77	49.8 (2.6)	-	58.7 (3.0)	19.4
1978-80	36.1 (2.4)	26.9 (1.8)	48.9 (3.2)	15.1
1981-83	26.1 (2.0)	28.0 (2.2)	43.1 (3.3)	12.9
1984-86	29.4 (2.5)	23.6 (2.0)	41.7 (3.6)	11.7

NOTES: 1. Rates are stillbirths plus neonatal deaths per 1,000 total births (live births plus stillbirths).
2. Numbers in parentheses are the Aboriginal:total population rate ratios.
3. See Note 3, Table 1.

SOURCE: Thomson, Honari and d'Espaignet in press.

Figure 2 Perinatal mortality rates by triennium for Aborigines and total Australian population, 1972-86.

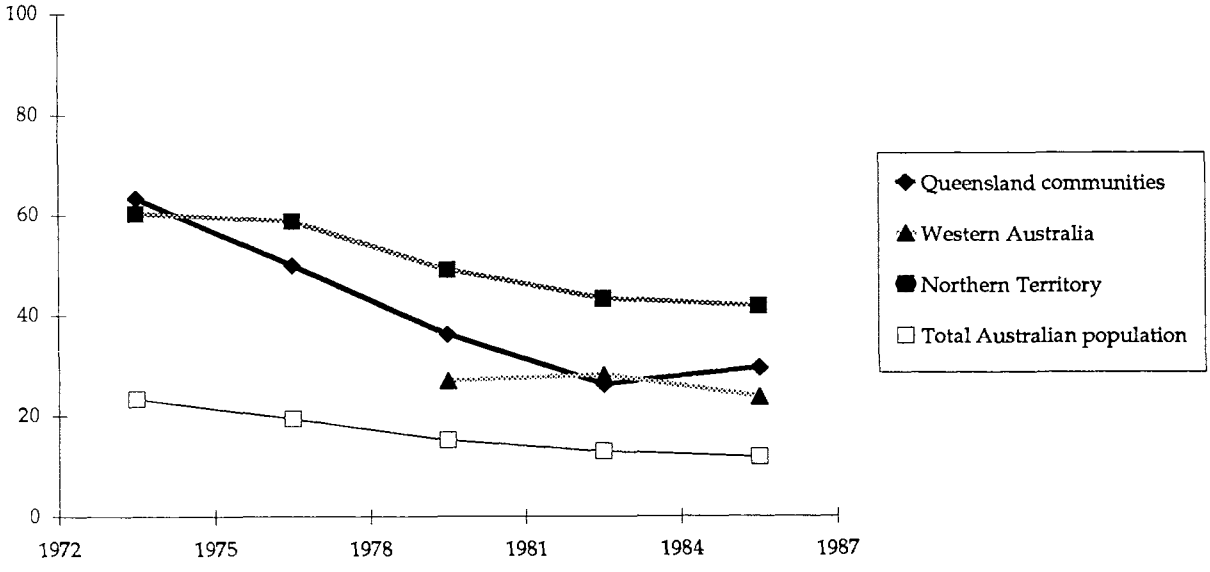


Table 3 Stillbirth rates[1,2] by triennium Aborigines and total Australian population, 1972-86.

Triennium	Aborigines		Total Population	
	Queensland[3] communities	Western Australia	Northern Territory	Australia
1972-74	30.8 (2.7)	-	26.7 (2.3)	11.6
1975-77	28.3 (2.8)	18.6 (1.9)	29.7 (3.0)	10.0
1978-80	23.1 (2.9)	12.7 (1.6)	28.7 (3.6)	7.9
1981-83	17.7 (2.5)	15.3 (2.2)	28.4 (4.0)	7.0
1984-86	22.7 (3.5)	12.5 (2.0)	24.0 (3.8)	6.4

NOTES: 1. Rates are stillbirths (late foetal deaths) per 1,000 total births (live births plus stillbirths).
2. Numbers in parentheses are the Aboriginal:total population rate ratios.
3. See Note 3, Table 1.

SOURCE: Thomson, Honari and d'Espaignet in press.

Figure 3 Stillbirth rates by triennium for Aborigines and total Australian population, 1972-86.

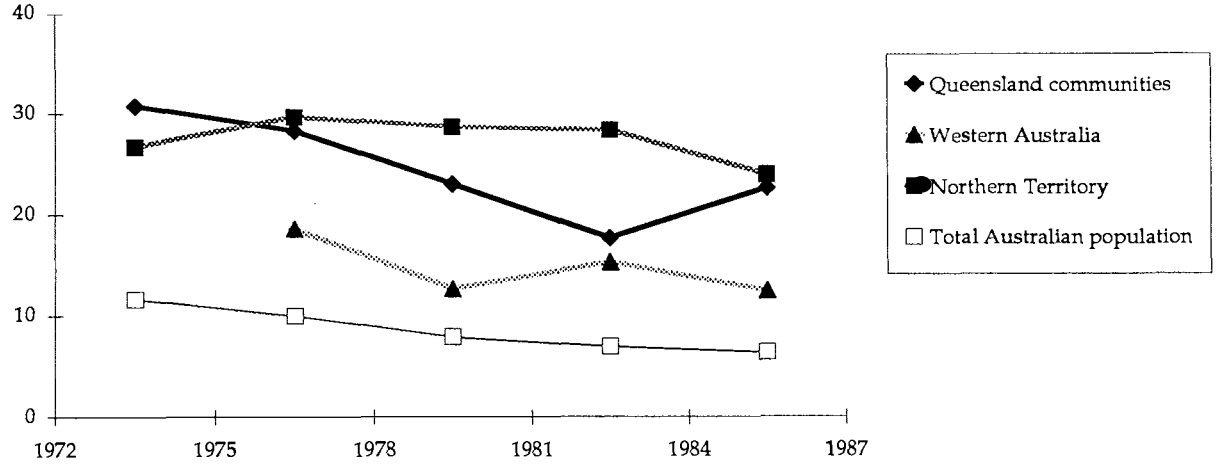


Table 4 Neonatal mortality rates[1,2] by triennium for Aborigines and total Australian population, 1972-86.

Triennium	Aborigines		Total population	
	Queensland[3] communities	Western Australia	Northern Territory	Australia
1972-74	33.6 (2.8)	-	34.4 (2.9)	11.8
1975-77	23.1 (2.4)	-	29.9 (3.1)	9.5
1978-80	13.4 (1.9)	14.1 (2.0)	20.7 (2.9)	7.2
1981-83	8.5 (1.4)	12.9 (2.2)	15.1 (2.5)	6.0
1984-86	6.8 (1.3)	11.3 (21.0)	17.8 (3.4)	5.3

NOTES: 1. Rates are neonatal deaths per 1,000 live births.
2. Numbers in parentheses are the Aboriginal:total population rate ratios.
3. See Note 3, Table 1.

SOURCE: Thomson, Honari and d'Espaignet in press.

Figure 4 Neonatal mortality rates by triennium for Aborigines and total Australian population, 1972-86.

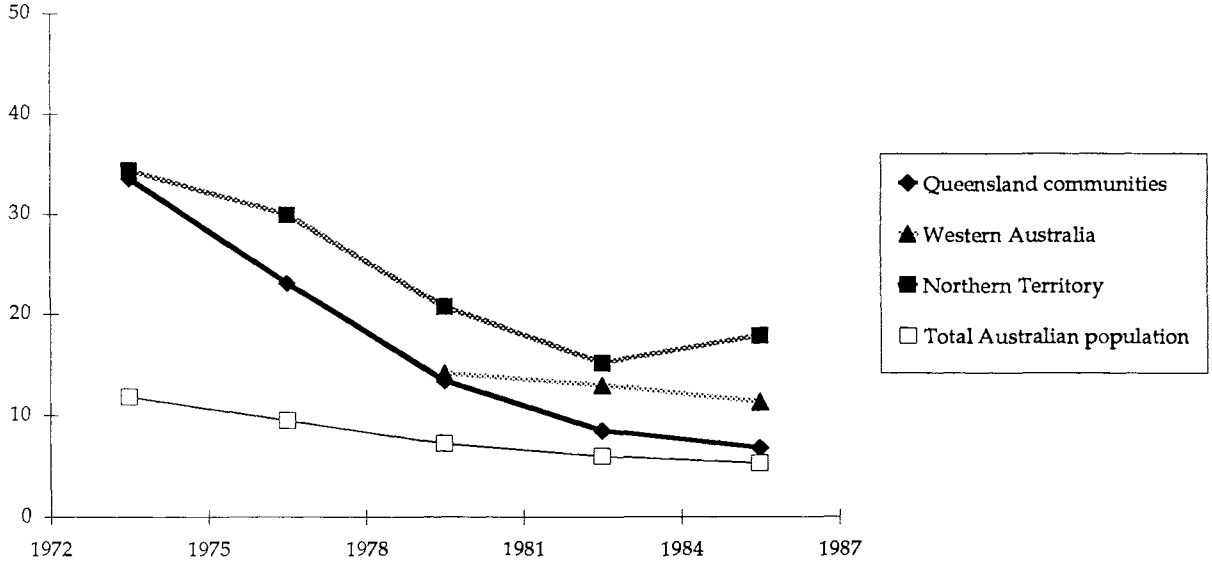


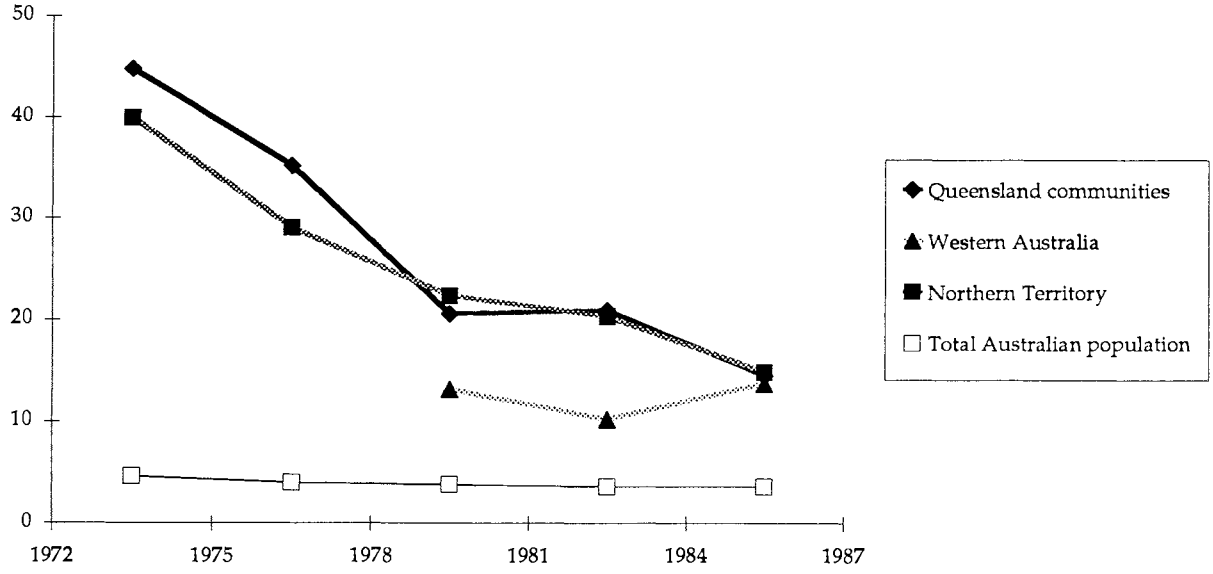
Table 5 Postneonatal mortality rates[1,2] by triennium for Aborigines and total Australian population, 1972-86.

Triennium	Aborigines		Total population	
	Queensland[3] communities	Western Australia	Northern Territory	Australia
1972-74	44.8(9.7)	-	39.9(8.7)	4.6
1975-77	35.2(8.8)	-	29.1(7.3)	4.0
1978-80	20.6(5.4)	13.1(3.4)	22.3(5.9)	3.8
1981-83	20.9(5.8)	10.2(2.8)	20.3(5.6)	3.6
1984-86	14.5(4.0)	13.7(3.8)	14.8(4.1)	3.6

NOTES: 1. Rates are postneonatal deaths per 1,000 live births.
2. Numbers in parentheses are the Aboriginal:total population rate ratios.
3. See Note 3, Table 1.

SOURCE: Thomson, Honari and d'Espaignet in press.

Figure 5 Postneonatal mortality rates by triennium for Aborigines and total Australian population, 1972-86.



In attempting to understand changes in the perinatal rate it is necessary to examine the trends in its two components, stillbirths and neonatal deaths. The rates and rate ratios for these two measures are shown in Tables 3 and 4, and the rates in Figures 3 and 4. The listed stillbirth rates provide the obvious explanation for the less substantial decline in perinatal mortality. For the Northern Territory, the stillbirth rate in 1984-86 was only 10 per cent lower than that documented in 1972-74. The decline for the Queensland communities over the same period was only 26 per cent. Of course, it is possible that better reporting of stillbirths may be counterbalancing a more substantial decline in actual stillbirths.

The Aboriginal neonatal mortality rates have declined much more impressively, parallelling similar declines occurring for the total population. These declines are probably mainly attributable to the development in the 1970s of much more effective neonatal care. The very impressive decline in reported neonatal

mortality for Aborigines living in the Queensland communities clearly warrants examination. If the recorded level is accurate, then the strategies used there should be more widely adopted. However, it is possible that there may have been some under-reporting of neonatal deaths, in that deaths occurring in regional hospitals away from the communities in which the mother lived, were overlooked (Thomson 1985a).

Postneonatal mortality

Since the early 1970s, the major component contributing to the decline in Aboriginal infant mortality has been the reduction in postneonatal deaths, those occurring between 28 days and one year of life (see Table 5 and Figure 5). The reduction in Aboriginal postneonatal deaths has occurred against a background of much less change in deaths among the total population, resulting in the steady decline in rate ratios seen in Table 5.

Conclusions

Overall, the Aboriginal infant mortality rate remains about three times higher than that of other Australians, but is relatively low by world standards, being comparable with levels in the Soviet Union, Yugoslavia and South Korea, and well below levels reported for Papua New Guinea, Indonesia and India (United Nations 1987). In contrast to overall mortality, the Aboriginal infant mortality rate is highest in the more remote areas of Australia, such as the Kimberley region of Western Australia and the Northern Territory where it is around 30 infant deaths per 1,000 live births.

The main decline in Aboriginal infant deaths since the early 1970s has occurred in the postneonatal period. In general, social factors are seen as most influential in the postneonatal period, with poverty and education both maternal and paternal being most important (Pharoah and Morris 1979; Antonovsky and Bernstein 1977; Pharoah 1976), though family size has an effect over and above poverty and education (Gortmaker 1979). However, it is unlikely that the decline in Aboriginal postneonatal deaths is mainly attributable to improvements in these socioeconomic factors. Firstly, the major decline in deaths coincided with the development in the 1970s of more intensive Aboriginal health programs and services, many of which included a major focus on maternal and child health. The programs and services also benefitted from the improved quality of care for neonates and other infants. Secondly, there have been no substantial improvements in two other measures also seen to be closely related to socioeconomic factors, the stillbirth rate and the proportion of babies of low birthweight (Gortmaker 1979).

Despite the substantial improvements that have occurred, the Aboriginal rates are still generally two to four times those of the total Australian population. Whereas the postneontatal mortality rate ratio, and consequently the infant mortality

rate ratio, has continued to decline since the 1970s, the neonatal mortality and stillbirth rate ratios have not declined. (Although the reported neonatal mortality rate for the Queensland communities has declined, as already indicated it may be an under-estimate.) A neonatal differential in this index is thought to be two-fold at the worst (Pharoah and Morris 1979; Antonovsky and Bernstein 1977).

The proportion of Aboriginal babies of low birthweight (less than 2,500 gm) no doubt contributes to the level of neonatal and, to a lesser extent, postneonatal deaths. While there is some regional variation, in recent years about 13.5 per cent of Aboriginal babies have been of low birthweight, compared with 5.7 per cent of non-Aboriginal babies (Thomson, Honari and d'Espaignet in press).

The improvements in Aboriginal infant survival in the 1970s occurred without evidence of any substantial changes in the low birthweight proportion, but it is most unlikely that further improvements will occur without a decrease in the proportion. The major factors associated with low birthweight include inadequate prenatal nutrition, alcohol consumption, maternal smoking, and lack of prenatal care, each positively correlated with low socioeconomic status. From international evidence, the socioeconomic status of the family, as measured by the mother's educational attainment, appears to be one of the most critical factors in determining birthweight (Gortmaker 1979). Thus, it appears that the levelling off of the improvements in infant survival probably reflect the real limitations of specific health and medical interventions, and that further declines will be dependent on improvements in the underlying socioeconomic status of Aborigines.

2 ARE DEMOGRAPHIC FACTORS IMPORTANT IN LOW BIRTHWEIGHT AMONG ABORIGINAL CHILDREN?

Kim Streatfield, Ric Streatfield, Miranda Korzy and IA Musgrave

It has long been established that infants born with relatively low bodyweight are at increased risk of infant mortality, and especially perinatal mortality, failure to thrive, and 'slow learner' problems (Stanley and Hobbs 1981, 370; Puffer and Serrano 1973, 41). Even in well-nourished societies, there are always some low birthweight babies, due to a variety of factors including prematurity (born before full-term) and/or genetic factors. In Australia as a whole, the proportion of low birthweight births is estimated to be about six per cent, fairly typical of developed countries. This can be compared to about 20 per cent in developing countries as a group, though some of the poorer nations may have levels of 40 to 50 per cent (UNICEF 1989, 66).

The data used here are from seven Aboriginal communities in Queensland. These communities have participated in the Aboriginal Health Program of the Queensland State Health Department since it commenced in 1972. The data refer to births up to 1988. The proportion of the births which were low birthweight was almost one in five overall, ranging from one in seven to one in three among the various communities. The purpose of this study is to explore the role of certain demographic factors: maternal age, birth order and other factors such as length of gestation, which may underlie these levels of low birthweight, and the variation across communities.

Trends in birthweight

The data for the 3,135 births up to and including 1988 show an average of 18.4 per cent of births being defined as low birthweight (Table 1), with an average birthweight for all births of 2,963 gm. The trend over time has been for a fluctuation within a range of 5.8 per cent to 23.4 per cent low birthweight, and an average birthweight ranging from 2,865 gm to 3,192 gm.

These birthweights are quite similar to the average birthweights from a number of studies in Asia and Africa, where average birthweights ranged from around 2,700 gm for poor Tanzanians to 3,247 gm for upper class Bombay Indians (Ebrahim 1983, 36). The above rates are somewhat lower than average European populations, which tend to exceed 3,200 gm (Ebrahim 1983, 36). The WHO references, based on mixed populations by race and economic status in the United States have a median birthweight of 3,200 gm for girls, and 3,300 gm for boys (World Health Organization 1983, 75, 81). The interpretation of birthweight data must be undertaken

in the light of the knowledge that birthweight is also determined by the stature of the mother. In general, 'the baby of a short woman is lighter and has less vitality and a lower survival than that of a tall woman' (Ebrahim 1983, 40).

Table 1 Trends in percentages and standard deviations of LBW births and mean birthweight, 1972-88.

Year	% LBW		Mean	SD	N
	All (<2,500gm)	Very low (<2,000gm)	birthweight (gm)	(gm)	
1972	20.6	4.9	2908	541	102
1973	14.4	4.1	2931	519	97
1974	21.8	9.2	2966	637	87
1975	14.9	4.0	3047	561	101
1976	5.8	1.0	3087	493	104
1977	14.8	0.0	3132	546	88
1978	16.9	2.8	2969	601	142
1979	23.4	5.5	2974	665	145
1980	20.8	7.7	2920	608	130
1981	21.3	7.1	2896	623	155
1982	17.2	6.1	3006	662	163
1983	18.8	7.3	2950	654	165
1984	19.4	8.1	2954	675	160
1985	18.2	6.8	2930	645	176
1986	17.3	6.8	2929	626	162
1987	19.8	6.6	2926	622	182
1988	19.0	7.1	2865	558	42
Total	18.4	5.5	2963	606	3135

NOTE: In this and subsequent tables, 'LBW' means low birthweight, 'SD' means standard deviation and 'N' means number or total.

Mother's age at time of child's birth

The reasons for the observed levels of low birthweight infants are of considerable importance. Childbearing at an early age is believed to be one such factor. In fact it is now generally believed that the major risk of producing a low birthweight infant occurs within two years of menarche (Ebrahim 1983, 41). As infant mortality patterns tend to indicate that births to teenage mothers are at greater risk than births to older mothers (Rutstein 1983), it may be postulated that low birthweight levels would be higher among teenage mothers than among mothers aged between 20 and 35 years.

The data in Table 2 indicate a generally positive relation between mother's age and birthweight with the infants of mothers under age 20 being, at about 2,800 gm, up to one-quarter of a kilogram less than those of mothers aged 30 and over. In terms of the proportion of births falling below a particular birthweight threshold, the younger mothers under age 20 are at increased risk of producing low birthweight babies (22.6 per cent), and especially very low birthweight infants (7.0 per cent).

The relatively high rates of low birthweight among teenage mothers are important because they account for almost one in three births (29.5 per cent). This pattern of high fertility among young mothers was noted by Seward and Stanley (1981, 81) who found that 29 per cent of Aboriginal births in their Western Australian sample for the years 1975 to 1978 occurred to teenage mothers compared to seven per cent for Caucasian teenage mothers.

In his review of 144 studies concerning this issue, Kramer (1985, 41) found that the largest reviewed study with sound methodology did not detect any independent age effect in a sample size of 31,604. In Table 2, the mean birthweights in several of the consecutive age categories (between age groups <20 and 20 to 24, and age groups 25 to 29 and 30 to 34) are significantly different at the five per cent level of statistical significance.

The pattern of birthweight according to mother's age is also affected by the changing birth order distribution of the births. For teenage mothers the majority of births are, of course, first births, while the births to older mothers tend to be higher birth orders. This birth order effect can be removed by standardisation of the mean birthweights for each age group, as shown in column 4 of Table 2, reflecting what the mean birthweight would be for each maternal age group if the mothers in those groups were having births of the same distribution of birth orders. The standardised age pattern is somewhat different, peaking for mothers in their 20s and being lowest for mothers over 35 years of age. After standardising for differences in birth order distributions, there are still significant differences in mean birthweights (between age groups <20 and 20 to 24, and age groups 30 to 34 and 35+).

There is a well-documented relation between mothers weight and height and the birthweight of her children (Shah 1981, 53), and there is evidence that the weight of Aboriginal women increases considerably with age after puberty. A study from the Kimberley region of Western Australia indicated that Body-Mass Index, which is weight in kilograms divided by height in metres squared, increased by some 50 per cent from 18.5 for 15 to 19 year olds to 27.6 for women aged 35 and over (Rutishauser and Mackay 1986, S9). This heavier maternal weight may result in heavier infants. The cause of the weight increase however, may be important. There is some reason to believe that the onset of diabetes among some relatively young adult Aboriginal females may be leading to birthweight increases which do not necessarily reflect healthier infants. It is hoped that further analysis of this data set, and related data sets may throw some light on this issue.

Table 2 Mean birthweights, mean birthweights standardised for birth order, and percentage LBW births (<2,500 gm and <2,000 gm), by age of mother.

Mother's age (years)	Mean birthweight (gm)	SD (gm)	Birthweight standardised birth order (gm)	%LBW (<2,500gm)	(<2,000gm)	N
<20	2831\	(561)	2858\	22.6	7.0	866
20-24	2976/*	(593)	2979/*	17.3	5.8	947
25-29	3036\	(619)	2979	16.7	3.8	629
30-34	3114/*	(630)	2956\	14.0	4.2	307
35 +	3060	(592)	2746/*	13.8	3.1	193

NOTE: Asterisk and brackets indicate a significantly different pair at five per cent level of significance.

Birth order of child

In this study, birth order was recorded to mean the number in the sequence of live births the mother had delivered before and including, the index child. As birth

order is closely related to age of mother in that younger mothers tend to have lower birth order babies, it might be expected that there would be a positive relation between average birthweight and birth order. The data in Table 3 reveal such a pattern. In the case of first births, the positive relation between birth order and birthweight is much stronger than that between maternal age and birthweight: first births average about 250 gm less than higher order births, and show almost double the levels of low birthweight. The pattern of increasing mean birthweight with increasing birth order persists even after standardisation for maternal age, as shown in column 4 of Table 3.

In the previous comparison in Table 2, the large proportion of births to teenage mothers contributed to the overall high proportion of low birthweight births. High order births act in the opposite direction, because they are associated with higher average birthweight. For example, almost one in four births are birth order five or greater, and these have an average birthweight in excess of 3.1 kg compared to less than 2.9 kg for first and second births. This high parity pattern is similar to that observed by Seward and Stanley (1981, 81), who found that 20 per cent of Aboriginal births were birth order five or more, compared to three per cent of Caucasian mothers.

This issue is worth pursuing a little further as one obvious policy implication of increased risk of low birthweight among teenage births is that efforts be made to reduce the proportion of teenage births, either through promoting use of family planning, delayed marriage, or other means. What can be seen from the pattern of mean birthweight according to maternal age and birth order is that even if all teenage births were delayed until the mothers were at least 20 years of age, the mean birthweights of the 859 teenage births would be increased only by 2.1 per cent or 60 gm, and the proportion of low birthweight teenage births would be reduced from 22.6 to 22.0 per cent.

Table 3 Mean birthweights, mean birthweights standardised for mother's age, percentage LBW births (<2,500 gm and <2,000 gm) by birth order of child.

Birth order	Mean birthweight (gm)	SD (gm)	Birthweight standardised mother's age (gm)	% LBW (<2,500gm)	(<2,000gm)	N
1	2838	(557)	2829	22.5	6.4	862
2	2880	(617)	2892	22.6	8.2	607
3	2972	(598)	2913	17.5	4.8	435
4	3046	(587)	3018	15.4	4.5	337
5	3126	(627)	3126	13.2	2.2	228
6+	3128	(587)	3105	11.5	3.4	468

Gestation period

As mentioned above, there are two main reasons for low birthweight. One is retarded foetal growth, the other is when the infant is born early or prematurely (less than 37 weeks gestation), though it should be noted that the term 'premature' is also sometimes used to refer to low birthweight infants born at full-term. There are a number of reasons for premature birth (Cameron and Hofvander 1983, 3):

> They include high maternal blood pressure, acute infections, hard physical work, or multiple births. ...Premature infants are usually thin, have muscle weakness, and a tendency to low body temperature. They may have difficulty suckling. The rates for infections of various kinds, particularly in a poor unhygienic environment, are considerably higher than for full-term babies, and death rates are high.

As a foetus grows rapidly during pregnancy, especially in the latter stages in the third trimester, it stands to reason that being born even a few weeks before term may result in a weight considerably below that which would have been reached by full-term.

The data in Table 4 show a rapid increase in average birthweight according to length of gestation. The average weight is just 1.4 kg for the group of births below 32 weeks, rising to 2.9 kg at 38 weeks and 3.2 kg at 40 weeks. This pattern is roughly consistent with the observation that the maximum rate of foetal growth is between the 32nd and 38th weeks of pregnancy 'when the weight virtually doubles' (Ebrahim 1983, 37). The significance of the month between 34 and 38 weeks is well-illustrated by the decline in proportions of low birthweight births—from around four out of five at 34 weeks, to about one in six at 38 weeks, and one in 20 at 39 to 40 weeks. Thus it is vital to ensure a gestation length as close to term as possible.

Table 4 Mean birthweight, percentage LBW births (<2,500 gm and <2,000 gm) by length of gestation (weeks since last menstrual period).

Gestation (weeks)	Mean birthweight(gm)	SD (gm)	% LBW (<2,500gm)	(<2,000gm)	N
<31	1380	859	95.0	90.0	44
32	1667	352	100.0	94.4	18
33	1933	490	80.0	70.0	10
34	2034	467	80.8	53.8	26
35	2290	348	74.2	19.4	31
36	2585	436	41.4	6.0	116
37	2813	458	24.1	3.4	87
38	2903	447	16.7	1.4	216
39	3034	387	5.3	0.0	132
40	3216	480	4.6	0.1	690
41+	3311	546	3.8	0.0	27

As mentioned above, a low birthweight infant may have been born early, or it may have not developed fully for its gestational age. The second type of low birthweight baby is called a small-for-gestational-age (SGA) baby, or a growth retarded, small-for-dates, dysmature or malnourished foetus. The best-known standards of mass-for-gestational-age are those given by Lubchenko (1968) for her population of babies at Denver, Colorado. In terms of these standards, babies are considered small-for-gestational-age if they are at or below the 10th percentile of mass for gestational age—about 2,600 gm at 40 weeks, 2,400 gm at 38 weeks, and 2,000 at 36, 1,750 at 35, 1,500 at 34, 1,400 at 33, 1,250 at 32, 1,125 gm at 31 weeks (see Philpott et al 1978, 46). More recently, the Australian Commonwealth Department of Health has developed a series of intra-uterine growth charts which take account of various factors known to affect birthweight. These factors include sex of the infant, maternal height, and parity of the

birth, but they are not presented separately by race (Commonwealth Department of Health 1985).

The infants born to Aboriginal mothers in the present study, assuming they are of medium height, fall between the 10th and the 50th percentiles of mass-for-gestational-age. With length of gestation being so powerfully related to birthweight, it might be hypothesised that the lower average birthweights associated with young maternal age and low parity might operate through higher rates of prematurity, that is, shorter gestation periods.

The teenage births are more likely to be SGA than births to older mothers, but these excess SGA births tend to be full-term, reflecting growth retardation in utero, as shown in Table 5. On the other hand, the excess levels of premature births to teenage mothers and older mothers tend to be appropriate for gestational age. For the teenage mothers it seems the low birthweight problem is a matter of not being able to deal with the absolute weight of the foetus, rather than being a matter of simple growth retardation.

Functional consequences of low birthweight

There are questions about the relation between an infant being born of low birthweight and its various body functions such as respiratory capacity and heart rate. The most widely used method of assessing the physical condition of a newborn is the scoring approach introduced by Apgar in the 1950s (Apgar 1953). It is a score from zero to 10 based on adding individual scores between zero and two for each of the five criteria of heart rate, respiratory effort, muscle tone, reflex irritability and colour of the infant. The scores are evaluated at one minute after delivery, then again at five minutes and sometimes at 10 minutes. Apgar scores of seven to 10 indicate no depression of function, scores from four to six indicate moderate depression, and scores below four indicates severe depression. There has been some disagreement about the value and interpretation of the Apgar score (see Gray 1987a, 11), but as the data are available here they will be examined.

The data in Table 6 show a clear positive relation between birthweight and Apgar scores at one and five minutes, though the threshold appears to be for birthweights below 2,000 gm. This pattern is similar to that noted by Gray (1987a, 10), though the levels here are lower than in his study area in New South Wales. The figures in Table 7 show the expected pattern of lower Apgar score for very young mothers under age 15 years, though infants of mothers aged 15 to 19 years have scores about average. The first-born infants also show a slightly lower score than later births, and conversely have a high proportion of births with moderate or severe depression of function. These patterns are consistent with patterns of low birthweights which have been shown to be closely associated with Apgar score.

Table 5 Proportions of births preterm (<37 weeks gestation), full-term (37 weeks or more), appropriate-for-gestational-age (AGA), and small-for-gestational-age (SGA) by age of mother and birth order.

Age of mother			Birth order		
Preterm <37 weeks	Full-term 37+ weeks		Preterm <37 weeks	Full-term 37+ weeks	
Age <20 years			First order birth		
17.7	71.1	AGA	16.0	73.0	AGA
2.9	8.3	SGA=11.1%	2.4	8.6	SGA=11.0%
Age 20 to 24 years			Second order birth		
13.7	78.6	AGA	16.8	73.7	AGA
1.9	5.8	SGA=7.7%	1.8	7.7	SGA=9.5%
Age 25 to 29 years			Third order birth		
14.2	77.9	AGA	11.9	79.5	AGA
1.6	6.3	SGA=7.9%	2.7	5.9	SGA=9.6%
Age 30 to 34 years			Fourth order births		
14.0	77.6	AGA	15.9	79.1	AGA
0.9	7.5	SGA=8.4%	1.5	3.7	SGA=5.2%
Age 35 years or more			Fifth order birth		
18.2	74.6	AGA	15.9	78.6	AGA
1.8	5.5	SGA=7.3%	5.0	5.5	SGA=10.5%

Table 6 Mean Apgar scores with standard deviations at one and five minutes and percentage of births scoring less than seven ('moderate depression') at one minute by birthweight.

Birthweight (gm)	Apgar 1 minute Mean	(SD)	%<7	Apgar 5 minutes Mean	(SD)	%<7	N
<1499	5.9	(2.3)	52.6	7.4	(2.5)	46.7	48
1500-1999	5.4	(2.7)	63.9	7.5	(2.2)	26.5	76
2000-2499	7.8	(2.0)	17.5	9.2	(1.6)	5.7	283
2500-2999	7.7	(2.0)	22.8	9.4	(1.1)	2.5	621
3000-3499	7.6	(2.2)	24.4	9.3	(1.2)	4.1	660
3500-3999	7.6	(2.0)	24.8	9.3	(1.3)	5.8	276
4000+	6.8	(2.5)	34.7	9.3	(1.1)	4.3	135

NOTE: An Apgar score of less than seven indicates moderate or severe depression of physiological function.

Differences across communities

The seven communities in this study vary considerably in terms of distance from urban centres, as well as environmental conditions. The variation in average birthweight is greater than 250 gm, while the proportions of low birthweight range from 14.2 to 33.3 per cent, as shown in Table 8. There is considerable variation in the levels of preterm and SGA births among the communities, though the levels do not always accord closely with the level of low birthweight. Communities A and B have relatively high levels of preterm births but average levels of SGA births. Community F, by comparison, has a similarly high level of preterm births and a very high level of SGA births. Most of these SGA births are full-term births, reflecting a serious problem of intra-uterine growth retardation. Indeed the proportion of full-term SGA births in community F is more than twice the average, and about three times the level in community A. The patterns of childbearing across the communities do not vary sufficiently to explain these marked differentials in birthweight, thus the causes cannot be explained by data available here.

Table 7 Mean scores and percentage of births scoring less than seven by age of mother and birth order.

	Apgar 1 minute			Apgar 5 minutes			N
	Mean	(SD)	%<7	Mean	(SD)	%<7	
Age of mother							
<14	6.5	(2.6)	38.5	8.8	(1.6)	17.9	33
15-19	7.5	(2.1)	26.2	9.2	(1.3)	6.0	534
20-24	7.6	(2.0)	23.9	9.3	(1.2)	3.9	640
25-29	7.5	(2.3)	24.4	9.3	(1.5)	6.3	499
30-34	7.6	(2.1)	23.6	9.3	(1.4)	4.8	255
35-39	7.5	(2.5)	26.5	9.0	(1.8)	6.5	129
40+	7.2	(2.7)	33.3	9.6	(0.9)	0.0	46
Birth order							
1	7.2	(2.2)	30.1	9.1	(1.4)	7.4	585
2	7.8	(1.9)	19.9	9.4	(1.1)	3.2	440
3	7.8	(2.1)	22.5	9.4	(1.3)	5.0	339
4	7.6	(2.3)	23.6	9.1	(1.6)	4.8	273
5	7.7	(2.2)	22.1	9.3	(1.5)	6.3	200
6	7.5	(2.3)	29.4	9.2	(1.2)	2.9	159
7+	7.6	(2.3)	24.5	9.4	(1.1)	0.0	313

Table 8 Mean birthweights, proportions of LBW births, preterm births and SGA births by community.

Community	Mean birthweight	(SD)	% LBW (<2,500gm)	(<1,500gm)	% Preterm	%SGA	N
A	2957	(616)	20.5	3.6	22.2	8.1	(1124)
B	2930	(583)	23.0	1.8	21.8	9.7	(390)
C	3021	(619)	14.4	3.4	12.3	7.6	(404)
D	2945	(597)	14.2	2.3	11.9	6.0	(485)
E	2965	(575)	17.5	3.3	11.0	10.3	(349)
F	2773	(641)	33.3	8.3	20.0	20.0	(142)
G	3090	(588)	12.2	1.1	15.0	5.0	(241)
Total	2963	(606)	20.1	6.8	17.3	8.0	(3135)

NOTE: 'Preterm' means less than 37 weeks gestation and 'SGA' means small-for-gestational age.

It was seen above that premature births are more likely with first births, or births of young mothers, so the question arises of the importance of these factors of low parity and young maternal age in these communities where there are high percentages of low birthweight babies. In Table 9 it can be seen that there is a wide range of proportions of births occurring to mothers under age 20 years, but the pattern is not consistent with the patterns of low birthweight. There is a narrow range of proportions of first and second births, but the pattern does fit rather better with the pattern of low birthweight. Except possibly in community E, young maternal age does not appear to be the cause of the high levels of low birthweight but the stronger association of low birthweight is with low parity births.

Table 9 Proportion of births to mothers aged under 15 and under 20, proportion of births of order less than three or greater than six by community.

Community	% Under 15	% Under 20	% Order<3	% Order>6
A	1.0	33.2	31.5	22.9
B	2.4	29.0	40.4	10.9
C	1.8	23.9	26.0	28.1
D	1.7	21.4	36.7	16.9
E	3.5	38.5	32.3	18.7
F	3.4	20.5	45.9	18.9
G	1.9	31.2	68.6	0.6

There has been considerable interest in the question of whether or not low birthweight children, defined by the WHO as weighing less than 2,500 gm at birth, can catch up with their heavier fellows. Data to explore this question are rarely collected, though some studies suggest that they do not catch up but remain permanently relatively undernourished, following low projectile growth curves.

This appears to be the case, as the average gestation period for births to mothers of different ages increases from 37.5 weeks for those under 15 years, to 38.1 weeks for mothers aged 15 to 19 years, to 39.0 weeks for those 35 years and older. A similar pattern applies to birth order with the average gestation varying from 38.2 weeks for birth order one, 38.2 to 38.6 weeks for birth orders two to six, and 39.5 weeks for birth orders seven or more. Analysis of variance of these groups indicates differences significant at the five per cent level for both variables.

While the average gestation periods for the youngest mothers and lowest order births appear to be only slightly shorter than for older mothers and higher order births, those few weeks can account for several hundred grams in birthweight, and it is differences of this scale which distinguish the young mothers and first births from the rest. Thus it can be concluded that a substantial part of the link between young maternal age, low order births, and low birthweight operates through such births resulting from gestation periods somewhat shorter than average.

Conclusions

These data from seven Aboriginal communities in Queensland indicate a level of low birthweight fairly constant at around 3.5 times higher than is found in the general Australian population. Many of these low birthweight births are associated

with reduced physiological capacity as indicated by Apgar scores. Demographic factors associated with risk of low birthweight are a maternal age at childbearing of less than 20 years, and first order birth. It appears that both of these demographic factors are associated with low birthweight because of shorter than normal gestation periods.

While low birthweight can result from premature delivery, it can also result from less than adequate foetal development, producing a SGA baby. The comparison of birthweight by gestational age of the study children with the Australian intra-uterine growth charts, indicate quite clearly that foetal development of these Aboriginal children is up to several hundred grams below the average (50th percentile) for Caucasian children at all stages of gestation above 32 weeks.

The levels of low birthweight among the seven communities vary considerably, and it is difficult to identify the precipitating factors in each case. While the communities vary somewhat in the proportions of first births and births to young mothers, the patterns are not consistently linked to the levels of low birthweight. It does appear that there is no single factor underlying the low birthweight births common to all communities, but in some cases it is more a problem of poor intra-uterine development, in other cases, more a problem of premature delivery.

It is not suggested here that demographic variables are the only factors of importance in the low birthweight story, but there are patterns consistent with well-documented demographic patterns of infant mortality, even if the parity pattern does not fit. Other factors, about which no data are available here, include behaviours such as cigarette smoking and alcohol consumption during pregnancy. Cigarette smoking by the mothers in this study is known to be common during pregnancy, and it has been observed elsewhere that the proportion of low birthweight infants born to mothers who smoke more than 20 cigarettes per day is twice that of infants born to non-smoking mothers (Davies et al 1976, 385-87).

The incidence of low birthweight has also been observed to increase by 2.7 times among infants of mothers who consume excessive quantities of alcohol during pregnancy. The risk of low birthweight was increased by 1.8 times in mothers who only smoked, whereas it was 3.9 times as great in mothers who both smoked and consumed alcohol (Sokol et al 1980, 135-45). At this stage, data from Aboriginal communities on such behaviours are purely anecdotal and are not available to be incorporated into the analysis, but it is necessary to acknowledge their potential importance in the problem of low birthweight births.

Future analysis on this data set will follow the development of these low birthweight infants through the first few years of life. There are numerous questions remaining to be answered about whether premature, but normal-for-dates, infants are more likely to catch up to normal rates of physical development than small-for-dates infants. There are also questions regarding the relationship between birthweight of the mother, and the birthweights of her children, and how low birthweight might better be predicted, and hopefully prevented.

3 CAUSES AND HEALTH POLICY IMPLICATIONS OF LOW BIRTHWEIGHTS OF ABORIGINAL CHILDREN

AE Dugdale, IA Musgrave and K Streatfield

The birthweights of infants are important for two reasons. Clinically birthweight is one of the major determinants of the survival of the infant. Epidemiologically birthweight has been used as an indicator of the general health and welfare of the community. A health indicator should parallel some important health function and should change promptly when that function changes. To allow international comparisons, the indicator should be independent of ethnic background and to have practical value the underlying health function should be correctable. In this chapter we will show that there is a consistent difference between the birthweights and percentage low birthweight of infants of Aborigines and the general Australian population. Some of this difference seems to arise from more frequent smoking and alcohol consumption among Aboriginal women but some is also due to anthropometric factors which originated in the early life of the mother. Appropriate changes in behaviour should alter birthweights rapidly but anthropometric factors will take at least a generation to correct, so birthweights will lag behind health status.

There are many reports of the birthweights of Aboriginal and Caucasian infants in Australia. A selection of these with about 100 or more infants in the group is given in Table 1. There are several important features in the data in this table. First, the percentage of low birthweight infants (less than 2,500 gm) is consistently higher for Aborigines than for the general Australian population. Second, the percentage of low birthweight infants is greatest among Aborigines in the remote parts of Australia and moves closer to the general Australian level in urban and near-urban Aboriginal communities. Also, the mean birthweights are lowest where the percentage with low birthweight is highest, but in the limited data available, the standard deviation of the birthweights remains approximately constant.

Many of the studies have not reported the standard deviation of the birthweights, and it has been suggested (World Health Organization 1980) that a coefficient of variation of 16 per cent can be assumed. When this is done, the calculations show that the excess of low birthweight Aboriginal infants can be explained entirely by the shift to the left of the mean birthweight. This indicates that the whole community of mothers is affected rather than a small vulnerable group contained within an otherwise 'normal' population. Small changes in the mean birthweight can lead to large increases in the percentage of low birthweight infants. This can be shown using Western Australian Caucasian data (Seward and Stanley 1981) and making the assumption that birthweights are normally distributed about the mean, which is a very close approximation to the actual data. The mean of birthweights of Western Australian infants was 3,342 gm with a standard deviation of 577 gm. Using these assumptions the expected rate of low birthweight infants was 7.2

per cent. If the mean birthweight dropped 100 gm to 3,242 gm with the same standard deviation, then the rate of low birthweight infants would rise to 9.9 per cent. A further drop of 100 gm would give 13.1 per cent low birthweight infants, while a total drop of 300 gm in the mean would lead to 17.4 per cent of infants having low birthweight.

Table 1 Mean birthweights and percentages of LBW births of Aboriginal and other Australian infants by region.

Location	Year of survey	N	Mean	SD	%LBW
Aboriginal					
Elcho Island	1979-82	216	2889	485	26.0
North Queensland	1979-82	629	2889	-	22.0
East Arnhem Land	1984	177	-	-	21.0
Katherine	1984	192	-	-	19.0
Oenpelli	1976-80	99	-	-	18.0
Northern Territory	1984	1002	-	-	17.0
Darwin	1984	306	-	-	17.0
Western Australia	1978	1007	3081	629	13.0
Alice Springs	1984	321	-	-	13.0
Cherbourg (Qld)	1982-83	104	3170	566	13.0
Murray Valley	1973-83	223	3167	-	12.0
Country NSW	1981	242	3140	-	11.0
South Australia	1983	556	-	-	11.0
Urban WA	1968	100	3290	-	10.0
Cairns	1979-82	324	3200	-	9.0
Victoria	1983	261	3280	-	7.0
Other Australian					
Queensland	1965	16267	-	-	5.8
Brisbane	1974	9261	3440	425	
Western Australia	1978	18850	3342	629	6.1

Table 2 Secular changes in birthweights of Aboriginal infants by region.

Location	Year	N	Mean	SD	%LBW
Northern Territory	1958-65	392	2880	376	-
	1984	216	2889	485	26.0
Central Australia	1965-67	-	-	-	12.5
	1984	321	-	-	13.0
Cherbourg (Qld)	1952-53	110	3310	527	5.0
	1962-63	94	3258	567	9.0
	1972-73	129	3115	538	9.0
	1982-83	104	3170	566	13.0

SOURCES: Cameron and Debelle (1986); Cox (1978); Dugdale (1968; 1980); Dugdale et al (nd); Hart et al (1985); Keeping et al (1979); Morrison (1968); Rae (1985); Reid and Kerr (1983); Seward and Stanley (1981); Thomson (1985b); Watson (1984).

Throughout Australia infant mortality rates among Aboriginal people have improved (Cox 1978; Thomson 1985b). On Cherbourg settlement the infant mortality rate has dropped from about 150 per 1,000 live births to a much lower level, probably between 20 and 40 per 1,000 live births now (Dugdale 1980). Growth during infancy and childhood has also improved and is now close to international standards (Muller et al 1984; Dugdale et al nd). These changes have almost certainly been associated with an improvement in general community health. This improvement has not been reflected in the birthweights of Aboriginal infants. Table 2 shows that the percentage of low

birthweight infants and mean birthweights have not changed over a period of 20 to 30 years.

Factors influencing birthweight

There are many epidemiological studies showing which factors affect the birthweights of infants. Most studies have been done in Western countries but have used widely differing methods of reporting their findings. This makes comparisons difficult. The factors can be divided into two main groups, anthropometric and lifestyle. In addition, there are factors with doubtful or unknown mechanisms and also disease states that can affect mother and/or foetus. We first consider anthropometric factors.

There is evidence from several studies that the height of the mother affects the birthweight of her child (Jones 1978; Baird 1962; Niswander and Jackson 1974; Lechtig et al 1978). Pre-pregnancy weight also has a significant effect (Niswander and Jackson 1974; Lechtig et al 1978). Subcutaneous fat thickness and weight-for-height have varying effects (Morrison 1968; Lechtig et al 1978) but weight gain of the mother during pregnancy is significant (Picone et al 1982). We have also found that the birthweight of the mother, but not that of the father, has a large effect on the birthweight of the infant—see the next chapter in this volume. A difference of 100 gm in the mother's birthweight is associated with a change of about 25 gm in the birthweight of her infant. The mother's birthweight accounts for eight to 24 per cent of the variability in the birthweight of her infant. Most of these maternal factors are determined in childhood and cannot be altered in adult women. It is unlikely that the mechanism relating maternal and infant birthweights is direct cause-and-effect. The importance of maternal size in determining the birthweight of offspring was shown in the classical cross-breeding experiments using Shire horses and Shetland ponies (Walton and Hammond 1938) as well as in later studies in humans (Cawley et al 1954).

Food intake, alcohol consumption and cigarette smoking are the main lifestyle factors which influence birthweight. Famine lowers the mean birthweight by about 400 gm (Antonov 1947) and food supplements can help offset this decrease. However, there is a threshold limiting the benefits from food supplements. If the mother is above a relatively low plane of nutrition, food supplements do not increase the birthweight of the child (Prentice et al 1983). It is most unlikely that extra food given to Aboriginal mothers during pregnancy would increase the birthweights of their children.

Smoking is common among Aboriginal women, including pregnant women. Overseas studies have shown that smoking can reduce birthweights by 100 to 200 gm depending on the number of cigarettes smoked and the other circumstances of the study. Marijuana also appears to lower the mean birthweight (Kline et al 1987). Alcohol has a specific action on the foetus and may lead to the foetal alcohol syndrome. In addition, the intake of alcohol during pregnancy may lower the birthweights of otherwise healthy infants, although the evidence is not consistent (Little 1977; Stein and Kline 1983). Pre-eclampsia, hypertension and related disorders reduce birthweight by diminishing the mother's ability to nurture her foetus. Other

conditions such as infections with rubella and herpes virus and with syphilis slow the foetal growth by their direct action on the growing tissues.

Among the factors with unknown and mixed mechanisms is social class, which is an important determinant of birthweight (Baird 1962; Lumley et al 1985). For example, among the Caucasian population of Tasmania, the percentage of low birthweight infants rose from 4.8 per cent in families with a trade or profession to 5.9 per cent for semi-skilled and reached 7.6 per cent among the unemployed (Lumley et al 1985). The same progression has been documented in overseas studies (Thomson et al 1968). Social class is positively associated with maternal height and smoking and alcohol intake during pregnancy are also partly determined by social class. The effects of social class must operate through physiological mechanisms and are not confined to Aboriginal people.

There appears to be no clear resolution of the question whether the ethnic background of the mother is itself a determinant of birthweight, or whether the differences in birthweight can be explained by other factors already considered. In countries where different ethnic groups live under similar conditions there can be large differences in birthweight and percentages of low birthweight infants. For example in Fiji in 1975 (World Health Organization 1980), the rate of low birthweight infants with ethnic Fijian mothers was 4.5 per cent, while the rate for infants of Indian mothers was 22.9 per cent. There appear to be no studies in these groups to determine whether the differences can be explained by known factors.

It is unlikely that women of childbearing age are ignorant of the effects of cigarettes and alcohol on their health and the well-being of their foetus. Yet even those who have been exposed repeatedly to this information may still smoke and drink during pregnancy. These women use tobacco and alcohol to meet social goals or to satisfy desires which have greater immediacy for them than do potential risks to them or to their infants. The ethos of health professionals is that immediate gratification does not justify these long-term risks to self and others. By their behaviour these young women demonstrate in the most practical way that they have different priorities to the health professionals. Some may justify smoking as a way of reducing the birthweight of the infant to make labour and delivery easier.

Implications for social and health policy

The reason for concern about a large proportion of low birthweight infants lies not in the phenomenon itself, but in its effects. There are two contrary effects to be considered. A smaller infant puts less nutritional strain on the mother during pregnancy and lactation and also reduces the difficulties and risks of delivery. In some developing countries where obstetric services hardly exist, mothers deliberately try to avoid having large infants for these reasons. These factors cannot be lightly dismissed. On the other side, a smaller infant is more likely to have neonatal complications and is less likely to survive. Jones (1978) has shown that in evolutionary terms, the mean birthweight should be slightly below the birthweight with the lowest mortality. There is evidence that low birthweight does not always carry the same penalty for babies of different ethnic groups. In their study of the mortality rates of Aboriginal and

Caucasian infants, Seward and Stanley (1981) found that for infants weighing 2,501 to 4,000 gm at birth, the Aboriginal infants had a higher mortality rate than Caucasian infants (9.3 versus 6.4 per 1,000 live births). For smaller infants the trend was reversed. For infants weighing less than 2,500 gm at birth, we have calculated from their data that the death rate of Aboriginal infants was the same as for Caucasian infants weighing 100 gm more. These small Aboriginal infants are better adapted for survival than Caucasian infants of the same birthweight.

The mean birthweight and percentage of low birthweight infants are used internationally as an indicator of social well-being and health. To be useful, an indicator must parallel some important determinant of community health. It must also respond rapidly and quantitatively to changes in the determinant. If the indicator is to have positive value then there must a known and feasible action to correct the aberrant aspect of health. Birthweight data, either existing or potential, do not meet all these criteria.

The numbers of low birthweight infants can be used in planning resources for neonatal care. Policies designed to change the height and other anthropometric indicators in the mothers will not produce changes in birthweights for a generation. In Australia, unlike some African countries (Picone et al 1982), financial benefits are available to all, so absolute deprivation of food and medical care should not be major factors in low birthweight. Resources are available to all for adequate nutrition as well as antenatal and obstetric care, even though some may choose not to use the services. Studies are needed into the nutritional status of Aboriginal women to find whether this is a major factor in small birthweights of infants. Ethnic factors, if they should apply to birthweight, are not amenable to manipulation. Smoking, alcohol intake and other practices detrimental to the health of adults and infants are matters of personal choice and of priorities. They are rarely undertaken in ignorance of the possible consequences. It is important to know what influences young people to set their priorities in the way they do. The practical steps that can be taken by health authorities include the following:

1. At the most immediate level, more treatment facilities could be supplied to cope with the consequences of the problem. This has been done in North Queensland where all pregnant Aboriginal women are brought to Cairns base hospital some time before their expected date of delivery. This hospital has excellent facilities for prenatal care and a neonatal unit where small infants can receive appropriate treatment. Although this policy would appear to be merely a symptomatic response, it will detect conditions which might worsen later pregnancies and deliveries. However, it causes social disruption by taking the mother away from her family for periods up to several weeks. There is a dilemma here. Improved obstetric services in each of the remote settlements might allow women with low-risk pregnancies to be delivered near home, but there could be problems of recruiting and retaining suitably trained staff in remote areas and also of the evacuation of women with an unexpected emergency during labour.

2. At the level of secondary prevention, high-risk women and their families can be identified in their home situation. Measures to reduce risk could be strengthened. Mothers who smoke and drink heavily during pregnancy or who have infectious diseases that directly affect the infant could be counselled and treated effectively in their own environment. If necessary, high-risk mothers could be counselled to move to a main centre.

3. The underlying attitudes and problems could be modified. This is the long-term solution but there are several major difficulties in using this approach to improve the birthweights and reduce the prevalence of low birthweight infants. Some risk factors such as smoking, alcohol and complications of pregnancy, might be treated during pregnancy with some reduction of low birthweight. Others such as the birthweight of the mother and her height are determined early in the mother's life and can be reduced only by early childhood care. The recent improvements in the growth and health of Aboriginal children should give some increase in birthweights, but only a generation from now. The lifestyle habits that reduce birthweight are also difficult to change in today's adult women. Those who have worked with Aboriginal women agree that there has been an increase in the numbers smoking and drinking alcohol during their pregnancies, which must contribute to the lower birthweights of their infants. These attitudes and practices are found in some sections of the Caucasian Australian population so the same consideration should be given to women according to their need, regardless of ethnic background. It seems unlikely that most young women who are just entering the childbearing age and are now smoking and drinking will make major and permanent modifications of these habits so low birthweights due to these factors are likely to continue. Peer pressures within their own community and education which fosters self-esteem and appreciation of the value of the life and health of their infant will help, but the decision not to smoke and drink during pregnancy must finally come from individuals themselves.

Available evidence indicates that smoking lowers birthweights by 100 to 200 gm. The figures presented here indicate that if all pregnant women stopped smoking then the mean birthweight for these women might increase by 200 gm, which for some groups would bring birthweights close to the general Australian level. The calculations based on the mean and standard deviations of birthweights indicate that this would reduce the incidence of low birthweight from say 13 per cent to seven per cent. As always, rapid increases in birthweight are unlikely as changes in lifestyle occur slowly. In other more remote groups with lower mean birthweights, an increase of 200 gm would still leave the mean birthweight lower and the prevalence of low birthweight higher than in the general Australian population. It will take a generation before the birthweights and heights of mothers increase sufficiently to make up the deficit in the birthweights of their infants.

4 THE EFFECT OF THE MOTHER'S BIRTHWEIGHT ON THE BIRTHWEIGHTS OF HER CHILDREN

AE Dugdale, J Hendrikz, IA Musgrave and K Streatfield

The pattern of birthweights is widely used as an indicator of the overall well-being of populations as it is influenced by the health and nutrition of mothers. This involves a secondary but unproven assumption that birthweights respond rapidly to changes in the welfare of the mothers and the community. We have data that suggests that this assumption may not be valid.

The means and medians of the birthweights of Australian Aboriginal infants are lower than those of the general Australian population and the percentage of births below 2,500 gm is greater. In spite of improvements in the growth, health and mortality rates of Aboriginal infants the data on birthweights indicate that the deficit between Aboriginal and general Australian birthweights is unchanged (Dugdale 1980; Reid and Kerr 1983). In one Aboriginal community it was reported that the mean birthweight in the period 1958-65 was 2,800 gm and by 1979-82 the birthweights in the same area were almost the same at 2,889 gm (Muller et al 1984). There are similar findings in Central Australia where in 1965-67 Maxwell and Elliott (1968) found that 12.5 per cent of infants were less than 2,500 gm; in 1984 Rae (1985) found that the percentage was 13.1. Over the same period the mean birthweight for the general Australian population has been about 3,440 gm with a standard deviation of 425 gm and 3,390 gm with a standard deviation of 523 gm, with four to six per cent born at less than 2,500 gm (Keeping et al 1979; Seward and Stanley 1981). We therefore have the paradoxical situation where child health is improving but birthweights are remaining low.

There are many studies of the factors affecting birthweight, including the height of the mother, weight gain during pregnancy and pre-pregnancy, weight of the mother, parity and smoking (Keeping et al 1979; Baird 1962; Kline et al 1987; Niswander and Jackson 1974; Picone et al 1982; Thomson et al 1968; Stein and Kline 1983). The reported effects of alcohol consumption have been variable (Kline et al 1987; Stein and Kline 1983). Famine lowers birthweight (Antonov 1947) but food supplementation increases birthweight only when nutrition is severely impaired (Prentice et al 1983). Social class of the mother has also been implicated. The reporting of these studies is far from uniform so comparisons are difficult but these factors do not explain all of the variability in birthweights.

Methods

Cherbourg Aboriginal settlement is 280 kilometres northwest of Brisbane in Queensland, Australia. During the summer the temperatures are high, but the settlement is 800 metres above sea level and there are frosts in winter. The population is about 1,500 people, all of Aboriginal descent. Physical facilities have improved markedly over the last two or three decades. The people now live in separate houses of the same standard as welfare housing in Queensland, with electricity, water and flush toilets. Some of the houses have a nuclear family but others are more crowded with extended families. There is a supermarket on the settlement and there is a sealed road to the nearest country town, seven kilometres away, and also to Brisbane. Many people now own cars and there are daily buses to Brisbane. The settlement has a primary school for 300 children and a 40-bed hospital with full-time registered nurses and a doctor. There is a high level of unemployment and alcoholism among adults. The infant mortality rate has dropped from about 280 per 1,000 live births in the 1950s to about 150 per 1,000 live births in the 1960s to between 20 and 40 per 1,000 now.

The Infant Health Clinic on the settlement has been operating since 1950 and almost all infants are seen there. The first visit is usually within two weeks of birth, they are seen weekly until one year and less frequently up to five years. The records of all visits back to 1952 are held in the Clinic. Some of the children born on the settlement are still living there as adults, so we can compare growth and health across generations. In this study we have used data from two sources:

1. The main source is the data at the Infant Health Clinic. The data used are the date of birth and sex of the infant as well as the birthweight, or, where this is not available, the weight at the first visit between birth and two weeks. For the mothers and fathers of the index infants we have also used the weights at one year and five years when available. The parities and ages of the mothers were also used when available.

2. The other source is a data set collected by the Aboriginal Health Program. Since 1972 teams trained in anthropometry have been visiting this and other settlements around Queensland measuring children and collecting data on birthweights and other measurements. Participation in these surveys was voluntary, but at Cherbourg Settlement about 90 per cent of the children were measured, many on several occasions.

The data from the two sources were compared to check for accuracy and completeness. The data were then used in two ways. The first was to compare the birthweights and incidence of low birthweight over the 30 year period. All records of children born in the years 1952-53, 1962-63, 1972-73 and 1982-83 have been examined and the birthweights noted. For children born between 1983 and 1988, we also searched the records for the data on their mothers and where possible on their fathers. These data sources give us only the children who attended the clinic. They therefore exclude infants who were stillborn or who died before attending the clinic.

Results

For the years 1952-53, 1962-63, 1972-73 and 1982-83 we have records of 526 infants. This number excludes twins and those infants who died before they could attend the Infant Welfare Clinic but includes infants who died later. There was no birthweight (or weight within 14 days of birth) for 79 of these infants, so the effective sample size is 447. The mean birthweights for each two year period have been calculated and also the percentage of infants weighing less than 2,500 gm. The data in Table 1 show that the birthweights of Aboriginal infants have remained constant since 1952-53. There is no significant trend in the birthweights over the 30 years. The mean birthweight is about 200 gm less than that for the general Australian community and the incidence of low birthweight is considerably higher.

We have searched the clinic records of children born between 1983 and 1988 to find those with suitable maternal data in the earlier years of the data set. We have located suitable records on 119 mothers born between 1951 and 1974. The mothers ranged in age from 14 to 35 years at the time of delivery. Sixty-six mothers had only one delivery in the interval from 1983 to 1988 and 53 had two deliveries. We do not have reliable information on the heights of mothers, their pre-pregnant weight or their weight gains during pregnancy. However, we have data relating to their birthweights and growth during infancy and childhood.

Table 1 Means and standard deviations of birthweights of infants born at Cherbourg settlement 1952-83, and some comparative data for other Australian infants.

Year	N (with birthweight)	Mean (gm)	SD (gm)	N	<2,500 gm %	Birthweight not available
Cherbourg settlement						
1952-53	110	3310	527	6	5.0	23
1962-63	94	3258	567	8	9.0	17
1972-73	129	3115	538	11	9.0	9
1982-83	104	3170	566	13	13.0	20
Brisbane						
1974	9261	3440	425	-	-	-
Western Australia						
1978	18850	3342	629	-	6.1	-

A preliminary analysis using a generalised linear model showed that the variability in mean birthweights between families was four to five times greater than between children in the same family, a result with a high degree of statistical significance ($p<0.001$). To avoid consequent bias due to the similarity of children within families, only the last child born during the years 1983-88 in each family was included in the first set of analyses. A second round of analyses was done later using the second to last child. Siblings born before 1983 were not included in the analyses.

In these analyses the dependent variable was the birthweight of the child. The maternal factors were the birthweight of the mother, her weight at one year and at five years, together with her age and parity at the birth of the child. All variables were

approximately normally distributed except for the age of the mothers which was slightly skewed to the right. As would be expected age and parity of the mothers were highly correlated ($p<0.0001$), so grouped parity was used in the analyses. The results of analyses using the generalised linear model are shown in Table 2.

Table 2 Adjusted effect of mother's early history on birthweight for last birth.

		N	Mean	SD	% Additional variation explained	Probability
Sex of infant	F	56	3189	499	0.5	0.8011
	M	59	3068	495		
Parity of mother	1	26	3029	414	0.4	0.5003
	>1	89	3155	519		
Birthweight of mother		115			8.0	0.0025
Regression coefficient: 0.23570 ± .0707						
Sex of infant	F	51	3188	510	0.9	0.3207
	M	55	3075	492		
Parity of mother	1	24	3062	400	1.0	0.2807
	>1	82	3148	528		
Weight of mother at one year		106			3.0	0.0622
Regression coefficient: 88.809 ±47.0915						
Sex of infant	F	52	3180	507	0.9	0.3355
	M	50	3077	509		
Parity of mother	1	22	3051	413	0.6	0.4434
	>1	80	3151	532		
Weight of mother at five years		102			0.6	
Regression coefficient: 8.233 ± 33.4991						

The first set of analyses, using the last-born child, show that: the parity of the mother and therefore her age had no significant effect on the birthweight and explained only one per cent of the variation; the sex of the infant had no significant effect; the birthweight of the mother had a significant effect ($p<0.0025$) on the birthweight of the child and accounted for eight per cent of the variability; the regression coefficient for mother's birthweight showed that a difference of 100 gm in the birthweight of the mother would produce a difference of 24 gm in the birthweight of the infant, independent of the sex of the infant and parity of the mother; the mother's weight at one year explained only three per cent of the variability in birthweight of the infant and her weight at five years less than one per cent.

The separate analysis of the second to last child born between 1983 and 1988 was done on smaller numbers. The results are shown in Table 3 and are generally similar to those for the first analysis. However, in this group of infants the mother's birthweight accounted for 24 per cent of the variability in birthweight of the infant. A similar analysis was also done for fathers, but there were fewer fathers than mothers with available data. The results of the analyses for the fathers are shown in Tables 4 and 5. Unlike the mothers, none of the variables for fathers had any significant effect on the birthweight of the infant.

Table 3 Adjusted effect of mother's early history on birthweight for second to last birth.

		N	Mean	SD	% Additional variation explained	Probability
Sex of infant	F	27	3141	354	0.4	0.8658
	M	24	3266	354		
Parity of mother	1	12	3083	228	5.0	0.0788
	>1	39	3236	383		
Birthweight of mother			51		24.0	0.0002
Regression coefficient: 0.2736 ± 0.06903						
Sex of infant	F	25	3069	308	12.0	0.0201
	M	21	3289	345		
Parity of mother	1	11	3075	237	3.0	0.2453
	>1	35	3199	365		
Weight of mother at one year		46			35.0	
Regression coefficient: 50.481 ± 42.6621						
Sex of infant	F	26	3112	362	4.0	0.2434
	M	26	3284	338		
Parity of mother	1	11	3075	327	6.0	0.0885
	>1	32	3232	386		
Weight of mother at five years		43			10.0	0.0328
Regression coefficient: 69.440 ± 31.3804						

In a separate study of 30 women living on Cherbourg settlement we have data on the birthweights, height and weights. The women are not the same group as in the main part of this study, but there is some overlap between the two groups. In this group the mean birthweight was 3,262 gm (standard deviation 589 gm), the mean adult height 159.8 cm (standard deviation 5.6 cm), the mean adult weight 74.6 kg (standard deviation 17.0 kg) and the mean Body-Mass Index 28.3 (standard deviation 6.4). The correlation coefficient between birthweight and adult height was 0.14, between birthweight and adult weight was 0.34 and between birthweight and Body-Mass Index was 0.29. In this small series these correlations did not reach statistical significance but their magnitudes were similar to those reported in other studies.

Conclusions

The birthweight of infants is important for the clinician because it indicates the risk of neonatal death and hence the level of care the infant will need. To the epidemiologist, the distribution of birthweights has been used as a marker of health standards of the community. If a marker is to be useful, it must not only parallel some important aspect of community health and welfare, but must also respond promptly to changes. In Cherbourg Aboriginal settlement, the health, growth and mortality of infants and children have improved dramatically over the last 30 years, but birthweights have remained unchanged. Birthweight does not seem to be a responsive indicator of community well-being. Our finding that the birthweight of the mother has a considerable effect on the birthweight of the child suggests that there will always

Table 4 Adjusted effect of father's early history on birthweight for last birth.

		N	Mean	SD	% Additional variation explained	Probability
Sex of infant	F	27	3336	577	3.0	0.1945
	M	33	3162	525		
Age of father		60			2.0	0.2441
Birthweight of father		50			3.0	0.1582
Regression coefficient:		-0.201 ± 0.141				
Sex of infant	F	30	3301	576	35.0	0.2100
	M	34	3158	518		
Age of father		64			0.2	0.1040
Weight of father at one year		64			4.0	
Regression coefficient:		-103.126 ± 62.477				
Sex of infant	F	32	3270	570	2.0	0.3164
	M	34	3158			
Age of father		66			0.001	0.9757
Weight of father at five years		66			4.0	0.1076
Regression coefficient:		-59.803 ± 36.624				

be a lag in the change of birthweights. Although improvements in other factors may bring some increase in birthweight, the full increase in mean birthweights and reduction in the percentage of low birthweight infants will occur over generations rather than years.

Table 5 Adjusted effect of father's early history on birthweight for second to last birth.

		N	Mean	SD	% Additional variation explained	Probability
Sex of infant	F	15	3155	439	1.0	0.6176
	M	13	3228	391		
Age of father		28			0.7	0.6768
Birthweight of father	60	28			0.7	0.6919
Regression coefficient:		0.600 ± 14.950				
Sex of infant	F	17	3132	434	1.0	0.5643
	M	14	3222	377		
Age of father	64	31			0.01	0.9504
Weight of father at one year	64	31			0.005	0.9719
Regression coefficient:		-2.984 ± 83.9987				
Sex of infant	F	18	3185	476	0.1	0.8420
	M	14	3215	364		
Age of father		33			0.3	0.7554
Weight of father at five years		33			0,3	0.7791
Regression coefficient:		-13.669 ± 46.6842				

Reports in the world literature show that many factors have a significant effect on the birthweight of infants. The most important is the duration of pregnancy, followed by the height of the mother, smoking, weight gain during pregnancy and pre-pregnancy weight. There seems to be no other investigation of the effects of mother's infancy and childhood on the outcome of her pregnancies, probably because no data are available. In our separate study of 30 women at Cherbourg Aboriginal settlement there were indications associating birthweight and adult height, weight or weight-for-height, so it is possible that the relationship between the birthweight of mother and child is mediated through mother's adult height and body build. A direct genetic mechanism is unlikely. If this were the cause then the father's birthweight should have an equal effect to the mother's. Although height, weight and now birthweight of the mother have been shown to have a significant statistical association with the birthweight of her children, the mechanism of action is not yet clear. However, the classic cross-breeding studies on horses done by Walton and Hammond (1938) showed that birthweight was almost entirely controlled by the size of the mother and that foals that were small at birth tended to grow more slowly later. Humans rarely differ in size as much as do Shire horses and Shetland ponies, so the effects of maternal size on birthweight will be less obvious in humans.

In this study we have found that a deficit of 1,000 gm in the birthweight of the mother is associated with a deficit of about 250 gm in the birthweight of the child. The effects of other known factors on the birthweight of the child have been found to vary among different groups. This finding among Aboriginal mothers must therefore be confirmed among other groups, if the necessary data can be located. The importance of the finding lies not so much in the clinical needs of the slightly smaller infants, but rather in the implications for use of the birthweight as an indicator of community well-being. This finding shows that improvements in birthweights reflect not only present conditions, but also mirror past events. The results of earlier deprivation may take a generation to disappear and during that time birthweights will not provide an accurate indication of the current health and well-being of the community.

5 ABORIGINAL BIRTHWEIGHT RELATED TO FUTURE GROWTH AND DEVELOPMENT[1]

RW Streatfield, K Streatfield, Miranda Korzy and IA Musgrave

In this retrospective study of Aboriginal birth, growth and development, the determining effects of birthweight and percentiles on future growth are considered. This chapter describes and attempts to interpret how birth percentile is associated with the child's future growth percentiles. Several international studies have demonstrated the relationship between birthweight and future morbidity and mortality (Puffer and Serrano 1973; Saugstad 1981; Voorhoeve et al 1984). Other studies have described the relationship to future growth and development (Ounsted and Taylor 1971; Fitzhardinge and Steven 1972; Ramey et al 1978; Jansen et al 1984) and there have been a few papers documenting the relationship between low birthweight and poor growth and development specifically in Australian Aboriginal children (Dugdale and Lovell 1981; Cameron and Debelle 1986; Roberts et al 1988).

The graphs in this chapter were derived from analysis of the anthropometric data from birth up to 15 years age, or to current age if less than 15 years old, of approximately 3,500 Aboriginal children in total. Children from seven Far North Queensland Aboriginal communities are included. The bulk of the anthropometric data was obtained, with permission, from the records of the Queensland Department of Health Aboriginal Health Program. The Aboriginal Health Program has been running nutrition monitoring and intervention programs in these communities since the early 1970s.

Methods

Birth details and anthropometric results were collected from each individual child's Personal Record Form. These are kept up-to-date by the Aboriginal Health Program Teams on the various communities. All birth details, such as birthweight, date of birth, gestation, mother's name and parity, are verified in writing, by the hospital in which the birth took place and only entered on the record when this information is received by the Health Team. Nearly all births from these communities occurred in Cairns Base Hospital so data on birthweight, gestation and birth length, are those derived by the hospital obstetric or paediatric staff.

On average, every child under the age of 15 years, on these communities, is 'screened' every two to three years by the local Aboriginal Health Program Team. This screening includes precise and accurate anthropometric measurement of the child by the especially trained Public Health Nurse using such reliable equipment as beam-balance scales and stadiometers. This data is entered directly on the record of each

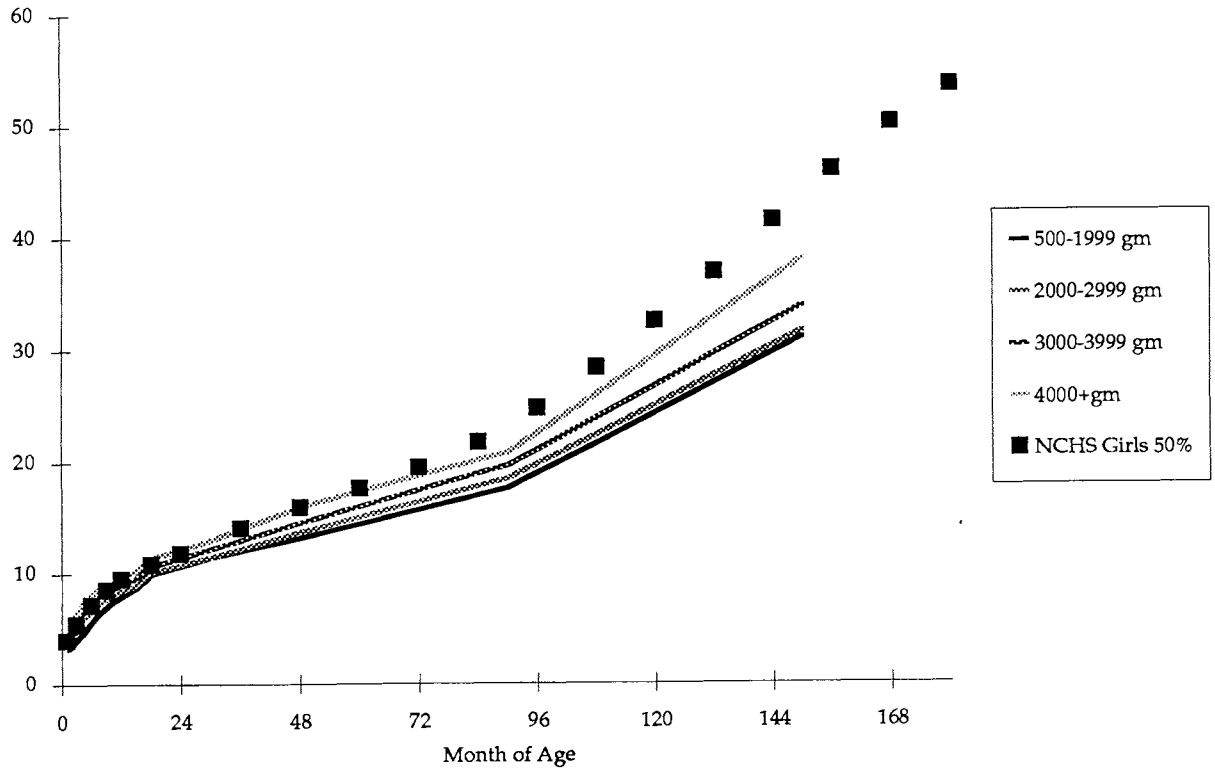
child. It is only this precise data obtained at screening that is used in this study. If there was another screening done in the period of the age groupings used in this study, say two screenings in the period when the child was five to 10 years old, that is 60 to 119 months, then the data from the earliest screening were used as being representative of this period of the child's life. Thus the graphs represent cross-sectional data.

The Aboriginal Health Program Team has, up until now, used the Harvard Standards as the anthropometric standards for comparison and evaluation of programs and interventions. In this study the NCHS/CDC/WHO standards were used to convert the raw anthropometric data to percentiles. The 'CASP 31' software program was used to calculate the percentiles.

Results

Figure 1 depicts the findings in growth in weight according to birthweight group, up to the age of 15. The 50th percentile weight-for-age for girls is included as a reference. Children with birthweights over 3,000 gm remain above the 50th percentile for girls up until about eight to nine months of age, and by one year of age have dropped below, the difference steadily increasing from about five years of age onwards.

Figure 1 Mean weight by age and birthweight.



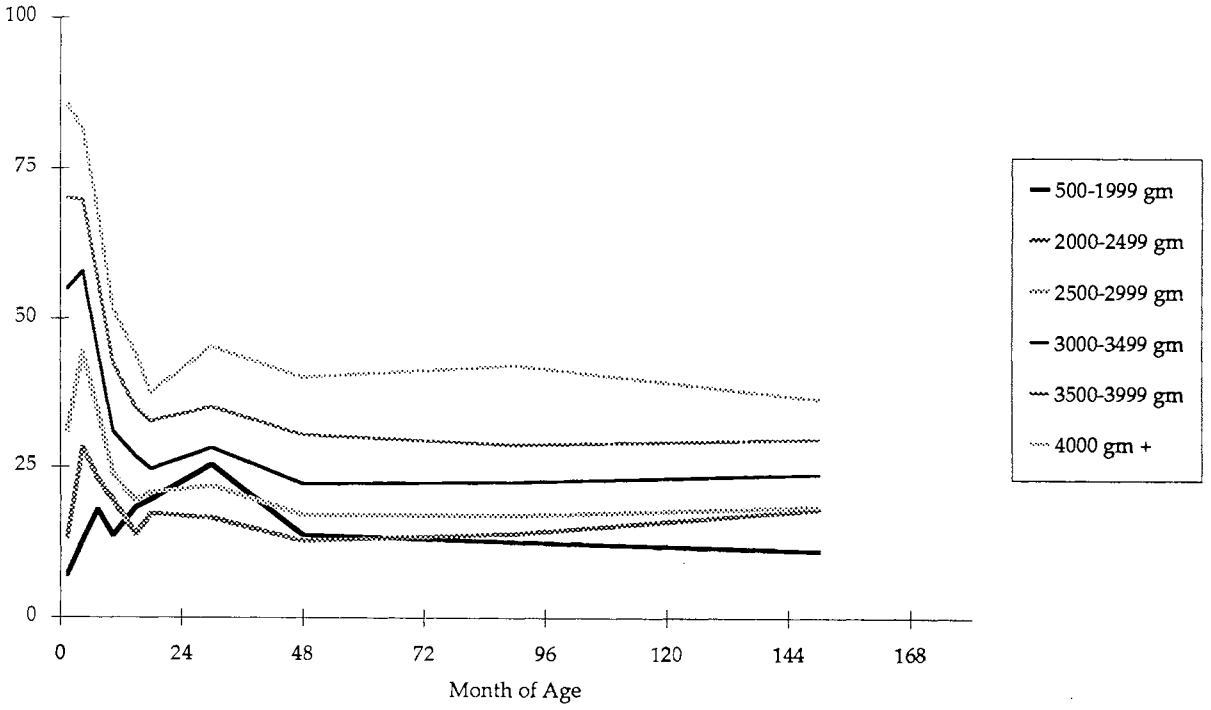
NOTE: In figures in this chapter, the Aboriginal growth projectiles extend only to a maximum of 150 months—the mid-point of the 120-80 month category.

Even children in the heaviest birthweight group (4,000 gm and over) drop to the 50th percentile by age one year, and below by five years. The average weights of the birthweight groups remain relatively distinct right through the 15 years. However, the gap between the birthweight group below 2,000 gm and the next heaviest group narrows considerably from 18 months to about five years of age, and remains relatively close from there on. This lowest birthweight group appears to remain parallel with the 50th percentile for a longer period than do the higher birthweight groups. Diversion from the parallel comes at about six months of age in the latter groups whereas, in the former, this does not seem to occur until past two years of age.

The pattern in regard to growth in height is much the same, but the lines depicting average heights for the birthweight groups are closer together and cannot be shown effectively in graphical form. By 18 months to two years there is not much difference in height, and the lowest birthweight group has caught up. Steady diversion from the parallel with the 50th percentile (NCHS, girls) occurs at about 36 months of age for the lighter birthweight group but is more obvious and persistent in all groups from five years of age onwards. Statistical significance tests show that the weight-for-age differences between the birthweight groups remain significant generally to the 0.1 per cent level through to age 15 years. The height-for-age differences are also consistently significant in the younger age groups but less so in later years.

Figures 2, 3 and 4 should be considered together. The birthweights have been grouped and the three major anthropometric percentiles, weight-for-age (W/A), height-for-age (H/A) and weight-for-height (W/H) have been plotted for the ages up to 15 years. There is a universal improvement in W/H in all birth groups in the first six

Figure 2 Mean weight-for-age percentile by age and birthweight.



months of life, quite spectacular in the lower birthweight groups. In fact, in all but the highest birthweight group there is a trend towards higher percentiles of W/A and H/A in the first six months of life. This is probably related to the degree of recent starvation in utero plus the fact that on-demand breast milk alone is adequate for the first four months or so.

Figure 3 Mean height-for-age percentile by age and birthweight.

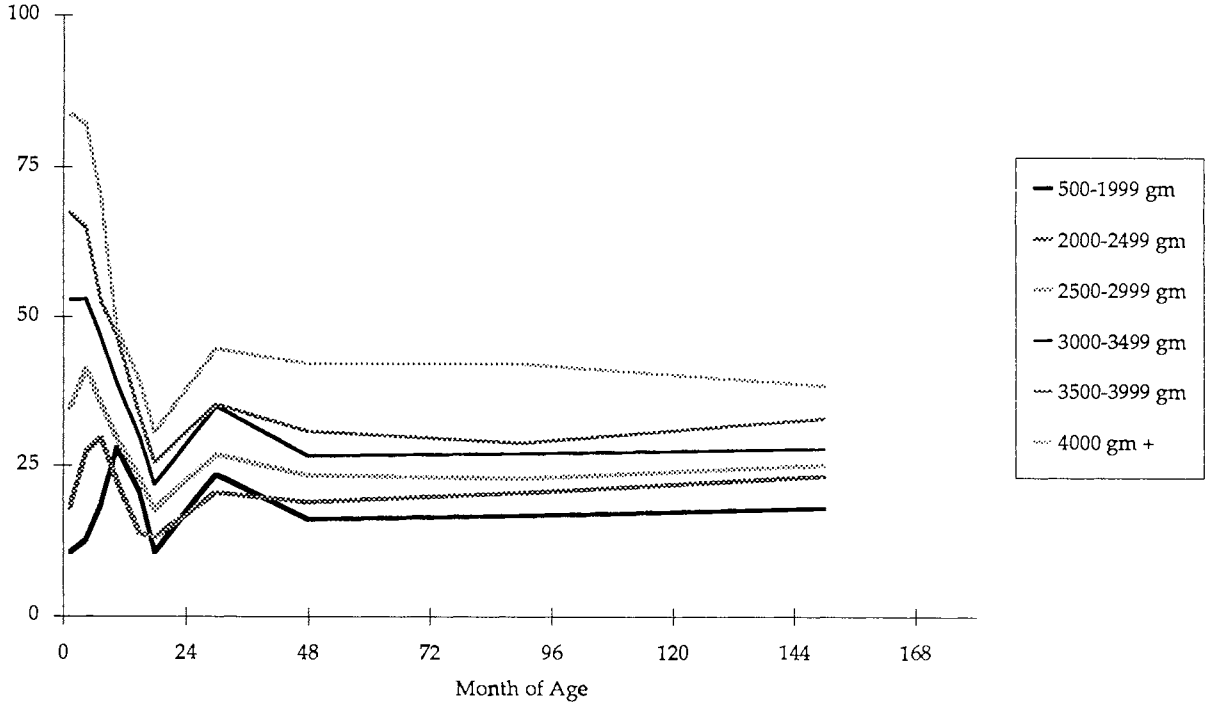
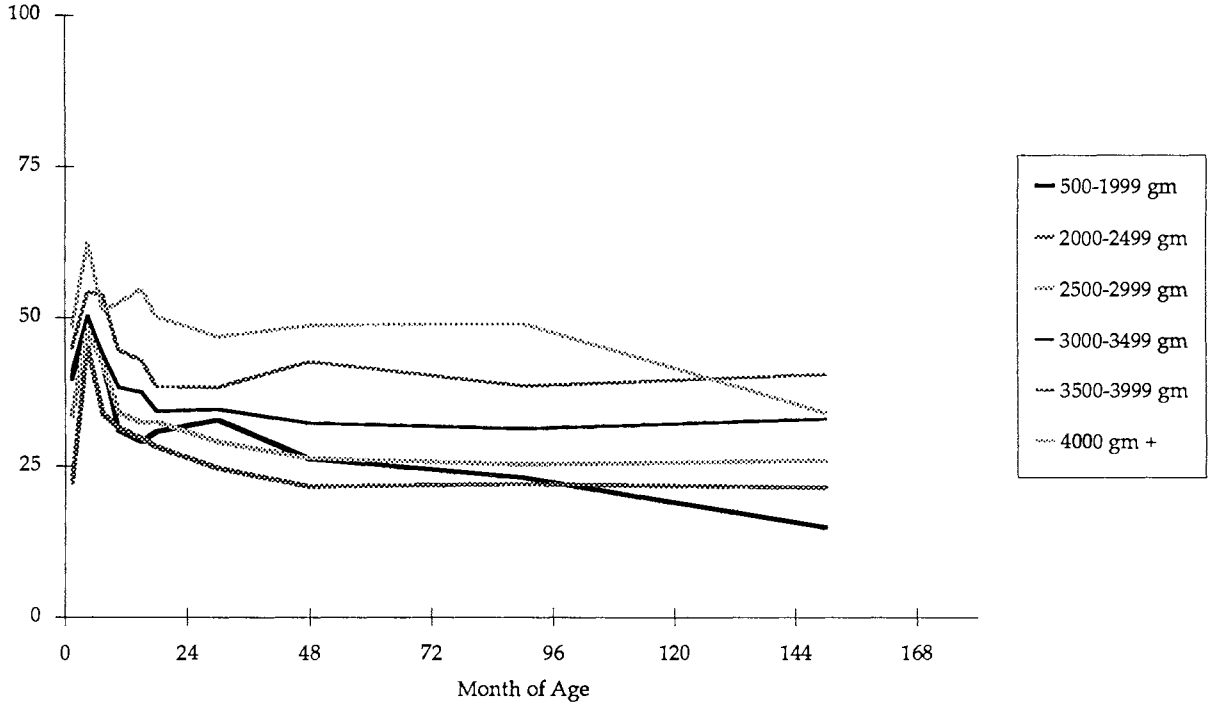


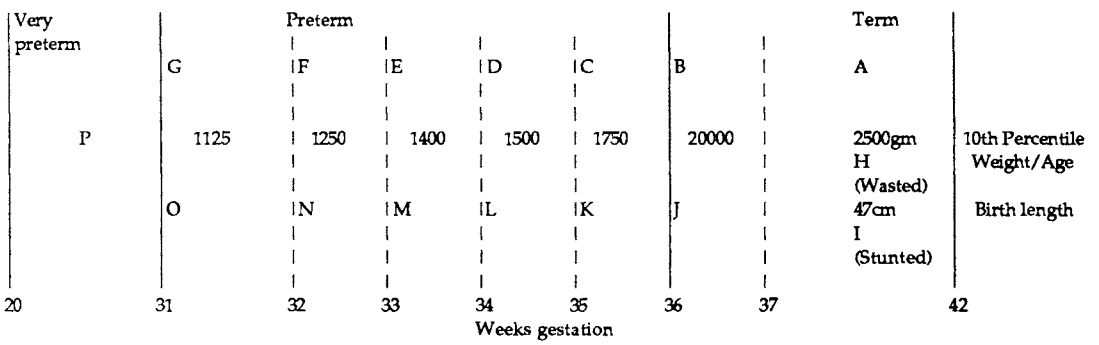
Figure 4 Mean weight-for-height percentile by age and birthweight.



Except for the lowest birthweight groups, this early growth spurt reverses in the period from about six months to two years of age. The lowest birthweight group (1,500-1,999 gm) is different to all the others in that there appears to be, overall, a steady increase in percentiles W/A from birth through to two years of age, but to only one year of age for the H/A percentiles. The differences between the means, W/A and H/A generally do not reach significance at the five per cent level for these two lowest birthweight groups when, as here, gestation is not considered. The small but significant height recovery at two to three years of age, across all birthweight groups, is interesting and is reflected to a lesser extent in weight gain as well. These trends across percentiles with age give the W/A and H/A charts a 'wind-sock' appearance, with the final average percentiles attained by each birth group much closer together at 15 years of age than they were shortly after birth. Though closer together by 15 years these differences generally remain significant at least at the five per cent level for the W/A groups.

The above analysis, using just birthweight as the variable to study relationships with future growth of children, is very crude and only yields limited information. Several workers have suggested a much more refined classification of birth groupings, taking into consideration gestation and birth length (Villar and Belizan 1982). Figure 5 summarises the cut-off points for these birth groupings. The biggest division is the term births as opposed to the preterm births. Gestation of 37 to 42 weeks is considered a term delivery, less than 37 weeks is preterm. The tenth percentile W/A (Lubchenko et al 1972) divides births into four major groups, those at the 10th percentile and above, and those below, whether term or preterm. The 10th percentile is around 2,500 gm for term births, but of course decreases with declining gestation. Babies born less than the 10th percentile W/A are considered SGA (cells H through to O in Figure 5) as opposed to being born AGA (cells A through to G in Figure 5). To further refine this classification, Villar and Belizan (1982) have suggested a division of the infants less than the 10th percentile W/A at term by the 10th percentile length-for-age. This is at approximately 47 cm birth length for a term infant. These are cells H and I in Figure 5. The theory is that those born in cell H are 'wasted' or 'disproportionate', reflecting recent starvation *in utero*, whereas those born in cell I are 'stunted' or 'proportionate', reflecting a more prolonged period of chronic starvation *in utero*.

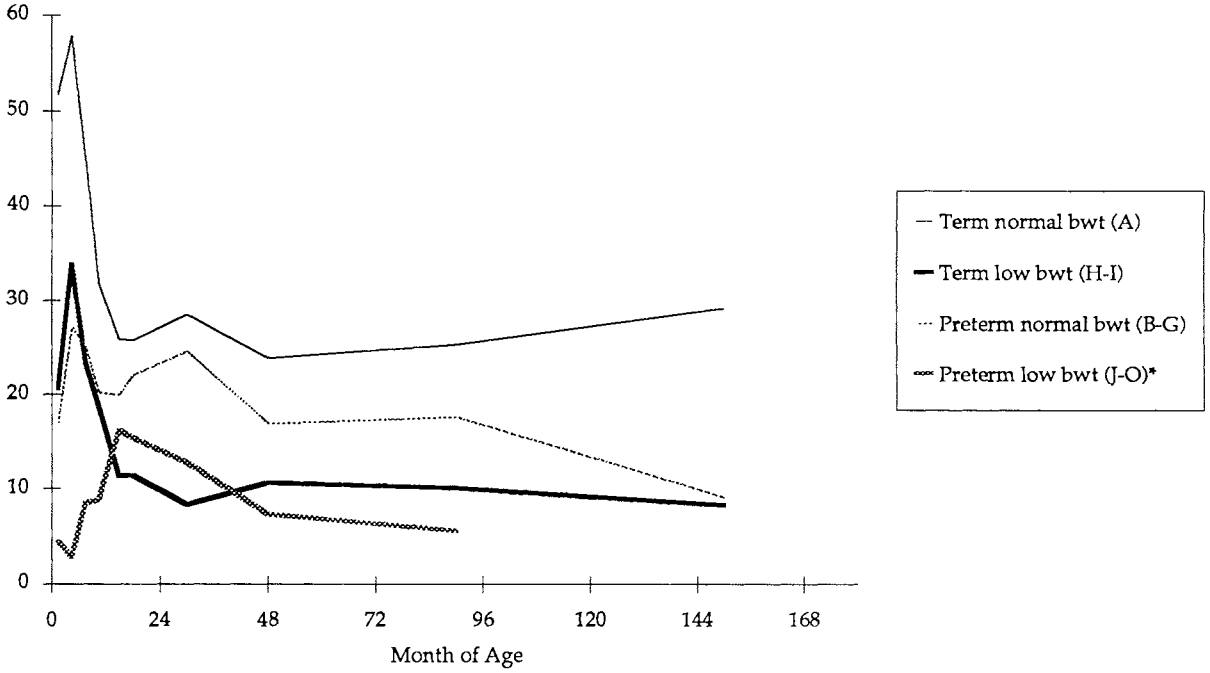
Figure 5 Classification of births by weight, gestation and length.



There is some evidence that classifying births into these six groups (in Figure 5, A, B-G, H, I, J-O and those below 31 weeks gestation) and following and contrasting the growth and development of each group, gives a much clearer idea of how the intra-uterine environment is related to the future growth and development of the child. Most of the following studies have not fully applied such a refined classification, but they have all at least used the four basic groups of term, preterm, AGA or SGA (see Beck and van den Berg 1975, Neligan et al 1976, Toth et al 1978, Bhargava et al 1979, Kaltreider and Kohl 1980, Martell et al 1981, Westwood et al 1983, Villar et al 1984).

There were a total of 1,392 births with sufficient data on birthweight and gestation to enable classification into the divisions suggested in Figure 5. In Figures 6, 7 and 8, an attempt has been made to separate the four major birth groups (A, H-I, B-G and J-O) and follow the percentile growth patterns. The dashed lines represent the average percentiles for age for the AGA and SGA at term whereas the solid lines are for the preterm births. The percentiles of W/H and H/A for the term births remain fairly parallel, finishing up 10 percentile points or so apart. The differences between the W/A means remain highly significant right through to 15 years of age, whereas H/A differences lose significance in the later age groups. The preterm infants, represented by the fainter lines, also remain distinctly apart. The preterm-SGA group (J-O) gain W/A and H/A percentiles for six months longer than the other three birth groups, but then slowly decline such that, by 10 years of age, they are back close to the low percentile they started on. The little 'height-spurt' at around two to three years of age is much more apparent in children born appropriate for gestational age than in those who were small.

Figure 6 Weight-for-age percentile by age, gestation and birthweight.



NOTE: In Figures 6 to 11, there are no data beyond the 84-96 month age group for those groups marked with an asterisk.

Figure 7 Height-for-age percentile by age, gestation and birthweight.

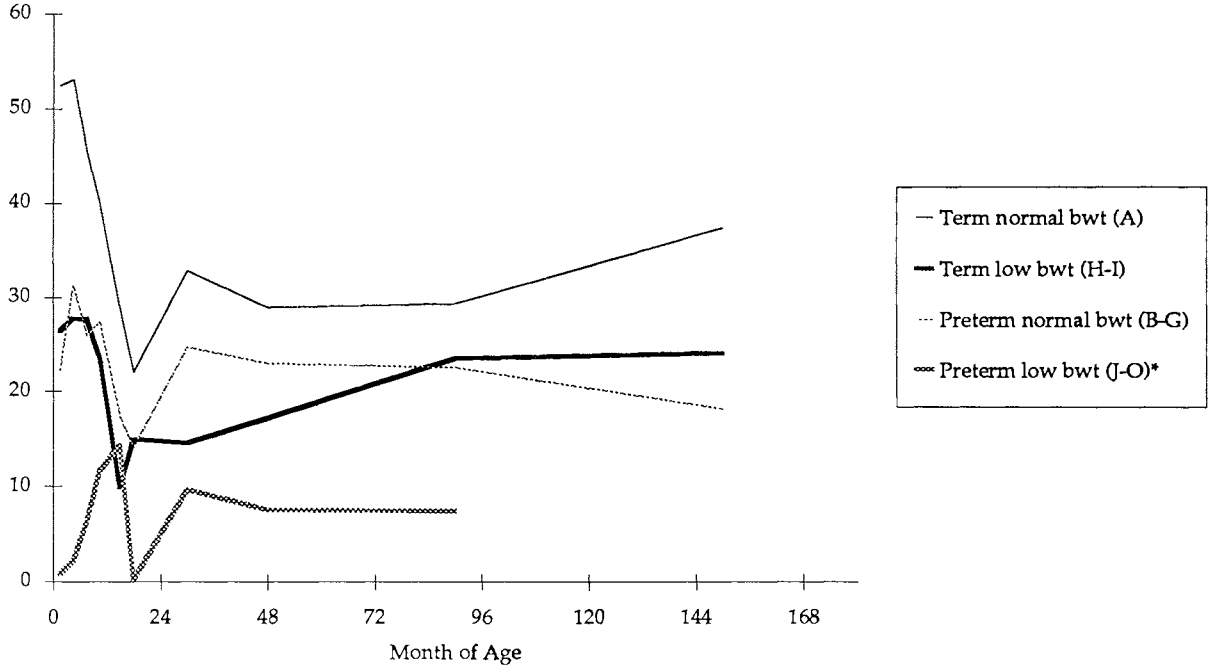
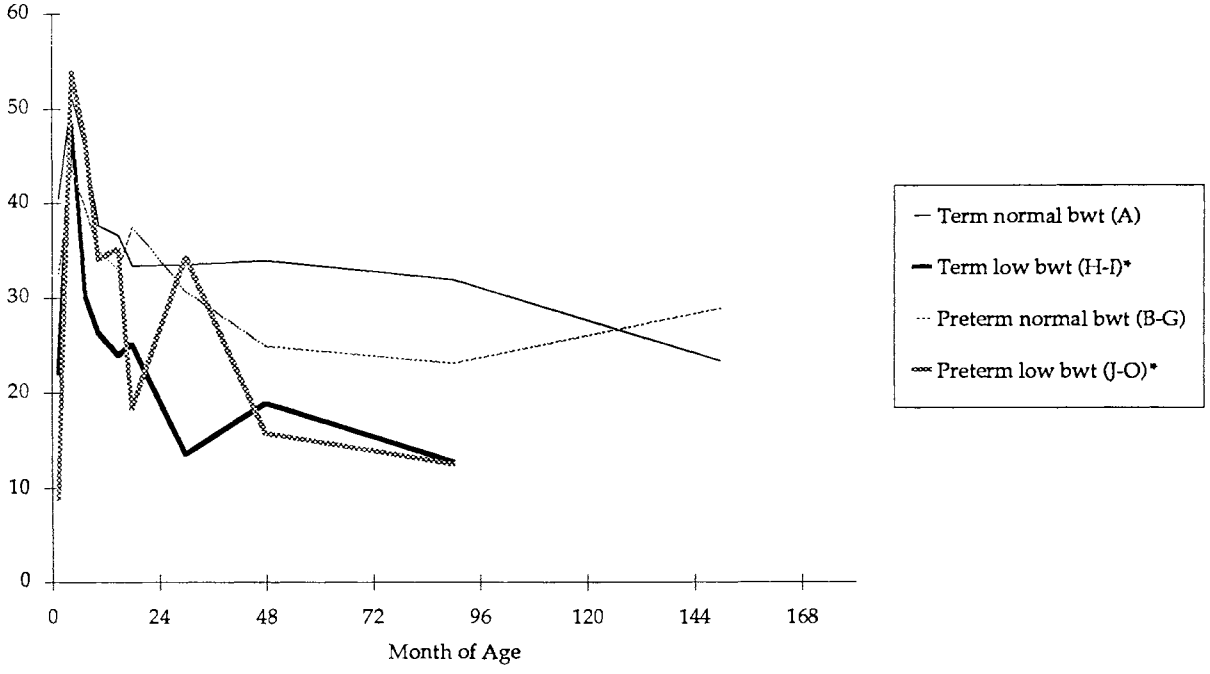


Figure 8 Weight-for-height percentile by age, gestation and birthweight.



In Figures 9, 10 and 11 the percentiles for the term normal, the term wasted and the term stunted are compared. The suggestion has been that the children born stunted and reflecting longer term malnutrition *in utero* do not do as well as those wasted at birth. There were 1,054 births in the term normal group (A), but only 97 births in which birth length was available as well as birthweight and gestation enabling the division into wasted (H) and stunted (I). Referring to Figure 9, there does not seem to be any significant difference in weight gain over the years between the wasted and stunted groups but from Figure 10, it would appear that the stunted remain stunted throughout childhood whereas the wasted tend to regain normal height although the tests of significance do not generally reach significance at the five per cent level for either W/A or H/A when these groups are compared.

Figure 9 Weight-for-age percentiles for infants born at term by age.

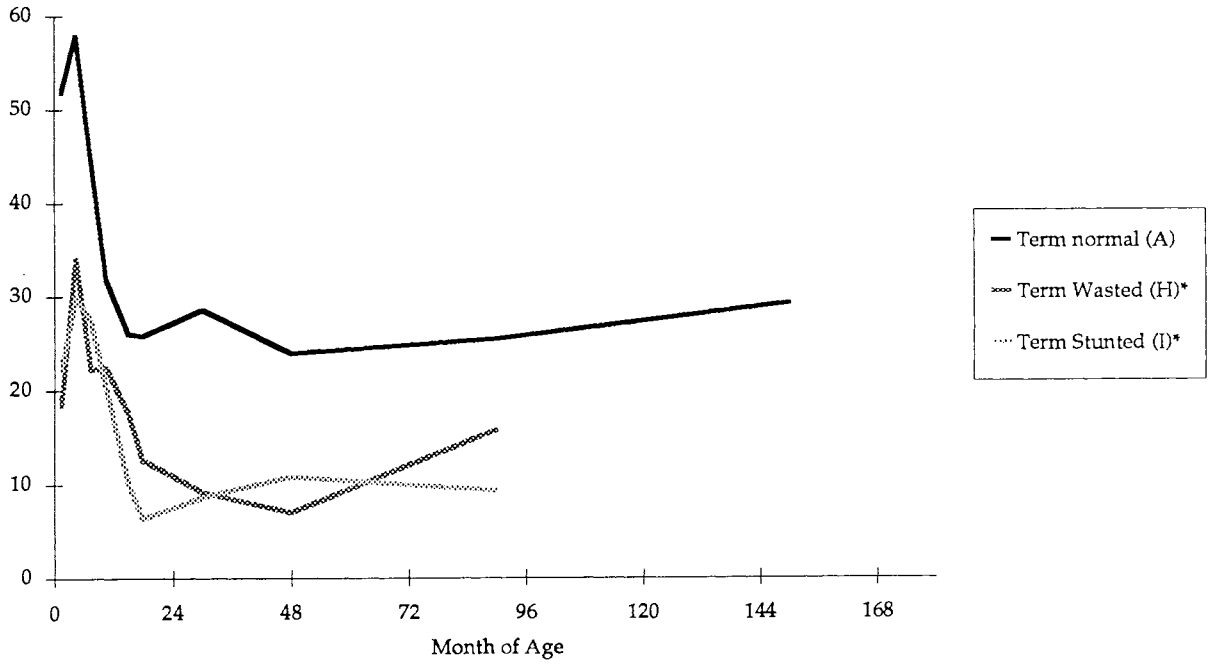


Figure 10 Height-for-age percentiles for infants born at term by age.

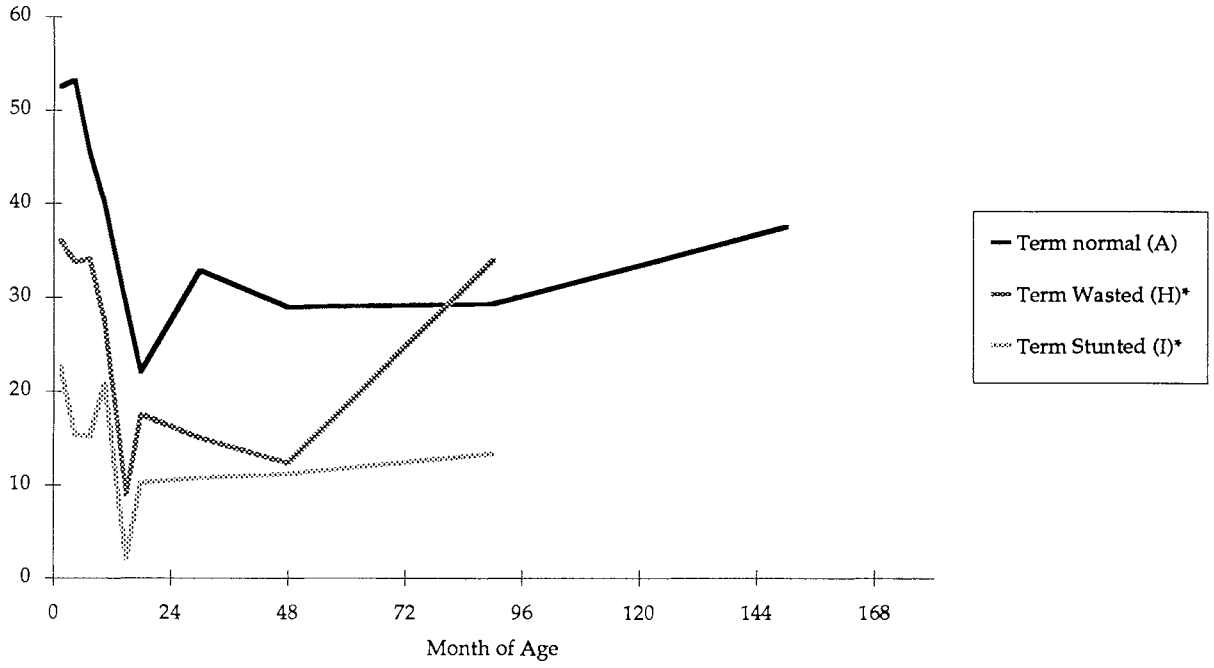
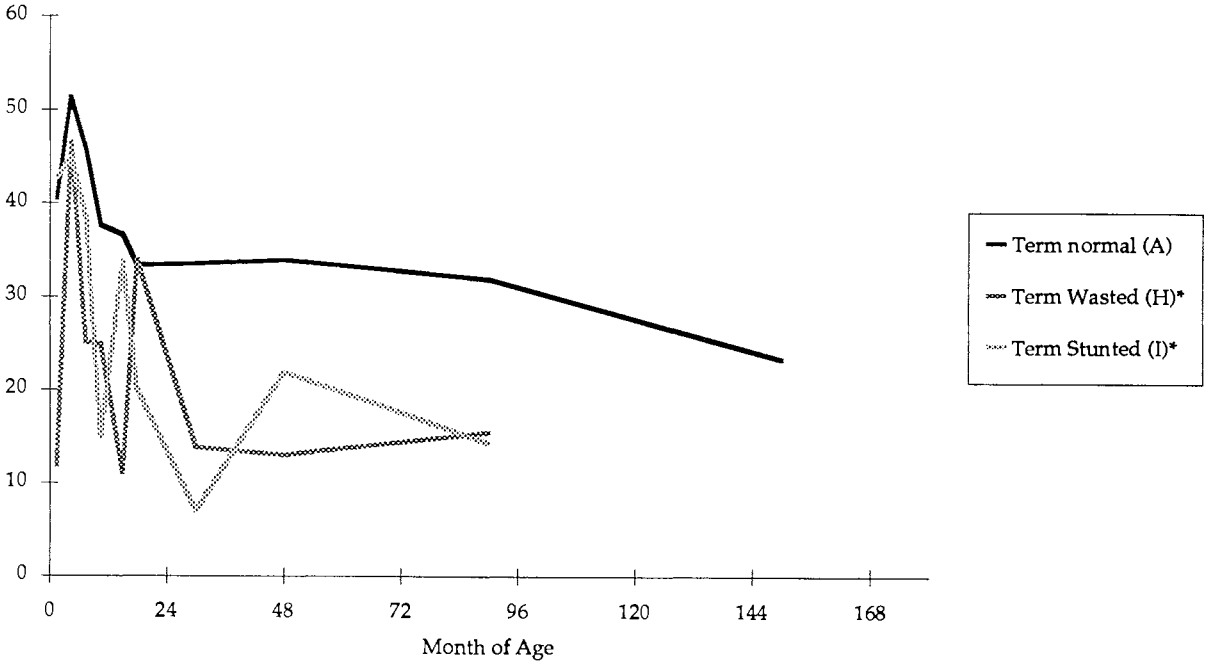


Figure 11 Weight-for-height percentiles for infants born at term by age.



Conclusions

The basic hypothesis to be tested, in this study on growth and development of Aboriginal children, is to determine whether this racial/geographical group of children's future growth is limited by growth retardation in utero as are children in other parts of the world. Growth retardation is indicated by being born with a W/A percentile less than 10.

Villar and Belizan (1982) analysed data from 11 different regions in the developed world and 25 areas in developing countries. They used a birth classification much like that depicted in Figure 5, and found that low birthweight births less than 2,500 gm were due 6.6 times more often in developing countries to starvation in utero than in developed countries. In developed countries low birthweight was less common anyway, and more likely due simply to being preterm, but usually 'appropriate'.

Roberts et al (1988) studied the growth and morbidity in children in a remote Aboriginal community in northwest Australia. They found evidence of widespread mild-to-moderate malnutrition and a high prevalence of infections, particularly of the respiratory and gastrointestinal tracts. The presence of poor growth at five years of age was significantly associated with low birthweight. The authors suggest that malnutrition in utero, during infancy, and in early childhood may impair the growth of young Aborigines permanently. Maxwell et al (1968), also found a close association between respiratory infection and malnutrition in Aboriginal children in the Northern Territory. In the Murray Valley, Cameron and Debelle (1986) found approximately twice the expected number of Aboriginal infants were born SGA, and there were higher levels of stunting and wasting in children aged less than 12 years than in non-Aboriginal children in the same district. Again, in the Kimberley region of Western Australia, Hitchcock et al (1987) found that the 1,887 Aboriginal school children studied approximated the 10th percentile W/A and the 25th percentile H/A of the non-Aboriginal Western Australian children.

In other parts of the world similar adverse findings regarding the effects of starvation in utero on the future growth and health of children have been made in recent years. In India, Bhargava et al (1979) followed the growth of three birth groups to 14 years of age. The three groups were—preterm AGA (B-G), full-term SGA (H-I) and full-term AGA (A). When the raw data is converted to NCHS percentiles and weight and height are plotted as in Figures 6 and 7, some similarities can be seen. There is no real recovery of percentiles of weight or height for age in the 14 years in the SGA group, as is the case in Figures 6 and 7. The preterm AGA group (B-G) in the Indian study make steady progress throughout childhood and attain percentiles close to the full-term AGA group by age 14 years. This does not seem to be so in the Aboriginal child growth data studied here. The preterm AGA's merely hold their own, and percentiles actually drop at puberty.

Beck and van den Berg (1975) found that preterm AGA infants generally experience a period of rapid catch-up growth in the first two years of life, and

subsequently, their heights and weights were similar to their full-term AGA counterparts. By contrast, children whose growth was retarded in utero (SGA) did not have the same amount of catch-up growth and remained shorter and lighter than their AGA counterparts. Toth et al (1978) found 'a significant retardation in weight, stature, head circumference and osseous development of SGA children was observed even at the age of three years'. Neligan et al (1976) had also found significant differences in the growth of premature, SGA and control AGA children. However, Martell et al (1981) found that, in their group of children, these differences disappeared when the children were on average 18 months old.

Several studies have demonstrated the links between low percentile, poor growth and development, with increased chances of morbidity and mortality. Kaltreider and Kohl (1980) found a perinatal mortality rate for preterm SGA births to be five times higher than that for a term SGA infant. Chen et al (1980) in Bangladesh and Heywood (1983) in Papua New Guinea found higher morbidity and mortality levels in under five year olds on the lower anthropometric percentiles. In Norway, Waaler (1984) found a relationship between height and the chance of death in a study of over two million Norwegians. Those on the lower height percentiles had a significantly higher chance of death in the 30 to 60 years age group. Taller people survive, on average, longer.

Delgado et al (1987) recently demonstrated significant catch-up growth in acutely affected children in Guatemala. Treatment of the causative infection plus nutrition intervention did tend to return most children to their projectile percentiles. Lack of intervention probably partly explains why the percentiles of all birth groups in the Indian study of Bhargava et al (1979) remain so low, on the NCHS standards, as compared to those of the Aboriginal children in this study. However, it would seem from this and other studies mentioned that, once an infant is born SGA, the child's growth potential is permanently limited. This suggests that programs aimed, in the long-term, at improving the nutrition status of children in poorly-nourished populations must focus attention on preventing malnutrition and poor growth *in utero*.

The findings of this current study of the growth and development of 3,500 Aboriginal children from birth to 15 years of age in Far North Queensland, tend to support those of similar studies in other locations around the world. That is, growth retardation or starvation *in utero* has a permanent limiting effect on the future growth and development of Aboriginal children living in these communities. Presumably, this occurs in spite of the presence of close monitoring, surveillance and treatment/nutrition intervention being carried out by very active and capable Aboriginal Health Program Teams on these same communities.

A second significant finding made from this study was the rather depressing fact that, even if an Aboriginal child is born well-nourished and develops along a high projectile on the percentile charts for the first six months of age, he or she, soon after, slides down 20 to 30 percentile points or more in the weaning period, never

to fully recover. A further study will take a look at this phenomenon more closely, together with the data linking mother's birth details with those of her infants—the essential dyad of maternal and child health.

NOTE

1. This chapter is an abridged version of a paper in which the statistical significance of differences is discussed in greater detail.

6 A COMPARATIVE ANALYSIS OF ABORIGINAL AND NON-ABORIGINAL PERINATAL OUTCOMES IN THE NORTHERN TERRITORY

Alan Gray and Noor Khalidi

Perinatal survival is avoidance of stillbirth and death in the period immediately after birth. Earlier chapters in this volume deal with the more general context of survival in the first year of life after birth and beyond. To open the present discussion, it is sufficient to record that a major component of Aboriginal infant and early childhood mortality actually occurs at very early stages of life, that is in the neonatal period. The Australian Institute of Health (1988, 113) reports that Aboriginal neonatal mortality for the period 1984-86, based on data from South Australia, Western Australia, the Northern Territory and 14 communities in Queensland, was 2.5 times the rate for the total Australian population. For the Northern Territory alone the Aboriginal neonatal mortality rate has been recorded as twice as high as the average rate for Australia as a whole (Devanesen et al 1986, 59).

Comparative analysis of perinatal outcomes for Aboriginal and non-Aboriginal mothers has been undertaken previously. Seward and Stanley (1981), examining data from Western Australia for the period 1976–78, noted lower birthweights and higher perinatal death rates for children of Aboriginal mothers. An interesting result of this analysis was the finding that maternal age and parity appeared to play no particular role in determining the differences between Aboriginal and non-Aboriginal perinatal outcomes, although the authors cite substantial medical literature which would back an *a priori* judgement that such effects would be expected. Julienne (1983), analysing data for Aboriginal people in New South Wales for the period 1978–81, placed more emphasis in her conclusions on comparatively young age and high parity in Aboriginal mothers. Her study also used birthweights and perinatal death rates as the primary focus of analysis.

The first full year of operation of a new perinatal death collection in 1986, by what was then the Northern Territory's Department of Health (now Community Services and Health) provided a fresh opportunity to examine the nature of the disadvantaged entry to life of Aboriginal infants. The collection of these data is based on records of each birth in the Territory, from both private and public medical services. The records include details of previous pregnancies, the birth itself and outcomes. There are 1,220 Aboriginal and 2,318 non-Aboriginal cases for the year 1986. These form the basic data set for the analysis in this chapter, but the records for a total of 52 births, 51 of them to Aboriginal mothers, have been omitted from all the analysis here because they lack many of the data items.

Outcomes for Aboriginal and non-Aboriginal women

In 1986 the Aboriginal stillbirth rate in the Northern Territory was 19.7 per 1,000 total births to Aboriginal mothers. This was nearly four times the rate for non-Aborigines in the Territory and was also more than three times higher than the stillbirth rate of the total Australian population.[1] Similarly, the Aboriginal neonatal mortality rate (12.5 per 1,000 live births) was 2.4 times higher than both the rate for non-Aborigines in the Territory and the rate for the total Australian population.[2]

There is no doubt that the risk of perinatal and infant mortality is higher among those Aboriginal infants born with low birthweight, as has been amply demonstrated in a number of studies (for example, Seward and Stanley 1981; Julienne 1983; Gray 1987b). However, birthweight is only an explanatory variable for perinatal mortality in an intermediate sense—the fact that many babies with very low weights now survive is one of the achievements of obstetric practice at which we marvel. Essentially, low birthweight is usually associated with short gestation, poor physical development of the baby *in utero*, the physical condition of the mother, or some combination of these factors. A useful distinction is often made between premature low birthweight babies (those with gestation of at least 20 weeks but less than 37 weeks, and weighing between 500 gm and 2,500 gm at birth), and dysmature babies (with gestation of 37 weeks or more but low birthweight), because care for these two groups is somewhat different. Like birthweight, length of gestation is an intermediate variable determining perinatal survival.

Also among the intermediate variables determining perinatal survival is the score devised by Apgar (1953). This score, which ranges from zero to 10, measures an infant's condition at birth by adding component scores, each zero, one or two, for each of five vital signs: heart rate, respiratory effort, muscle tone, reflex irritability and colour. Apgar scores are calculated routinely in Australian hospitals at one minute of life and again at five minutes. A stillborn infant is almost by definition one who achieves an Apgar score of zero, although a substantial proportion of infants with scores as low as zero now actually survive in Australia. Recent research (Page et al 1986) favours splitting Apgar scores into two categories, namely 0-6 and 7-10, as a summary measure of neonatal condition. A score of six or less indicates difficulties with at least two of the five categories of vital sign, and if only two are affected then a score of six means that the difficulties must be severe for those two categories. All of these intermediate variables are very closely associated with perinatal outcomes in the Northern Territory in 1986, as is shown in Table 1.

The approach of this chapter is to regard perinatal death as mediated through these intermediate variables of the condition of the newborn infant. There are good reasons for adopting this approach, illustrated in the table. There are substantially greater numbers of infants born with poor condition, as measured by these intermediate variables, than actually die. This means that the association between these mediating variables of perinatal survival and background demographic and social factors can be analysed using larger numbers of cases than can be done with perinatal survival itself, and the analysis will be less confounded by the strength of medical intervention aimed at saving the lives of babies born in poor condition.

Table 1 Perinatal survival[1] of children born to Aboriginal and non-Aboriginal mothers, Northern Territory, 1986.

	Aboriginal		Other		Total		
	Died	Survived	Died	Survived	Died	Survived	Total
Birthweight							
<1500 gm	23	12	16	12	39	24	63
1500-1999 gm	6	25	1	29	7	54	61
2000-2499 gm	2	117	3	90	5	207	212
2500-2999 gm	2	354	1	423	3	777	780
3000-3499 gm	4	393	0	864	4	1257	1261
3500-3999 gm	1	169	2	663	3	832	835
4000+ gm	0	61	2	211	2	272	274
Gestation							
<33 weeks	25	23	12	25	37	48	85
33-36 weeks	8	118	2	108	10	226	236
37-39 weeks	3	387	4	548	7	935	942
40+ weeks	2	539	2	1003	4	1542	1546
Not stated	0	65	5	608	5	673	678
Apgar, 1 minute							
0	22	5	12	3	34	8	42
1	6	10	3	10	9	20	29
2	3	46	3	21	6	67	73
3	1	44	2	38	3	82	85
4	1	44	2	70	3	114	117
5	1	51	1	98	2	149	151
6	0	91	1	164	1	255	256
7	3	147	1	274	4	421	425
8	0	258	0	512	0	770	770
9	1	418	0	1056	1	1474	1475
10	0	17	0	46	0	63	63
Apgar, 5 minutes							
0	24	3	13	3	37	6	43
1	3	0	2	0	5	0	5
2	0	1	0	2	0	3	3
3	1	8	1	2	2	10	12
4	2	5	1	4	3	9	12
5	1	15	3	8	4	23	27
6	2	24	0	11	2	35	37
7	1	28	0	52	1	80	81
8	2	96	3	111	5	207	212
9	2	624	2	1464	4	2088	2092
10	0	327	0	635	0	962	962
Total	38	1131	25	2292	63	3423	3486

NOTE: 1. 'Died' in this table means that the baby has been recorded as a stillbirth or has been recorded as 'died' for method of separation from hospital.

Birthweights, gestational ages and Apgar scores are all less favourable for babies born to Aboriginal mothers than they are for babies of other mothers. Thus if we regard these intermediate variables as the ones we need to explain, then we will be likely also to achieve a good explanation of higher perinatal death rates for Aboriginal children. All of the intermediate outcome variables are somewhat inefficient for determining perinatal survival. Possibly the best is neither length of gestation nor birthweight, but the Apgar score, which is the assessed condition of the infant at birth. High Apgar scores of seven or more at one minute are associated with subsequent death in only five cases here, while there are slightly larger numbers of near-full-term and medium-to-heavy babies that fail to survive. In other words, we are possibly slightly less likely to make an error in identifying a baby as at low-risk when it has a high Apgar score than we are with birthweight or gestation alone. Because obstetric practitioners assign Apgar scores rather summarily ('Better give it zero for colour'),

they are sometimes sceptical about its utility. It is in fact a very useful measure, as can be seen here, and it is only a pity that the five components of the score are not kept for analysis.

Table 2 Mean birthweight and mean gestational age by pregnancy outcome for Aborigines and non-Aborigines, Northern Territory, 1986.

Pregnancy outcome	Birthweight (gm) Mean	N	Gestational age (weeks) Mean	N
Aborigines				
Live birth	3029	1147	38.4	1083
Stillbirth	1472	23	29.1	23
Non-Aborigines				
Live birth	3307	2305	39.0	1695
Stillbirth	1953	12	33.4	9

It is necessary to point out that the three mediating variables are related with each other, and it would be dangerous, *a priori*, to assume that their effects are independent of each other. This is illustrated in Table 2, which shows average gestational ages and birthweights for stillbirths and live births. The data in Table 2 show that live-born babies are nearly twice as heavy as stillborn babies no matter whether their mothers are Aborigines or non-Aborigines. This is no doubt directly related to the much shorter gestational age of the stillborn babies. As would be expected from what has already been seen in Table 1, a noticeable difference exists between the average birthweights of both live-born and stillborn Aboriginal and non-Aboriginal babies. While Aboriginal live-born babies are, on average, about 300 gm lighter than their non-Aboriginal counterparts, the non-Aboriginal stillborn babies are also more than 500 gm heavier than the Aboriginal stillborn babies on average. Again, as can be seen from the table, this could be related to the shorter gestational age, in the case of live births as well as stillbirths.

Those newborn babies who did not survive had, on average, very low body weight (less than 2,000 gm) at birth and reduced physiological capacity as measured by Apgar scores, as is shown in Table 3. The average birthweight of newborn babies who were discharged from the hospitals were up to 1.7 kg more than those who died and the average Apgar score at one minute was also up to five times more for those babies who were discharged than those who died.

Table 3 Separation of baby from hospital by mean birthweight, mean Apgar score at one minute, mean gestational age, and mean parity of mother for Aborigines and non-Aborigines, Northern Territory, 1986.

Indices	Aborigines Discharged or transferred	Died	Non-Aborigines Discharged or transferred	Died
N	1131	36	2293	24
Mean birthweight	3050	1449	3318	1606
Mean Apgar score	7.3	1.5	7.8	1.7
Mean gestational age	38.6	29.2	39.0	31.2
Mean parity	1.6	1.8	0.9	1.0

Any comparative analysis of Aboriginal and non-Aboriginal perinatal mortality stands or falls on whether the fact that a mother is Aboriginal is found to remain significantly associated with outcomes after controlling for background variables, for if Aboriginality determines outcomes then there can be no complete explanation of why Aboriginal births are different from non-Aboriginal births. The aim of the analysis in the rest of this chapter is therefore to determine which of a range of possible contributing factors are most closely associated with the intermediate outcome variables.

Before we proceed to an analysis of how the intermediate outcome variables are determined, we should be quite sure that there is no extra effect of Aboriginality in the way in which these variables determine perinatal survival. In fact, as the logit-linear analysis shown in Table 4 confirms, there is no statistically significant effect (at the 95 per cent level of confidence) of Aboriginal origin on perinatal outcome once the effects of the intermediate outcome variables are controlled. This is of course what would be expected.[3]

Table 4 Effect of intermediate outcome variables on perinatal survival, Northern Territory, 1986.

	Odds	Significance (Z)
Overall odds	0.0216	15.10 *
Aboriginal origin	1.3044	1.65
Birthweight <2500 gm	2.3294	3.81 *
Gestation <37 weeks	2.7701	4.49 *
Apgar 1 minute <7	4.3495	6.07 *

Likelihood ratio chi-square = 6.148 df 11, P=0.863

NOTE: In the following tables an asterisk signifies an effect significant at the 95 per cent level.

The results of this model-fitting show in quite a dramatic way how important each of the three intermediate variables shown is in mediating perinatal outcome, each acting individually. No significant interaction between any of the three measures was detected in the analysis. What the results mean can be summarised like this: an infant who obtains a low score on every one of the three explanatory variables has odds of not surviving or having been stillborn of about 0.61:1, which translates to about 38 per cent; an infant who obtains a low score on any of the three measures has odds of not surviving of about 0.07:1, or about six per cent; while a child who obtains a low score on none of the measures has odds of not surviving of less than 0.001:1, or 0.1 per cent.

It seems that a reasonable summary measure of the three intermediate measures is provided by identifying the rather large group of at-risk infants who have birthweight less than 2,500 gm, gestation of less than 37 weeks, or a one minute Apgar score of less than seven. There were 1,048 such infants recorded in the data set for the Northern Territory in 1986, 473 of them to Aboriginal mothers and 575 to other mothers.

Backgrounds of at-risk births

Quite a number of economic, social, environmental and behavioural factors may contribute to excess mortality risks for newborn Aboriginal children. While some of them are available for this analysis, it would be only fortune if they formed a reasonable proxy for all the differences in social environment which exist in an area like the Northern Territory. The most standard demographic background variable for analysis of perinatal and infant survival is age of mother. As noted in the introduction, previous analysis has not clarified the role that age can possibly play in determining successful outcomes for pregnancies of Aboriginal mothers.

The evidence for an effect of mother's age on risk for the child is just as confused in the international literature, summarised by Majumder (1989, 55-57). Part of the reason for this confusion is the fact that parity, the number of previous live births, progresses monotonically with age and different results of dissociating the effects of these two characteristics have been obtained. There is very substantial evidence in the literature for the effects on child survival of having or not having had previous children, although increased risk specifically in the perinatal period has not been extensively documented. The same observations are true for the effect of short intervals between births.

Extensive data on all these variables is available in the Northern Territory's 1986 data set. Additionally, the data set contains information about the antenatal care that mothers have received, in the form of date of first attendance for antenatal care and the total number of attendances. Table 5 shows percentages of births at-risk for a range of background variables drawn from the data set.

The table illustrates potently how Aboriginal births are at more risk than non-Aboriginal births, as measured by the three criteria of low birthweight, short gestation or low Apgar score, on every one of the background variables shown. Take first the case of mother's age. For both Aboriginal and non-Aboriginal women, the lowest levels of risk are in age group 25 to 29, which happens to be the peak age group for childbearing in non-Aboriginal women but not Aboriginal women. The data suggest a U-shaped risk curve, high for very young mothers less than 15 years, of whom almost all are Aboriginal, decreasing to the low point in age group 25 to 29, then gradually increasing again. Aboriginal mothers are shown to be at more risk in virtually every age group. As in the case of all the variables in this analysis, there is no way yet of knowing whether the differences which have been identified are actually due to age, or to Aboriginal origin either, or whether they constitute the hidden effects of some other variables.

Table 5 Percentages of births at-risk[1] for certain demographic/social variables, Northern Territory, 1986.

	Aboriginal		Other		Total	
	N	% at risk	N	% at risk	N	% at risk
Age						
<15	29	58	2	0	31	55
15-19	344	46	155	31	499	41
20-24	344	39	504	30	848	34
25-29	190	34	653	29	843	30
30-39	108	46	498	36	606	38
40+	9	44	13	31	22	36
(Missing cases)	638					
Marital status						
Not married	311	44	383	31	694	37
Married	817	41	1447	32	2264	35
(Missing cases)	529					
First antenatal visit						
None	79	57	11	55	90	57
First trimester	133	41	891	31	1024	33
Second trimester	594	41	629	29	1223	35
Third trimester	276	38	246	36	522	37
(Missing cases)	628					
Number of antenatal visits						
None	84	55	16	50	100	54
1-5	312	46	157	54	469	49
6-10	478	37	746	29	1224	32
11+	214	39	858	28	1072	31
(Missing cases)	622					
Gap since previous pregnancy						
<18 months	82	40	155	32	237	35
18-23 months	88	41	111	29	199	34
24-35 months	180	33	173	31	353	32
36-47 months	53	26	82	30	135	29
48+ months	86	33	145	34	231	33
(Missing cases)	2332					
Outcome of previous pregnancy						
Death of infant	102	43	389	33	492	35
Other/none	1025	42	1441	31	2466	36
(Missing cases)	529					
Number of previous pregnancies						
0	357	47	796	34	1153	38
1	277	43	592	29	869	33
2	213	38	282	32	495	34
3	140	30	111	32	251	31
4	74	46	42	29	116	40
5+	67	42	7	14	74	39
(Missing cases)	529					

NOTE: 1. 'At-risk' means having a birthweight of less than 2,500 gm, length of gestation of less than 37 weeks, or a one minute re Apgar score of less than seven.

Marital status appears from the data in the table to have no particular association with outcomes. There could, of course, be confounding influences in operation here. Julienne (1983) has identified babies of young, single mothers as being at considerably elevated risk in New South Wales.

For the next background variable, the date of first antenatal attendance, there can be less doubt about the existence of any confounding influences because the pattern shown is so clear-cut. It is that babies of women who did not receive antenatal care, whether they were Aboriginal or non-Aboriginal, were at an elevated risk.[4] Apart from having antenatal care at all, it did not appear to make much difference whether women started to receive care in the first, second or third trimester of the pregnancy. However, Aboriginal children remained at more risk for those women who did receive antenatal care.

If antenatal care has as much to do with birth outcomes as it appears, then the mechanism is obvious. It consists of identification of women at-risk in medical terms and extension to them of whatever special care might be necessary. The table also gives a clear picture of how much antenatal care is necessary to achieve the beneficial effect. It is clear that babies of women who have fewer than about six antenatal attendances are in the category of elevated risk, while numbers of visits after the sixth attendance make little difference to outcomes. Again there remains a large effect of Aboriginal origin in the comparisons.[5]

Seward and Stanley (1981) mention that little information has been available in the past on the effect of antenatal attendance on Aboriginal birth outcomes, although it has generally been assumed that Aboriginal women have had poorer attendance. Julienne (1983) found that information about antenatal attendance was not included in enough cases to obtain reliable indications of its effect. Gray (1987a) found that on the north coast of New South Wales poor antenatal attendance by Aboriginal mothers was associated with increased risk for their babies, and also noted that the main prospects for improving outcomes were from redoubling efforts to achieve better antenatal care. In that area, and in Julienne's earlier analysis of data from New South Wales, the extent of lack of antenatal care was similar to what has been recorded for Aboriginal mothers in the Northern Territory. What this emphasises is that extension of Aboriginal health services over the continent during the 1970s has created what is a flat background of health service delivery, the effects of which are likely to be similar throughout the country.

The next variable shown in Table 5 is the gap since the end of the previous pregnancy. The results shown here are perhaps surprising, but they should be regarded with considerable caution because of a very large number of cases with missing information. The evidence here is that there are smaller proportions of Aboriginal babies born after short intervals of less than 18 months than there are of non-Aboriginal babies. Aboriginal babies are more concentrated in the third year, 24 to 35 months after the previous pregnancy. Additionally, there is little evidence here that short or long intervals have much to do with risk to the child, although infants born in the third year after the previous child do seem to be at least risk. The proportion of short birth intervals for Aboriginal mothers is much less than would be found in other parts of Australia. For instance, Gray (1987a) found that 18 per cent of births, other than first births, occurred within 12 months of the previous birth and 37 per cent within 15 months, among Aboriginal women in northern New South Wales. Because of the large number of missing cases, it is possible that the Northern Territory data are misleading.

The outcome of the previous pregnancy, measured in terms of whether the pregnancy and neonatal period were negotiated successfully for the previous child of the woman, is another variable which shows little evident relationship to the outcome of the current pregnancy. Again this is surprising, as Gray (1987a) noted elevated risk in northern New South Wales for Aboriginal women whose previous pregnancies had had problems.

The number of previous pregnancies that a woman has had does seem to be related to risk of adverse outcomes, as the last panel of Table 5 shows. The pattern is

not as clear as in the case of age, with which it is naturally related, but there does seem to be an elevation of risk for first-born children of both Aboriginal and non-Aboriginal mothers.

There seem to be many overlapping influences on perinatal risk, on the evidence in Table 5; and Aboriginal mothers seem to be worse off in all cases. It is therefore somewhat surprising to find that in a multivariate analysis of all the influences considered together, quite a clear pattern emerges and it actually has rather little to do with Aboriginality or age. The relevant information from a logit-linear analysis is shown in Table 6.

Table 6 Effect of background variables on perinatal risk,[1] Northern Territory, 1986.[2]

	Odds	Significance (Z)
Overall odds	0.6957	5.60 *
Aboriginal origin	1.1271	1.85
Mother <20 years	0.9925	0.12
Not married	0.9431	1.14
First pregnancy	1.1437	2.93 *
Antenatal visits <6	1.4578	7.05 *
Interactions		
Aboriginal origin & age	1.1300	2.08 *
Aboriginal origin & antenatal visits	0.8285	3.52 *
Likelihood ratio chi-square = 25.39583 df 24, P=0.385		

NOTES: 1. 'At-risk' means having a birthweight of less than 2,500 gm, length of gestation of less than 37 weeks, or a one minute Apgar score of less than seven.
2. For interpretation of logit-linear models see Note 3.

This is a more complicated logit-linear model than was seen in the previous example, in that there are significant interactions between some variables. To simplify matters, some of the variables which were found to play no significant role in determining risk were omitted. These were the gap between the previous birth and the current birth, and the outcome of the previous birth. On the other hand, some variables which do not play an important role (except possibly in interactions) have been left in the model to illustrate their lack of import. These are mother's age and marital status, and Aboriginal origin.

Easily the most striking result of the modelling is the massively significant role that is played by antenatal attendances in determining risk for the infant. For women who have fewer than six antenatal attendances, the odds of having an at-risk infant are 1.01:1, which implies that about 50 per cent of them will have at-risk births, while for women with six or more antenatal attendances the odds are reduced to 0.48:1, resulting in 32 per cent at-risk births. All of the other effects are very small by comparison, although some are statistically significant.

Were it not for the finding of Seward and Stanley (1981) that mother's age does not play any significant role in determining Aboriginal perinatal outcome, it would have been a very surprising outcome of this analysis that age plays no important role; it certainly appeared to do so in the single-variable analysis in Table 5. It is possibly even more surprising that marital status plays no particular role, given the emphasis on the combination of young age and single status in Julienne's (1983) work. Age only plays a significant role in some of the interactions: the combination of

being young and Aboriginal as a mother (or equally the exact opposite, over 20 and non-Aboriginal, in the strange way in which these interactions operate), increases risk. Risk is reduced also for Aboriginal women who have few antenatal visits, but equally the much lower risk for non-Aboriginal women with adequate antenatal care is attenuated. With the exception of the first interaction term, all of the significant factors shown in the table actually operate in favour of non-Aboriginal mothers aged more than 20 with six or more antenatal attendances and not having their first birth. The child of such a woman has odds of being at-risk of 0.45:1, while the first child of a young Aboriginal mother with inadequate antenatal care has odds as high as 1.09:1.

Tough terms were set out for the program of analysis in this paper, namely that a successful comparison of risk for Aboriginal and non-Aboriginal mothers could not be defined to be 'successful' if it reached a conclusion that children of Aboriginal mothers were more at-risk because their mothers were Aboriginal. Table 6 does not quite achieve that aim, in that ethnicity is a significant component of some of the interaction terms. The main reason is that the definition of risk used for the analysis was very sweeping and included many infants at rather low-risk as well as some at very high-risk, so the analysis was made fuzzy by this lack of specificity. Suppose instead that we are highly specific. Instead of declaring a baby at-risk to be one weighing less than 2,500 gm or having less than 37 weeks gestation or having a one-minute Apgar score of less than seven, suppose that we stipulate high-risk consisting of the combination of these three things using 'and' instead of 'or', and perform exactly the same analysis as was performed in Table 6. The results are shown in Table 7, with all non-significant effects deleted.

Table 7 Effect of background variables on high perinatal risk,[1] Northern Territory, 1986.[2]

	Odds	Significance (Z)
Overall odds	0.0484	27.67 *
First pregnancy	1.3707	2.90 *
Antenatal visits <6	2.6054	8.75 *

Likelihood ratio chi-square=30.86468 df 29, P=0.372

NOTES: 1. 'At high-risk' means having a birthweight of less than 2,500 gm, and length of gestation of less than 37 weeks, and a one minute Apgar score of less than seven.
2. For interpretation of logit-linear models see Note 3.

This is a considerably different, and very much simpler, picture from the one shown in Table 6. Aboriginal origin is no longer implicated in any way in determination of risk, and the interaction terms which complicated the previous analysis are also no longer present. In fact, the analysis in Table 7 suggests that there are just four categories of women when it comes to determining high-risk pregnancy outcomes:

1. women having their first births with inadequate antenatal care, about 15 per cent of whom are expected to have high-risk babies;
2. women having subsequent births with inadequate antenatal care, about eight per cent of whom are expected to have high-risk babies;

3. women having first births with adequate antenatal care, about 2.5 per cent of whom are expected to have high-risk babies; and
4 women having subsequent births with adequate antenatal care, only 1.3 per cent of whom are expected to have high-risk babies.

Ultimately, this analysis has been highly successful, at least for the category of high-risk babies, and it throws into perspective the role of health services and the antenatal care they deliver to Aboriginal communities. High-risk has been shown to have nothing to do with Aboriginality, but a great deal to do with lack of antenatal care, and it affects women having their first children more than women who are already mothers. For the fuzzier category of women who are at-risk on at least one of the three criteria which have been used here, the analysis has not been so successful and it can be surmised that the reason is lack of specificity in the category of children at-risk.

Conclusions

In the end, the models which have been developed in this analysis provide a very simple explanation of why some children born to Aboriginal mothers in the Northern Territory are at more risk than children born to non-Aboriginal mothers, and the explanation has nothing to do with the ethnic origins of mothers and almost everything to do with lack of sufficient antenatal care for some women. Along the way we have ruled out age, marital status, the length of birth intervals and negative outcomes of previous pregnancies as useful components of explanation, even though most of these variables are superficially related in some way to apparent risk.

Yet the result will hardly be surprising to health workers, in the broadest sense, who are working in Aboriginal communities. We have the distinct feeling that they would respond to the results of the analysis with an emphatic 'we could have told you that!' Others might find it less evident that increased perinatal risk for Aboriginal children could be determined in the very simple way that has been demonstrated here.

Perhaps the strongest evidence in support is quite indirect, and it is historical. There can be no doubt that the vast improvements in survival chances for Aboriginal infants in the last 20 years or so coincided exactly with the expansion of basic health services to Aboriginal communities throughout the country, and even less doubt that the initial priority of these new services was maternal and child health. How did access to basic services improve perinatal survival as dramatically? The answer, according to the analysis in this paper, is that simple antenatal care, and the consequent identification and referral of pregnancies with medical complications, not only could have had the effect observed but that it very probably did.

The possible size of this effect can be illustrated by noting the numbers of Aboriginal mothers in the four categories set out at the end of the last section. Excluding a small number of cases with missing information, there were 140 first-births to women with less than six antenatal visits, and 263 subsequent births, and 226 first-births to women with six or more antenatal visits, and 499 subsequent births. Using the proportions calculated in the previous section, a total of more than 50

women would have had high-risk births, but if no women had had antenatal care then this total would have doubled to more than 110. This calculation, taking no account of possible improvements in the level of care during delivery during the last 20 years, already goes a long way towards explaining such decrease in perinatal mortality as has occurred.

There are dangers in developing models of explanation from data sets that are inherently limited in their scope. In the present case, there is the possibility that identifying women who do not seek antenatal care we are identifying a category of women whose lifestyle carries risk for the unborn foetus, and that the antenatal care if it had been obtained would not have had much effect on the outcomes. Let us recognise that possibility and, having noted that the explanation would have to apply equally well to Aboriginal and to non-Aboriginal defaulters, set it aside because the data are not available to address it. It will be an attractive explanation to people who are inclined to blame the mothers for their failure to seek antenatal care, or to those who do not believe that there would be any value in expanding Aboriginal health services any further.

What is really required, and we have said it before (Gray 1987a), is redoubling of efforts to obtain adequate antenatal care for all Aboriginal women. Once this much is recognised, it will be possible to come to grips with the real issues of reticence about women's business that hinder Aboriginal women from seeking the assistance in health matters that they want.

NOTES

1. The stillbirth rate for Australia is given as 5.3 per 1,000 total births for the period 1984-86 (Australian Institute of Health 1988, 113).
2. The neonatal mortality rate for Australia is given as 5.3 for the period 1984-86 (Australian Institute of Health 1988, 113).
3. The overall odds ratio is the odds of perinatal death against perinatal survival. This would be multiplied by 2.3294 to give the odds ratio for low birthweight babies, but the total odds ratio would be divided by 2.3294 to give the odds ratio for other babies. Effects are cumulative, that is they may be multiplied by each other for combinations of categories.
4. There were eight times as many of these mothers who were Aboriginal as non-Aboriginal.
5. There appear to be discrepancies in the data set between numbers of women who had no antenatal attendances, as measured by the two different variables discussed here. These discrepancies have not been corrected in the analysis because they are mostly due to missing data. The number of antenatal visits is completed more reliably.

DISCUSSION

Dr L Smith Very interesting points came out of some of those analyses. What I want to comment on is this: we are at a time when we have just had a report of an Aboriginal Health Strategy Working Party when a lot of new directions are being set in Aboriginal health in Australia and one of the things that's happening is that the NH&MRC has set up a small working group to identify priorities for research in Aboriginal health. That is a result of the fact that the NH&MRC's special purposes committee and public health research and development committee, which has access to a separate research grant fund, have identified Aboriginal health as a priority area for funding and research.

Having listened to the papers today, and thinking back to all the other sources of data that exist around the country, comparable to the Northern Territory perinatal collection, and the increasing amount of information that is becoming available through the vital statistics system, what is missing in this is a systematic plan for using this data so that in the first instance we can say that we have got the descriptive epidemiology right.

A lot of data are there. What is not happening is the participation of skilled analysts, and the people who can make sense of it, as we found when we looked at the situation with communicable diseases research at a comparable workshop recently. One of the possible outcomes of a workshop like this is for us to start setting a national agenda for research into Aboriginal mortality. One of the first things I would have thought one could do is to try to identify the priorities.

Alan Gray has argued on a number of occasions that what we should be looking at first is getting indicators of impact or excess risk, and that's a data issue, so there is a potential outcome from this workshop above and beyond just sharing of our data and our analysis, and that is to think about future directions for research. What I am talking about here is getting the statistical epidemiology right, but it goes beyond that because getting the descriptive epidemiology right is a way of identifying the priorities so we can talk about what the priorities are in the more conventional forums. If this is not tackled in a systematic way then what will happen is that people with strong research records especially in medical areas will be pursuing their own, doubtlessly important, areas but without it being done within any coherent framework. It is probably a group like this that's really best-equipped to talk about how that coherent framework might be developed. Obviously it's not up to me to determine how the workshop goes but it seems to me that this would be a very valuable outcome and with the time we have, we should at least give some thought and some discussion to it.

Dr D Hicks I worked in the Community Health Section of the Health Department in Western Australia from 1974 to 1982. I am interested in Ric Streatfield's paper. We have the same sort of information as Ric had, except that in 1982 instead of looking at birthweight and then seeing what happens, I looked at percentage of children below the third percentile. When you look at their weight, by the age of 12 months about 20 per cent of children are below the third percentile, then there is a gradual catch up over

the next two, three, four years. Then if you look at their height in the same way, the problem occurs later but it's much greater. It goes up to about 30 or 40 per cent and then there is a gradual catch-up later on.

I am not sure whether you can work out whether it's nature or nurture, whether the factors which occur prenatally are still working postnatally, or the mother who has the small baby is also the mother who doesn't look after her small child so well. I think that for this percentage of children below the third percentile at the age of 12 months, girls are better than boys, there are less girls below the third percentile. From 1974 through to 1982 there was a gradual decrease in the number of children below the third percentile. In the case of height, the third percentile started off at 40 per cent but it got down to about 30 per cent. Girls are better again, so I'm not sure whether you can separate the girls from the boys. This is definitely unpublished data that you will never see again.

The other comment I was going to make at this stage was that people who know the gestational age in Aboriginal women are much smarter than anybody in Western Australia. We could never work out the gestational age in Aboriginal women. In a retrospective survey I did on antenatal care and postnatal care among Aboriginal women who gave birth to live children in the first half of 1980 in Western Australia we attempted to measure it. Among other things we were examining the effectiveness of the community health services in Western Australia. After getting information from a [birth] notification first, I sent out a letter to the base where the application should have been looked after by community health services and the letter said, 'Dear Sister, I noticed that — has delivered a live boy baby. Could you please go through your notes and tell me the following points.' Twenty-three percent (107) of those Aboriginal women weren't clients of Aboriginal health services or community health services, so there were 362 that were clients.

Even excluding these cases it appeared that the organisation that is supposed to be looking after Aboriginal women didn't know what happened in 30 per cent of cases of Aboriginal births. And do they have a postnatal check-up? The percentages are 'Yes' 60 per cent, 'No' 13 per cent, and 'Don't Know' 27 per cent.

Community health services do not actually supply the service, most times they supply the motivation and the access and the health knowledge that the people could use. If a woman had a baby in the first half of the year you might expect that in the second half of the year she may be using some form of family planning, but 33 per cent of these cases have no record of family planning, 17 per cent had discussed but made no decision, 7 per cent wanted another child, and the remainder were using all the different methods of family planning. The reason I have raised all this here is just to demonstrate the problems you have trying to get decent information.

Dr A Gray I have a question for Ric Streatfield. I am trying to get some idea of what your data represents. It appears that it is not a single cohort of women, because there are different current ages. I am wondering if there is a possibility that the experience of women from earlier cohorts was different from children of women in later cohorts.

Dr R Streatfield You see that the per cent normal [size and weight] has steadily gone up from below 80 per cent to 100 per cent of one to four year olds. The per cent normal has always been pretty good, that is the wasting and stunting was always pretty low. Five to nine year olds are always the group that we have had trouble with in Aboriginal communities, mainly with wasting. The percentage normal has fluctuated a bit but now it is back up fairly high, obesity is next to zero; among 10 to 14 year olds obesity is increasing. What I wanted to show you was that the reason Tony Musgrave set up such a tight and precise anthropometric screening routine, originally back in 1972, was to draw up as soon as possible, growth standards for Aboriginal kids. I can show you community after community in the north and steadily over the years the percentage of normal right across the age range has approached the Harvard Standard which is the one that we use up in North Queensland still, so I am very much against any introduction of NH&MRC or any other standards other than NCHS. I am trying to get the Queensland Government to recognise the world standards so that we can compare our results with others.

I think if you try and draw up growth standards for different racial sub-groups, you are setting yourself up for failure. You are setting yourself up to accept less than optimal growth in children. We have shown that they can grow according to international standards. We haven't attained the Harvard percentile, but I wouldn't think that we should draw up growth standards for Aboriginal kids just because they haven't attained that yet. In Queensland the Aboriginal Health Program is officially still using Harvard Standards.

These figures are not from our best community but a middle-type community. You can see as the kids get older they do drop down, there are fewer normal, but it has gone up steadily. The percentage normal on Harvard Standards over those years has steadily improved, and if you look at Yarrabah and Weipa South they are far better than this, it is almost like parallel lines going up to the Harvard Standard. In these communities too it is not achieving the Harvard Standard yet, but I don't think we should settle for that.

Prof R Douglas I would like to ask a very broad question, connected with the fact that we have been looking at little fragments of the Aboriginal experience today. I think Neil Thomson underlined the fact that he was talking about an Aboriginal infant mortality rate experience based on about 35 per cent, at most, of the Australian Aboriginal community. Now it seems to me that perhaps one of the serious statements we can deliver is to indicate just how ignorant we are. That in 1989 our health system does not enable us to make inferences about Aboriginal mortality on more than one-third of the sample. Can any of those here who have worked on infant mortality data comment on whether it seems reasonable to infer that the remaining two-thirds are worse off or better off and on what basis they would make that inference? Can we come to any conclusion about how the system can capture a greater proportion of the Aboriginal mortality experience?

Dr A Gray For the first time in its long history the ABS in the 1986 Census coded the information which it has always collected on numbers of children ever born and

numbers of children still surviving of all women. This enabled us to look at Aboriginal child survival right across Australia. The ABS has brought out an analysis of that which shows that the experience in that 36 per cent is replicated in the other 64 per cent almost exactly and there is very little geographical variation in apparent Aboriginal child survival.

Dr T Threlfall If there is nothing specifically in being Aboriginal that dictates a high-risk in children, then what do you have in the way of comments on whether we still need funding for specific programs for Aboriginal children?

Dr A Gray Well, I think that it all has to do with the cultural appropriateness of health delivery systems. This is exactly what we need to be looking at in extending antenatal care to all Aboriginal women. It is only the fact that basic health services were expanded to all Aboriginal communities from the late 1960s onwards that the improvement has occurred in that way. If there is still a gap and that gap is caused by lack of antenatal care, then it means more effort should be put into culturally appropriate health services that are going to remove the barriers caused by reticence and inaccessibility of services, due to whatever reason.

Dr R Streatfield There are two aspects there. One was the studies on infant mortality which show little geographic variation. I did a study back in 1983 of 900 births in Cairns Base [Hospital] comparing perinatal mortality of Aborigines whose place of origin or home was Cairns compared with the northern communities. I remember the stillbirth rate and the perinatal mortality rate were significantly higher from the communities as compared with Cairns. Cairns' Aborigines had a comparable perinatal mortality rate to that of the white people in Cairns, whereas the communities had a significantly higher perinatal mortality rate. Most of that difference was stillbirths, and inadequacies in antenatal care. Perinatal mortality reflects directly antenatal care or lack of or inappropriateness of antenatal care.

When you start saying this to obstetricians and doctors in Cairns they pull their hair out in despair and abuse you from one end of the ward to the other, saying we provide Aboriginal women with the best care, better than even white women, but from my point of view and from what the Aboriginal women keep telling me, it is not appropriate care. It is not culturally appropriate and I think that is where we fall down. The obstetricians and doctors think they are providing the best care, but that is the best Western care, with our ultrasounds and all the rest. We actually fly women in from the Aboriginal communities throughout Cape York, to get foetal growth from them with our $250,000 ultrasound machine.

It has been shown elsewhere in the world that it is far more accurate to get an estimate of foetal growth with a tape measure and this is where we are going wrong. We haven't been able to convince the medical people in the hospitals to modify the care. We are just about to get there now with this study. I am hoping to convince the Queensland authorities, doctors, obstetricians, pediatricians and the health department to allow us to modify the antenatal services that we are providing. I designed a system whereby Aboriginal women can actually do most of the antenatal

and 'under-fives' clinic care with all these charts—simple charts. They don't require any special training at all. No one as far as I know has died from having their tummy measured with a tape measure. No one as far as I know has died from having their blood pressure taken by a non-medical person. So I think appropriate antenatal care is what is lacking.

Mr D McDonald I would like to just follow up Bob Douglas's question and Alan Gray's answer, with regard to the amount and spread of data, given that those national aggregates from ABS appear to be similar to these three States. Where do we go from there? Do we conclude that things are okay? I suspect not, we know even from the data that Neil Thomson has given us, there appear to be major differences between the three States. We have just heard there are massive differences between adjacent communities within Cape York Peninsula, so in terms of developing interventions these aggregate data are not of tremendous help, it appears.

I am wondering what is your next statement after having responded directly to Bob Douglas's direct question about the comparability of national aggregates and these three States. Where do we go from there in terms of need and possibilities of improving the quantity and quality of the data available?

Dr L Smith Well, you can continue to do indirect analyses, but Alan Gray is using census data that can be taken down to local areas. That is only a snapshot of one point in time. Clearly the need is for routine data through the ordinary national vital statistics system. We have been pushing this for 15 or 20 years and progress is occurring. All of the States with one exception are now attempting to collect all basic vital statistics data about Aborigines but that is a massive system with enormous inertia. Just to change the collection system to identify Aborigines, as Dan Black will be telling us tomorrow, is only the first step.

So you want to know what needs to happen next? A lot more resources need to be put into getting those fundamental collection systems for births and deaths and population to yield the data we need out of them. I mean if you could get vital statistics and population data down to the community level, we could be doing an enormous number of comparative studies to identify the hot spots. It's not really within our grasp yet, except maybe in the Northern Territory.

There has been a group set up as a result of the Aboriginal Health Strategy Working Party's recommendations. The Institute of Health has been told to get on with the job of improving the national vital statistics system. Unfortunately it is an enormous system and we are small.

Dr R Streatfield I think one of the easier ways of going about it is what I suggested to the Working Party. One of my submissions was that we design a community health profile and we let the Aboriginal people do it themselves. There are people capable with a bit of guidance in North Queensland communities of collecting their own data and then you sidetrack all the intermediate government services. As long as we can convince them it is for their benefit we can have profiles of every Aboriginal community in the nation. In the north we already have a profile on two or three of

our communities, on everything from birth to immunisation status to services, the under-fives, the antenatals, the number of diabetics, the number of disabled.

Dr A Gray I don't think we need to convince Aboriginal organisations about that issue. I am sure that if Shane Houston had arrived by now he would have said his organisation is doing exactly that in a number of communities and doing a health profile system. It's not the Ric Streatfield design but their own design. I had a chance to have a look at a survey and it is done for whole families at a time, so it provides a family health profile as well as a community health profile. I was impressed with it.

Prof R Douglas The issue is which communities have enough cohesion to get a profile and what proportion are in fact more widely scattered through urban areas in the larger cities. I mean is that where most of this two-thirds is or is most of it in cohesive but unsurveyed community groups?

Dr N Thomson In response to Ric Streatfield's comment, it is easy in the communities in Queensland, with a captive audience almost. When you look at New South Wales which has a very large proportion of the Aboriginal population inside its borders, then you haven't got communities of the same type and you can't collect data quite as readily as you can in North Queensland or the Northern Territory. From recollection, the census figure shows about 60 per cent of Aborigines live in non-remote areas, so therefore you have a problem there in obtaining any of the type of information we are looking at.

Mr G Briscoe It is idealistic in many ways to say that the clients should be controlling the data collection as well. You can always cook the data, which is one problem. Also it is a technical job. You can't expect Aboriginal people, on the cheap, to get the kind of information that keeps analysts going. It is you who have got to overcome this problem. You simply cannot take short cuts with Aboriginal health and opt for a solution that puts questionnaires into the hands of every Aboriginal person in this society. I repeat it is a technical problem. There are many ways of overcoming paucity of data collection through a logical process.

What you have got to do is try to improve the kind of system that we are stuck with, and that is a Federal system. We have got to try to work through the States and get better quality data from them. We certainly have to make the point that Aboriginal Medical Services must not only provide health care services but they should somehow justify the quality of the health services that they are giving. The State health departments must be included in this whole process as well.

One of the big problems of the quality of access to health care has been the share of the health care dollar that has been going through our community-controlled services. That has changed, as Len [Smith] said, but getting information from Aboriginal communities is not only a question of imposing your requirements on a particular group purely to get data. There may be other ways and means that you can do it without eroding the privacy that Aborigines have left. It is a real problem to be continually under the bloody microscope for the whole of your life. I mean how do

these people feel? For 14 years they have been put under a microscope. These are the kind of things Aborigines are continually faced with and that is really part of the problem of getting information. You have a small group of people that are continually under the microscope.

The same problem occurs with anthropologists who are continually looking at Aboriginal people all the time. So you are just suffering from these same kind of problems but you still have to come to grips with the kind of system that we actually live in—a political system, a power system, a resource system. Recognising these mechanisms is going to be most effective and if you think you are going to try and short-cut that by getting Aborigines to collect the data for you, it's not going to be a better quality. That is the point I want to make.

I have been involved in Aboriginal data collection programs everywhere. It is just a question of circumstances, but Aboriginal communities are different, so that if you develop a system in Queensland, it's going to strike different application problems in South Australia. I don't agree we ought to attempt it. We ought to still go along the track we have been going on, improving the political aspects to structure the data collection process, through the States and the Commonwealth. This problem should not be put back on the shelf.

Dr R Streatfield What I was suggesting is that we are forever accused of taking information out of communities and bringing it down to places like Brisbane and Canberra and using it for our own ends. I was trying to say that we should empower each community with the importance of doing their own statistics, collecting their own statistics as we have been doing in North Queensland for quite a while.

If you say it is technically difficult to do surveys—Aboriginal people have been doing this environmental survey in North Queensland since we started it in 1979 when I first arrived. It includes demography, housing, employment. I have done all the bunny work in actually analysing the findings that the Aboriginal people have collected and fed them back directly to the Councils, and as a result Councils have got water supplies, sanitation, sewerage and better housing because they saw that these things were important. All I am saying is perhaps we should add on deaths, stillbirths and everything else.

I know North Queensland is a bit different to everywhere else. We have probably got better educated people in our communities who can do these things. Perhaps in the Northern Territory it might be a bit more difficult but I really don't believe it takes that much to collect data. I am sure there are young people out there who can do it for the community.

Dr D Hicks I really think you do have to go for State-wide data. I am never sure when you collect from specified communities whether you're catching the abnormal cases. Do people go back to a community to die? Do pregnant teenagers leave a community? I am not sure that when you only do a community survey that you really measure all the events that are happening and I think so far as possible you should try to obtain State-wide data.

In this regard it is necessary to recognise the number of Aboriginal children and Aboriginal adults that are closeted away in hostels because of brain damage and things like that. When they eventually die they will never be recorded as Aboriginal if you are doing community surveys. There are children with meningitis who have been in hostels for the retarded for 20 years sometimes and they have been lost to the Aboriginal community. They are still Aboriginal. The same goes for psychiatric institutions and other places like that.

Prof R Douglas It seems to me it is not a matter of 'either/or'. It must be both. There is no conflict with what has been said about the usefulness of data for strengthening community development. What Ric Streatfield is describing can be used to monitor the progress of that community and it against the rest of society, but it is unquestionable that we have to insist that the official data capture systems do in fact give to Aboriginal people's health the same importance as we have given to the rest of society and that seems to me to be a glaring omission, that after 16 years we still don't have it covered.

Mr G Briscoe I agree it is a question of dealing with both. You have to try and get resources to communities to handle these problems. Of course Aboriginal people can handle forms; as the National Trachoma and Eye Health Program showed, helping people to understand the data forms fairly quickly is not the problem. The problem is that the fundamentals of data collection are not understood at Aboriginal community level. The question of mortality statistics and the kind of expert language that you use is not comprehended. If somehow you can overcome this, not necessarily by training Aboriginal statisticians, but if you can somehow develop systems whereby this expertise can be used by Aboriginal communities and they can in turn have access to a central body like The Australian National University where they can come and learn about it, that would be fine. But there must be resources as well. It is not just a matter of handing forms around. I know that this is a bit oversimplified; it is a question of doing both.

Dr L Smith I would like in passing just to reinforce that message too, that you have to do both. What is so frustrating about the fact that we can't get Aboriginal data out of the national vital statistics system, is that Aborigines are in there already. They are just not identified. All we want is to be able to say which of those events are Aboriginal and which are not. I still find it difficult after all this time to understand why it is as hard as it is.

There is another source of data apart from the community-generated data and apart from State data, and that is the Commonwealth through the ABS. The Bureau is just going into the field with the National Health Survey. It is not giving away any secrets to say that it took a lot of time and a lot of effort and a lot of persuasion to get the Bureau even to identify Aborigines within that survey. The data that will come out of it are not going to be very useful, obviously, because only a very small proportion of what is a very small sample will be Aboriginal. However one of the things that did come out of the discussions that led up to that National Health

Survey was, I believe, an acknowledgement on the part of the ABS that there is a case for developing a special national survey mechanism for surveying the Aboriginal population and if that was to be put in place an Aboriginal Health Survey would be probably the first cab off the rank in a national Aboriginal social survey program. Now as I have said all the Bureau has done is acknowledge that there is a case for looking at this. It seems to me that another possible outcome of this meeting might be for us to reinforce that message to the Bureau, that there is a need for data from a source like that that no other vehicle can produce. Obviously that is the case because nobody else can do a national survey of the whole Aboriginal population.

Prof R Douglas It is said that Aboriginal data are already there included in the general population data, but, the question is that are we capturing all of the events or not?

Dr L Smith I think it is almost certainly true that for instance a slightly higher proportion of Aboriginal deaths would not even be captured. That is a trivial problem compared to the problem of getting the Aboriginal deaths identified.

Dr N Thomson I want to sound a note of warning about the attention to antenatal care, because I think the notion of antenatal care embodies two different sides. Whether you call it culturally appropriate or not. One side of course is the giver of the care, who obviously does the job in screening for some avoidable things. But the other one is the recipient of the care, who actually has an awareness of the health risks that she is exposed to through the whole process of pregnancy. It is the recipient who is far more important.

Probably Alan Gray and Noor Khalidi did not pay enough attention to it. In their analysis, the number of [antenatal] attendances is something which ignores many more issues—the awareness of the woman of the value of monitoring her pregnancy, and looking for those risks, rather than the nature of the care given. I would suspect that those women who are moderately healthy and are aware of the risks probably do not even need to have hands laid upon them by a doctor, even if he has done it in a 'culturally appropriate' way. The real essence is the awareness of the woman, and I guess in a more general way the awareness of all of us, of health risks of certain behaviours. The awareness is probably the real determinant in reduction of risks rather than the antenatal care *per se*. It certainly is the case that screening for certain things and watching a number of things will be of help, but I think in general it is actually the awareness of the mother. It is more the maternal side of it rather than the doctor's side which is the crucial component of antenatal care.

By implication, just to put out another hundred doctors in the field or even a hundred sisters in the field who may be delivering that antenatal care is really a naive way of actually trying to address the issue. It is really a matter of making Aboriginal people who are receiving the care more aware of what it all means.

Mr G Briscoe I wanted to come in on this discussion a little bit earlier but I was prevented from doing so by the Chairman. I am a historian, so I don't deal in the area of demographers. I find it a bit daunting, but at the same time I should sound a

warning to demographers straying into areas that they are unfamiliar with, like use of words such as 'culturally acceptable'. It sounds really great but unless you can define what you mean by that I think once it gets out there with the resource allocators there is a certain amount of discrimination that takes place. People will only get a share of the resources if it is politically popular to do so. So 'culturally appropriate' means political possibilities. I find 'culture' a real problem because you don't know whether it is standing still or running around all over the place. You don't know whether it is dynamic or not, so you cannot gauge change over time. Try to get some term that is measurable and understandable. I don't know what that is.

Dr R Streatfield Let us take an example. In Yarrabah, the most recent stillbirths have occurred in young teenage girls who have not had anything to do with our doctor or nurses or anything at the Yarrabah Hospital. They have had no antenatal care at all and gone off and had their stillbirth. Now, to me 'culturally appropriate' is a service that will rope those people in. It must be Aboriginal women who are doing the antenatal care, and that is what the women keep telling us time and time again. It is the older women, the traditional birth attendants who have been suppressed by doctors and nurses and told that they cannot have anything to do with antenatal care. They are the ones we have to train up and give them a system in which they work out what is a culturally appropriate for their community. And what is culturally appropriate for Yarrabah is not culturally appropriate for Aurukun. They have to work that out, not us. I didn't mean to define it myself.

Mr N Khalidi I would like to make a comment on Alan Dugdale's paper, which was presented by Kim Streatfield. From his paper we get the impression that the birthweight has not improved during the last 20 years. But judging from the Northern Territory data, especially from Central Australia where I collected the data in a consistent manner for the period 1976 to 1986, the picture is different. Average birthweight has improved from 2,900 gm in 1976 to 3,000 gm in 1981 and further to 3,200 in 1986. Parallel to that, the percentage of low birthweight births has dropped from 19.5 per cent to 1976 to 13.7 per cent in 1981 and 11 per cent in 1986. Also the percentage of very low birthweight births dropped from 4.9 per cent in 1976 to 2.5 per cent in 1981 and 2.4 per cent in 1986. Even if you take the whole Northern Territory into consideration and compare them with Dugdale's data, then you would see that mean birthweight increased from 2,889 in 1984 to 2,986 in 1986 and also the percentage low birthweight births dropped from 20 per cent for 1984 to 16 per cent in 1986.

Dr A Gray I am sure that Kim Streatfield will be much more pleased with that trend than the trend that was actually there in those North Queensland communities—this is putting importance on nutrition.

I am still answering Gordon's point about cultural acceptability or cultural appropriateness. I think that there is a definite core meaning there. There was a paper which was in *Australian Aboriginal Studies*, on what they call the borning centre, or birth centre, in Alice Springs. In the concept that was being presented, the thing that comes through very clearly is the need for antenatal care and care during the period of

delivery so that Aboriginal women won't feel shamed or embarrassed. There are so many things where white staff with no previous experience of Aboriginal communities can go dreadfully wrong in the cultural context and that's all that I am saying. It is a recognition of those cultural differences that exist in Aboriginal communities.

Mr G Briscoe Using measurements like access to health services, the distribution of resources to Aboriginal communities are going to be correct. You can measure the numbers of people that are being serviced by a particular medical unit, whether it is general practice, whether it is secondary health care or whether it is primary health care. But, with culturally appropriate services it means that you can discriminate. For example, people at Murray Bridge will get a lower priority to their cultural appropriateness because people in Adelaide simply say that they don't have any culture. Similarly, people who are in the Far North will get more resources; or vice versa. If you allow people to discriminate, then inequality is developed—as a result of those kinds of flowery language if you like. Whereas if you stick to the meaning of measurable terms, then I think that distribution of resources will be much more equal. That is the point I want to make.

Dr N Thomson I was not arguing against culturally appropriate antenatal care, what I was really questioning is what antenatal care may achieve, in terms of outcomes that one wants. I do not believe it achieves great outcomes in terms of improving, in itself, the proportion of babies of low birthweight, for example. I think there is a lot of evidence from big studies in the United States that, in itself, antenatal care has very little contribution to those components. It certainly will have some impact on deaths because it will identify very early in the piece, those things which are preventable risk factors which may have an impact there, but in terms of actual low birthweight, which was one of the other points discussed today, I doubt whether in fact it has very much to do with it. I just wanted to respond that I was not arguing against culturally appropriate antenatal care, in case anyone misinterpreted that.

Dr J Stuart One of things that the Congress Alukura said that they wanted to do after the baby was born was to expose it to smoke of certain leaves which was a cultural practice of Aborigines in that area. When the white doctors at Alice Springs heard this they threw their arms up in horror and said, 'Oh my God, what a terrible thing, they are going to smoke the babies just after they are born'. As far as I am aware there is no randomised control trial that shows that exposing a new-born Aboriginal baby briefly to smoke from leaves does it any harm, and certainly some of the practices that pediatricians have done in the past such as taking babies away from their mothers for extended periods of time after birth have in fact been very harmful. I think one of the important points is that we really need to educate a lot of the white medical care givers about what is reasonable and what is not.

Dr R Streatfield In Alice Springs in 1986 at an NH&MRC meeting, the obstetrician at Alice Springs Hospital showed us a distribution of Aboriginal birthweight from the

previous 10 years. I was just astonished how it had moved up to the right, in other words there were far fewer low birthweights and the median had moved up. The obstetrician took her time in telling us why that was. I wondered how on earth Aboriginal women in Central Australia could improve their nutrition so much in such a short time, but it was not improvement, it was malnutrition, it was overnutrition, it was diabetes. These ladies were putting out bigger babies, more immature babies, over 4,000 gm—which moves the whole distribution curve up—but in fact it was far worse than having an undernourished full-term baby. So be careful of those figures because it may not mean improvements.

Section Two

State and Local Studies

7 ABORIGINAL MORTALITY RATES IN WESTERN AUSTRALIA

DG Hicks

Detailed Aboriginal mortality rates for Western Australia have not previously been published. These figures represent the culmination of a Public Health Department initiative commenced in 1979 when Aboriginal racial group information was first recorded on death certificates. It was not until 1983 that the quality of the data was sufficient to justify analysis.

Methods

The mid-year 1983 Aboriginal population of Western Australia was calculated as a 'best estimate' from data available from the 1981 Census, the Aboriginal Affairs Planning Authority and the Public Health Department. Each source tends to underestimate various segments of the Aboriginal population. In order to calculate a 'best estimate' at 30 June 1983 the Public Health Department figure for 1981 was taken as a starting point. Where the Public Health Department figure for that age and sex group was the larger in 1981, its 1983 counterpart has been taken as representing the 'best estimate' for that age and sex group. Where the ABS figure was the larger in 1981, the 1983 Public Health Department figure has been increased by the same percentage as existed in 1981 to provide a 'best estimate' for those age and sex groups. This process was not followed in males over 65 years nor females over 60 years where the ABS figures tended to indicate some acceleration of ageing. This is thought to be due to old-age pensions becoming payable at these ages. The number of children aged less than 12 months at 30 June 1983 was obtained from information supplied by Bedford (1984) from midwives' notification of birth data available in Western Australia.

The results relate to Aboriginal deaths which occurred in 1983 as opposed to Aboriginal deaths registered in 1983. This necessitated excluding 1982 deaths which were not registered until 1983 and including 1983 deaths which were not registered until 1984. Information concerning 1983 deaths and their cause was obtained from the Registrar General's Office and checked against information from the Public Health and Hospital and Allied Services Departments. Aboriginal people from different parts of the State residing in Perth also checked the death register for their part of the State for incorrect identification of Aborigines.

The method of Chandra-Sekar and Deeming (1949) was used as a theoretical check on the number of deaths. Their formula estimates missed events when two reporting systems are used, both of which are expected to sample independently from the same universe of events. Using this method the calculated number of deaths missed by both the Registrar General and the Public Health Department was 19.7. The actual number of missed deaths which were identified either from hospital data or the

local knowledge of Aboriginal people who checked the death register was 20. These results are encouragingly similar.

Deaths were coded by the ABS to the ICD-9 (World Health Organization 1977) according to the criteria laid down by the Bureau (Australian Bureau of Statistics nd). There were 15 deaths in 1983 which at the time of data collection had not yet been reported to the Registrar General and these were coded by the author from available information and using the same criteria.

Results

Table 1 gives the Aboriginal population and the number of Aboriginal deaths in each age and sex group. Among the 18,011 male Aborigines, 200 deaths occurred and there were 123 deaths among the 17,505 females. Table 2 (males) and Table 3 (females) compare the Aboriginal age standardised mortality rates for each major ICD-9 code to the rates for the total West Australian population 1979-82 as reported by Holman (1984).

Table 1 Number of deaths by age and sex for the Aboriginal population, Western Australia, 1983.

Age in years	Males Population	Deaths	Females Population	Deaths
0-	528	19	558	9
1-4	2144	3	2018	7
5-9	2673	0	2490	1
10-14	2582	1	2471	1
15-19	2242	9	2036	2
20-24	1742	5	1753	3
25-29	1364	10	1385	4
30-34	935	9	1064	9
35-39	769	7	809	6
40-44	677	10	646	6
45-49	595	12	561	8
50-54	488	14	495	9
55-59	312	19	353	10
60-64	312	12	290	7
65-69	204	14	185	14
70-74	186	10	191	13
75-79	114	17	94	5
80-84	83	18	64	2
85+	61	11	42	7
Total	18011	200	17505	123

Table 2 Age standardised mortality rate per 100,000 person years for male Aborigines, 1983, compared to total population, 1979-82.

Cause of death (ICD-9 code)	Population	Rate	Probability	Ratio
All causes	1745	718.2	.0001	2.4
Circulatory system (VII)	552	318.9	.0027	1.7
Respiratory system (VIII)	275	67.2	.0001	4.1
Accidents, poisoning and violence (XVII)	242	67.8	.0001	3.6
Neoplasms (II)	164	162.3	.9602	1.0
Genito-urinary system (X)	105	9.1	.0027	11.5
Nervous system and sense organs (VI)	103	10.0	.0045	10.3
Digestive system (IX)	78	24.3	.0524	3.2
Congenital and perinatal (XIV &XV)	73	22.2	.0048	3.3
Infectious and parasitic (I)	42	4.4	.0512	9.6
Mental disorders (V)	40	6.2	.0950	6.5
Symptoms, signs and ill-defined (XVI)	36	9.0	.2006	4.0
Musculoskeletal (XIII)	19	1.4	.2186	13.4
Endocrine, nutritional and metabolic (III)	18	11.2	.6100	1.6

Table 3 Age standardised mortality rate by 100,000 person years for female Aborigines, 1983, compared to total population, 1979-82.

Cause of death (ICD-9 code)	Population	Rate	Probability	Ratio
All causes	1178	403.0	.0001	2.9
Circulatory system (VII)	389	182.2	.0019	2.1
Neoplasms (II)	169	98.9	.1142	1.7
Accidents, poisonings and violence (XVII)	129	25.6	.0007	5.0
Respiratory system (VIII)	128	24.4	.0093	5.2
Endocrine, nutritional and metabolic (III)	111	10.7	.006	10.3
Genito-urinary system (X)	110	7.5	.0056	14.7
Digestive system (IX)	31	13.2	.332	2.4
Symptoms, signs and ill-defined (XVI)	30	5.3	.1738	5.7
Mental disorders (V)	26	3.4	.1442	7.7
Congenital and perinatal (XIV & XV)	26	17.5	.4296	1.5
Infectious and parasitic (I)	25	3.4	.1142	7.2
Nervous system and sense organs (VI)	5	7.0	.6456	0.7

This population was chosen for comparison because it is relevant to health planners in Western Australia. Although the same years are not used, Holman's data collected over the preceding four year period was immediately available and relevant to the health of Aborigines in Western Australia. For both populations the mortality rates were standardised to the world population proposed by Doll (1976). As Aborigines represent only three per cent of the total Western Australian population, their higher mortality rate, which is included in the total Western Australian population rate, does not unduly influence the total mortality rate.

For all causes of death the Aboriginal male rate was 2.43 times the total Western Australian rate and for females it was 2.92 times higher. In both groups and for both sexes, diseases of the circulatory system were the most common cause of death. For Aboriginal males, the second most common cause of death was diseases of the respiratory system followed by accidents, poisonings and violence third, then neoplasms, diseases of the genito-urinary system and diseases of the nervous system and sense organs. For all these causes except neoplasms, the difference between Aborigines and the total male population was statistically significant at the .005 level. For Aboriginal females, the second most common cause of death was neoplasms followed by accidents, poisonings and violence third, then diseases of the respiratory

system, endocrine, nutritional and metabolic diseases and diseases of the genito-urinary system. Again, for all causes except neoplasms, the difference between the rates for Aborigines and the total female population of Western Australia was statistically significant at the .01 level. Age standardised rates give an overview of the data and permit the only interpretation possible when small numbers of deaths are involved. However, a single summary figure hides the excess Aboriginal mortality in young to middle-aged adults.

Table 4 shows the male and female Aboriginal age-specific mortality rate ratios for each five year age group relative to those for the total population of Western Australia. In males, the Aboriginal rate is 3.6 times that of the total population in the 15 to 19 year age group and rises to 7.6 in the 30 to 34 year age group before gradually declining to near unity at ages over 70 years. The pattern is similar in females, with the Aboriginal rate being twice as high in the 15 to 19 year age group and up to 10 times higher in the 30 to 39 year age group before approaching unity at age 75 years.

Table 5 compares the Aboriginal and total Western Australian rates for each sex for diseases of the circulatory system. Below the age of 20 there were few deaths related either to congenital or rheumatic heart disease. In the 25 to 44 year age group, Aboriginal males have a mortality rate for diseases of the circulatory system approximately 10 times higher than the total population. For females, the Aboriginal rate ratio is up to 20 times higher in the 30 to 44 year age groups. Most of these Aboriginal deaths were related to atherosclerosis.

For Aboriginal males aged 15 years and over, the age-specific mortality rate from accidents, poisonings and violence is approximately three times that of the total Western Australian population as shown in Table 6. For Aboriginal females, the pattern is different with the rate being at least 10 times higher in the 25 to 49 year age group but then virtually no Aboriginal female deaths occurring after the age of 50 years.

Table 4 Mortality rate ratio for all causes of death in the Aboriginal population, 1983, compared to total Western Australian population, 1979-82.

Age in years	Male	Female
0-4	2.8	2.8
5-9	-	2.1
10-14	1.1	2.1
15-19	3.6	2.4
20-24	1.9	4.0
25-29	6.0	7.2
30-34	7.6	16.6
35-39	5.3	9.6
40-44	6.0	6.5
45-49	4.4	6.3
50-54	3.9	4.6
55-59	5.3	5.0
60-64	2.0	2.5
65-69	2.2	5.2
70-74	1.1	2.8
75-79	1.9	1.2
80-84	1.8	0.4
85+	0.9	1.1

Table 5 Mortality rate ratio for diseases of the circulatory system in the Aboriginal population, 1983, compared to total Western Australian population, 1979-82.

Age in years	Male	Female
20-24	-	-
25-29	10.0	12.4
30-34	26.8	28.5
35-39	7.7	18.9
40-44	9.6	21.3
45-49	4.6	11.7
50-54	3.3	4.3
55-59	3.8	3.4
60-64	1.7	1.8
65-69	1.5	4.1
70-74	1.1	2.8
75-79	0.9	0.8
80-84	1.3	0.3
85+	0.4	0.7

Table 6 Mortality rate ratio for accidents, poisoning and violence in the Aboriginal population, 1983 compared to total Western Australian population, 1979-82.

Age in years	Male	Female
15-19	3.5	1.8
20-24	1.9	7.3
25-29	4.0	12.4
30-34	3.9	20.4
35-30	1.6	16.4
40-44	3.9	10.5
45-49	6.0	8.7
50-54	10.4	-
55-59	3.5	9.4
60-64	-	-
65-69	5.7	-
70-74	-	-
75-79	6.0	-
80-84	5.7	-
85+	3.9	-

For other causes of death, the actual number of Aboriginal deaths is small leaving many empty table cells so that rate ratio comparisons tend to fluctuate widely leading to the possibility of high figures being quoted out of context and causing misrepresentation. Even in the above tables, individually high rate ratios may be spuriously high due to the random occurrence of one or two extra deaths in a particular age group.

Diabetes was the major cause or a contributing factor in six per cent of deaths in Aboriginal males aged 20 years and over and in 16.5 per cent of females. This prevalence of diabetes among deceased Aborigines is less than that usually reported in diabetes prevalence surveys of Aboriginal communities (Wise et al 1970; Bastian 1979). It may mean that doctors sometimes fail to record diabetes on the death certificate or that the prevalence of diabetes across the whole State is lower than in the communities where surveys have been carried out. It may also mean that in some cases, diabetes has remained undiagnosed at the time of death.

Alcohol was either the main cause or a contributing factor in 11.9 per cent of Aboriginal deaths in males aged 15 years and over. In females it was lower at five per cent. The author gained the impression that there were several deaths in which alcohol was probably a contributing factor, but it had not been mentioned on the death

certificate, so the above figures represent a minimum estimate of the mortality contribution made by alcohol.

Conclusions

The mortality rates obtained were checked by Gray (personal communication) using a growth balance method for determining under-enumeration of deaths in small population groups. He considered that the correction factor needed for males was 1.0091 and for females 1.0280. This implies that the figures are close to being correct or that both the numerator and denominator have been under or over-estimated by a similar margin. These results are similar to those previously reported for regional groups of Aborigines in Queensland and New South Wales but because of the methods of standardisation used the results are not directly comparable. In Queensland, the Aboriginal mortality rate on 14 reserves was standardised to the total Queensland population and results for both sexes combined. The age-standardised mortality rate ratio was 2.6 (Lincoln et al 1983). In New South Wales, the mortality rate for rural Aborigines was compared to total New South Wales mortality, the latter being indirectly standardised to the Aboriginal population. The all causes age-standardised mortality rate ratio was 4.5 for males and 4.2 for females (Smith et al 1983).

This is the first time that age-standardised and age-specific Aboriginal mortality rates have been reported for Western Australia. They indicate that in both sexes, Aboriginal mortality is more than twice the rate experienced by the total Western Australian population. Age-specific rates for cardiovascular disease and accidents, poisonings and violence show that there is a large excess loss of life in young to middle-aged Aborigines of both sexes.

Some of the diseases causing the excess Aboriginal mortality rate are only partially amenable to intervention by medical practitioners, the necessary interventions having to occur earlier in the disease process. Neither do the interventions rest solely with the Aborigines whose economic, employment, geographic and social situations place them in a more vulnerable position than the rest of the Western Australian community. Any improvement has to come from wide-ranging changes which will include the involvement of politicians, social planners, Aborigines and the health profession.

8 WHAT DO WE KNOW ABOUT PREMATURE ADULT MORTALITY OF ABORIGINAL AUSTRALIANS?

Aileen J Plant

This chapter will discuss some aspects of premature adult Aboriginal mortality in the Northern Territory from 1979 to 1983 and where possible, make some comparisons with data from other States. It will examine potential years of life lost in terms of age, sex, geographical area and cause. It will then examine the causes of excess mortality in Northern Territory adult Aborigines with a more detailed examination of the specific cause of death within the categories of deaths attributed to the circulatory and respiratory systems.

The reasons for this approach include increasing concern by both Aborigines and health professionals at the alarming middle-life mortality of Aboriginal Australians and the necessity of looking at data already collected to determine points that are potentially suitable for public health action. Data giving both age-specific death rates and the relative risk of death at different ages and from various causes can be calculated. However the risk of dying is only one measure of mortality and does not give any indication of the size or importance of the problem in public health terms. For example, is the relative risk of death for diseases of the musculo-skeletal system at 5.7 for Aboriginal males, more or less important than the relative risk of death from circulatory system disease of 2.1? Given that in the five years in the Northern Territory only four males died of diseases of the musculo-skeletal system and 248 Aboriginal males died of diseases of the circulatory system it is obvious from a population perspective that diseases of the circulatory system are far more important.

The main purpose of this chapter is to present some of the data in a form that is more useful from a public health perspective, that is both in potential years of life lost from different causes and in examining the contributions of different causes to the excess mortality. It concentrates on premature adult mortality, that is mortality occurring between 15 and 64 years of age.

Methods

This study consists of all Aboriginal deaths from the start of 1979 to the end of 1983, of people usually resident in the Northern Territory. Two major data sources were used to ensure all deaths were counted and methods to ensure that deaths were not double counted were incorporated in the database construction (Plant 1988). Where necessary other data sources were used to validate data. Age and sex data were obtained from both sources. Cause of death was that coded by the Australian Bureau of Census and Statistics (now ABS) which in turn is derived from that recorded by either the medical practitioner or the coroner on the death certificate. The codes of cause of

death were derived from the ICD-9 (World Health Organization 1977). The denominator population used was that obtained at the 1981 Census, which was the mid-point of the study.

The data for comparison with other States was obtained from two sources: Smith et al (1983) examined mortality in country regions of New South Wales and Hicks (1985a) examined Aboriginal mortality in Western Australia in 1983. From the data, age- and sex-specific mortality rates were obtained and compared with rates in New South Wales and Western Australia. It was also possible to examine the relative risk of death by cause. The indirect method of age-standardisation was used with the total Australian population used as the reference population.

The potential years of life lost were calculated by using the life expectancy that would have been expected at the mid-point of each age group if the Aboriginal population had the same life experiences as Australians of the same sex, and multiplying this by the number of deaths at this age group. This was then converted to a rate per 100,000 using the population estimates from the 1981 Census data. Both total and age-specific potential years of life lost rates per 100,000 were calculated for males and females from the Northern Territory, Western Australia and country New South Wales. The proportion of potential years of life lost due to various causes was examined for male and female Aborigines in the Northern Territory. The excess mortality was calculated by subtracting the difference between the observed deaths and those expected if Aborigines had enjoyed the same death rates as other Australians. Excess potential years of life lost were calculated for male and female Northern Territory Aborigines within age groups.

The proportions of excess mortality due to various causes by age groups were calculated for males and females. Age groups were then aggregated into larger groups. The categories of disease within diseases of the circulatory system and the respiratory system were examined in greater detail, by determining the proportion of excess mortality due to different diseases within the major disease category and within age groups.

Results

There were 1,651 Aboriginal deaths in the Northern Territory in the period 1979 to 1983. The pattern of age-specific death rates is the same as that noted in other parts of this volume. As in other States, the Aboriginal death rates are all substantially higher than their age and sex equivalents for the total Australian population. The rates themselves are not reproduced here for this reason, and instead the differentials are presented in detail. However, an important point to note in the graphical comparisons given in other parts of this volume is that the Aboriginal rates do not have the 'honeymoon' period of a levelling of death rates at ages 15 to 34 years like other Australians; instead, the death rates continue to rise.

Figure 1 Relative risk of death by sex at each age group for Northern Territory Aborigines, 1979-83.

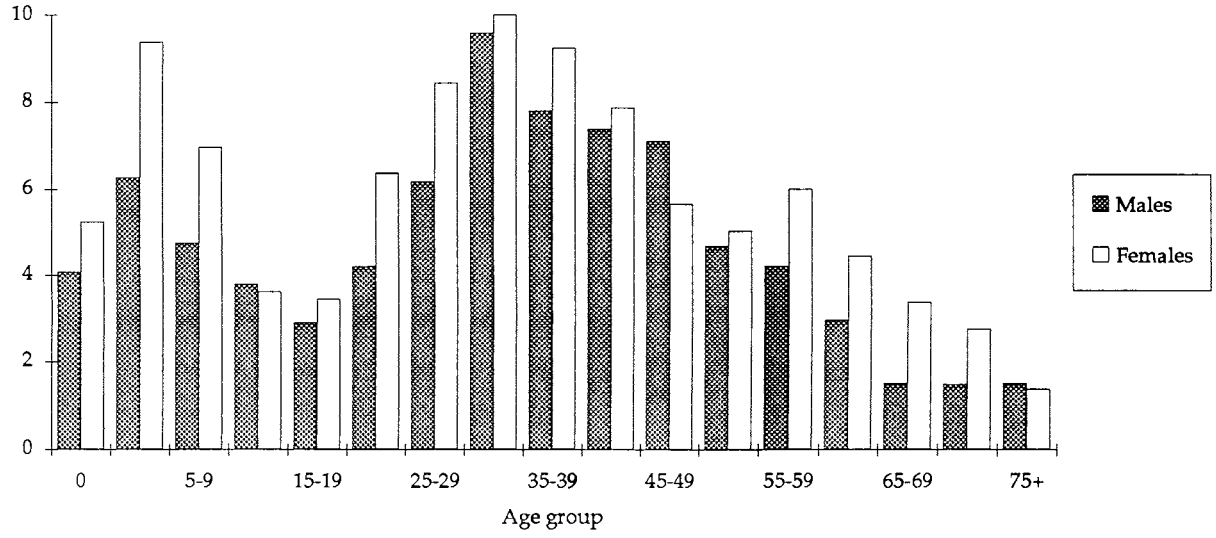


Figure 2 Total potential years of life lost per 100,000 population for Aborigines by region, compared to all Australians, 1979-83.

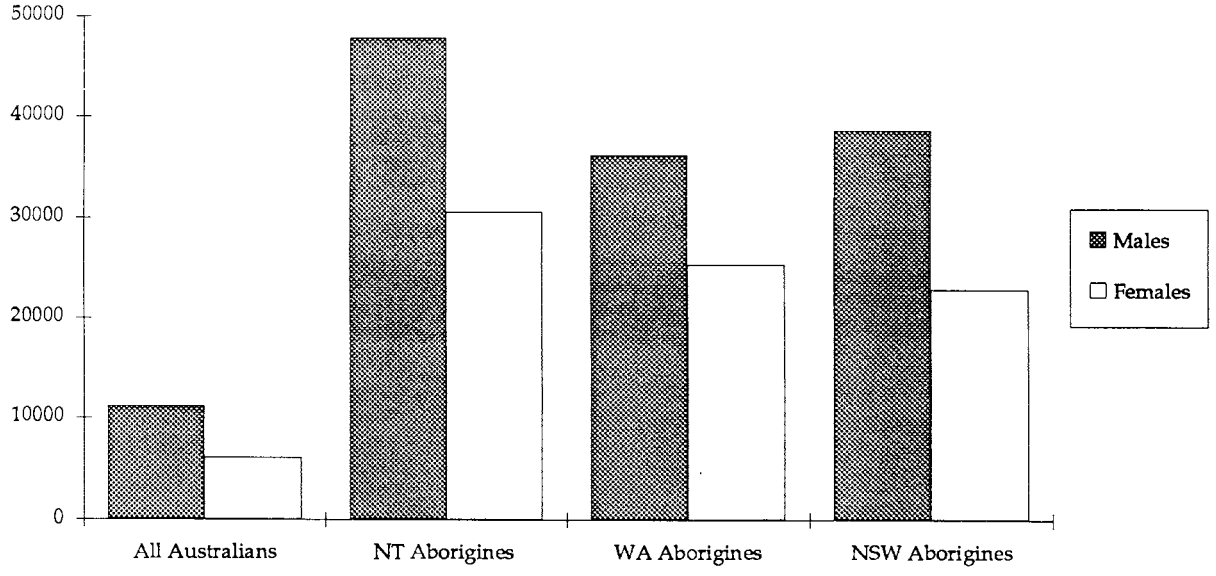


Figure 3 Potential years of life lost per 100,000 population due to premature death in adult males for Northern Territory Aborigines, compared to all Australians, 1979-83.

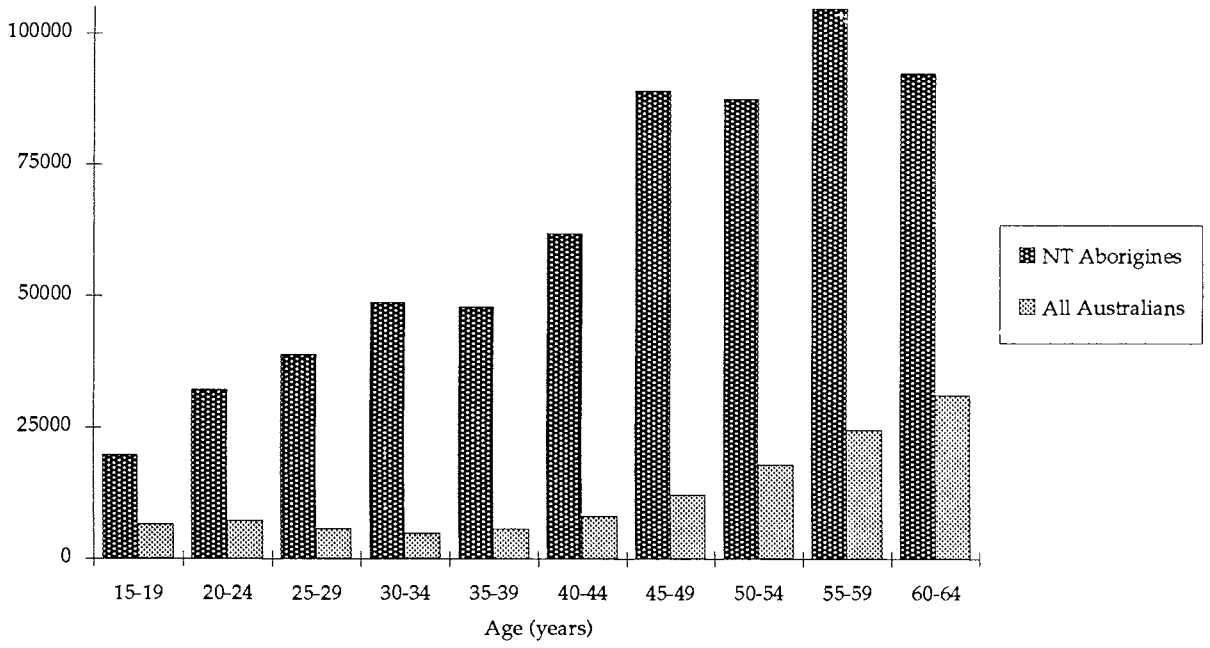


Figure 4 Potential years of life lost per 100,000 population due to premature death in adult females for Northern Territory Aborigines, compared to all Australians, 1979-83.

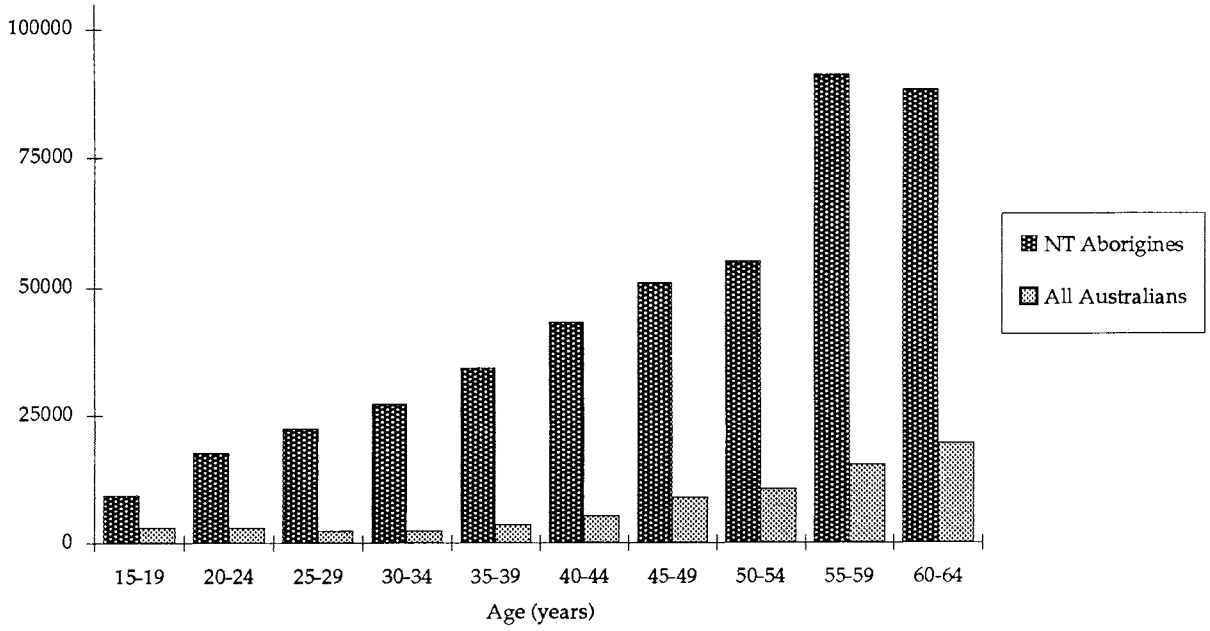
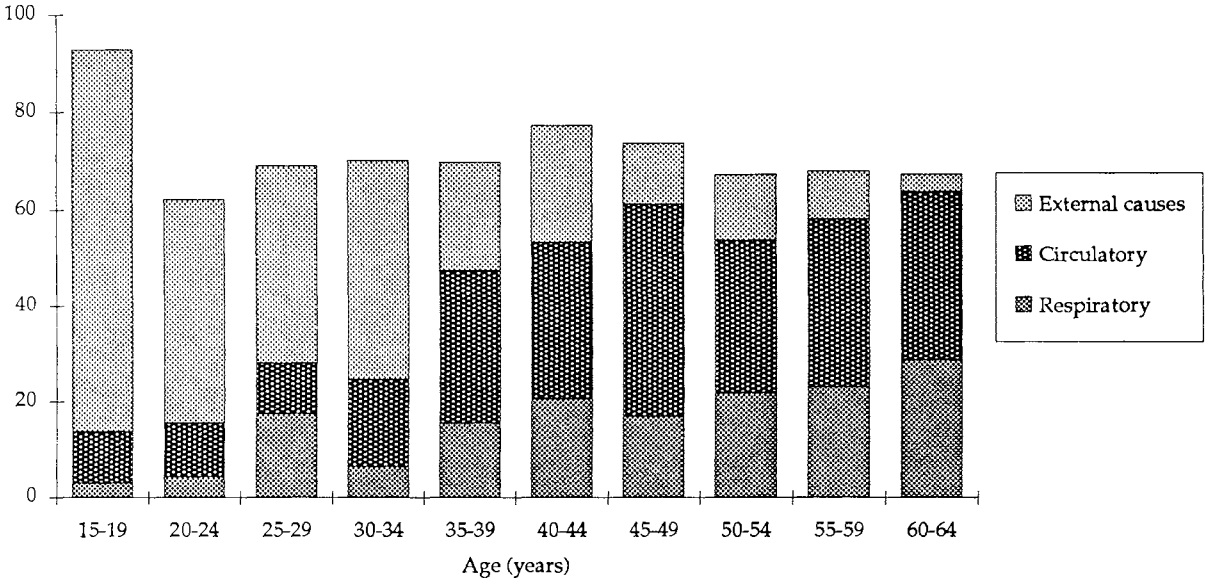


Figure 1 shows the relative risk of dying for Aborigines in the Northern Territory compared with the total population of Australia by age and sex, reaching a peak relative risk of death of approximately ten at the 30 to 34 age group for both males and females. Figure 2 indicates the total potential years of life lost due to premature mortality per 100,000 population for Australia and for Aborigines in the Northern Territory, Western Australia and country New South Wales. Deaths below the age of 65 are considered premature. Of particular note is that all Aboriginal potential years of life lost due to adult premature mortality are in excess of three times that seen in the rest of Australia with the potential years of life lost in the Northern Territory being about five times higher. Examining the potential years of life lost due to premature adult mortality in age and sex strata again shows the disparity between Aborigines and the rest of Australia (Figures 3 and 4). Very similar results are obtained when data from other States are used.

Figure 5 Potential years of life lost attributable to circulatory system diseases, respiratory system diseases and external causes for male Northern Territory Aborigines, 1979-83 — proportions.



Figures 5 and 6 show the proportion of potential years of life lost attributable to circulatory system diseases, respiratory system diseases and external causes in Northern Territory Aborigines. None of the other causes (the difference between the top of each column and the 100 per cent level) is prominent. This is particularly notable in males. By subtracting the figures for the total population from those for Aborigines in Figures 3 and 4, we obtain a measure of the excess potential years of life lost per 100,000 population within each age and sex group in Northern Territory Aborigines (see Figure 7).

Figures 8 and 9 display the proportion of excess mortality in each age group attributable to various causes according to the ICD-9. Only the major causes of excess mortality are shown. Of particular importance in young adult Aboriginal males is the

proportion of deaths due to external causes; this rapidly decreases with age but still accounts for a significant proportion of excess mortality. Respiratory disease and circulatory disease account for proportions that increase with age. A similar pattern, although less affected by external causes, is seen in Aboriginal women. There is also a significant proportion of excess mortality due to pregnancy-related deaths in the childbearing years, but this is not shown in Figure 9.

Figure 6 Potential years of life lost attributable to circulatory system diseases, respiratory system diseases and external causes for female Northern Territory Aborigines, 1979-83 – proportions.

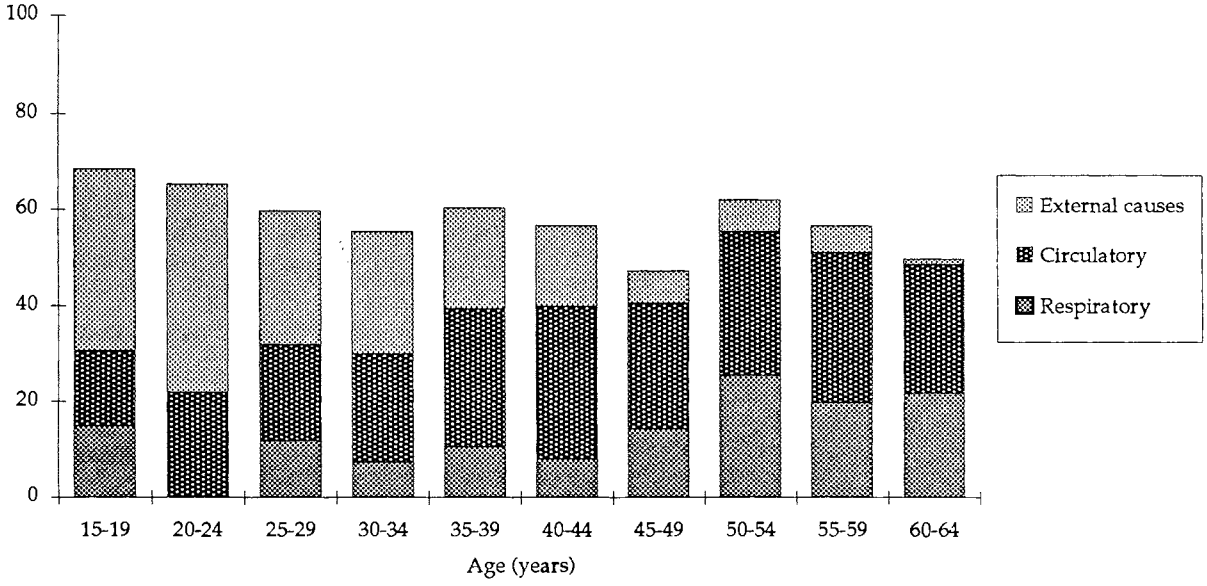


Figure 7 Excess potential years of life lost by Northern Territory Aborigines by age and sex, 1979-83.

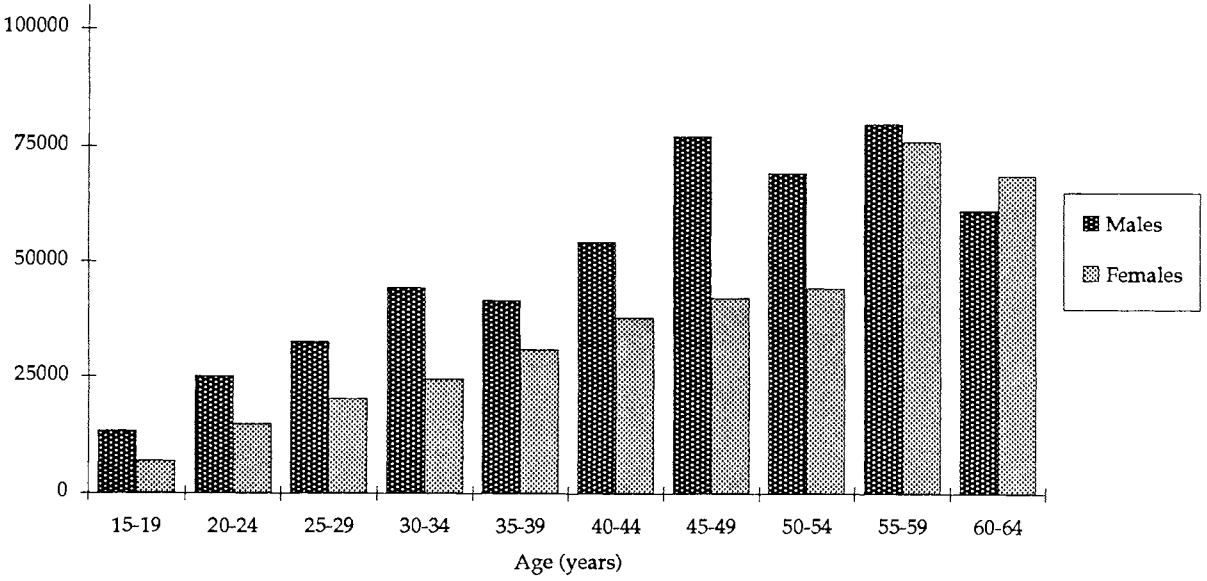


Figure 8 Percentage of excess mortality due to different causes in Northern Territory Aboriginal males, 1979-83.

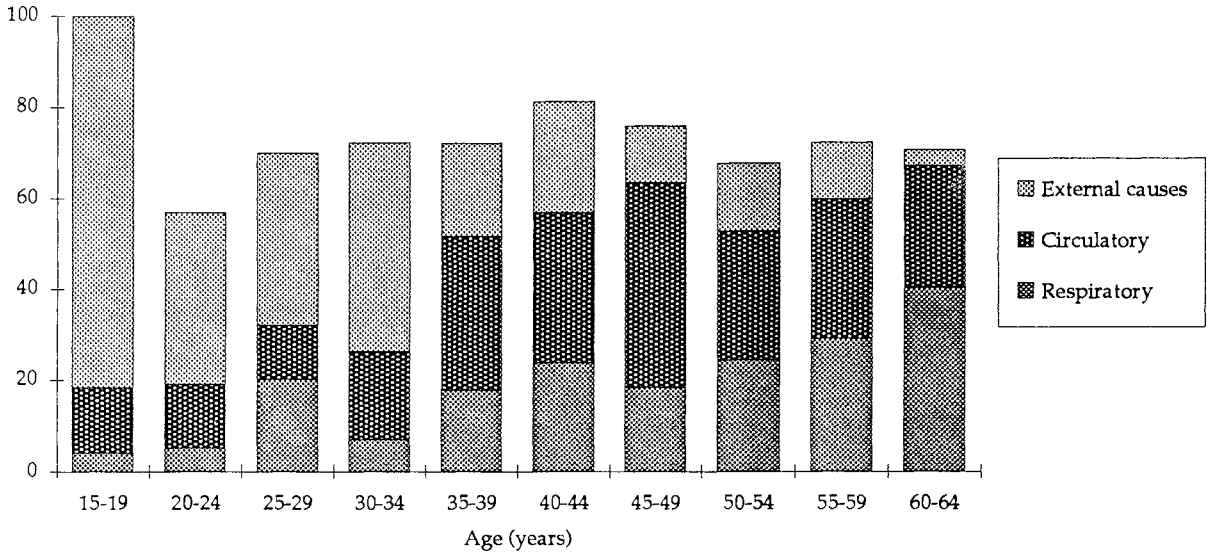
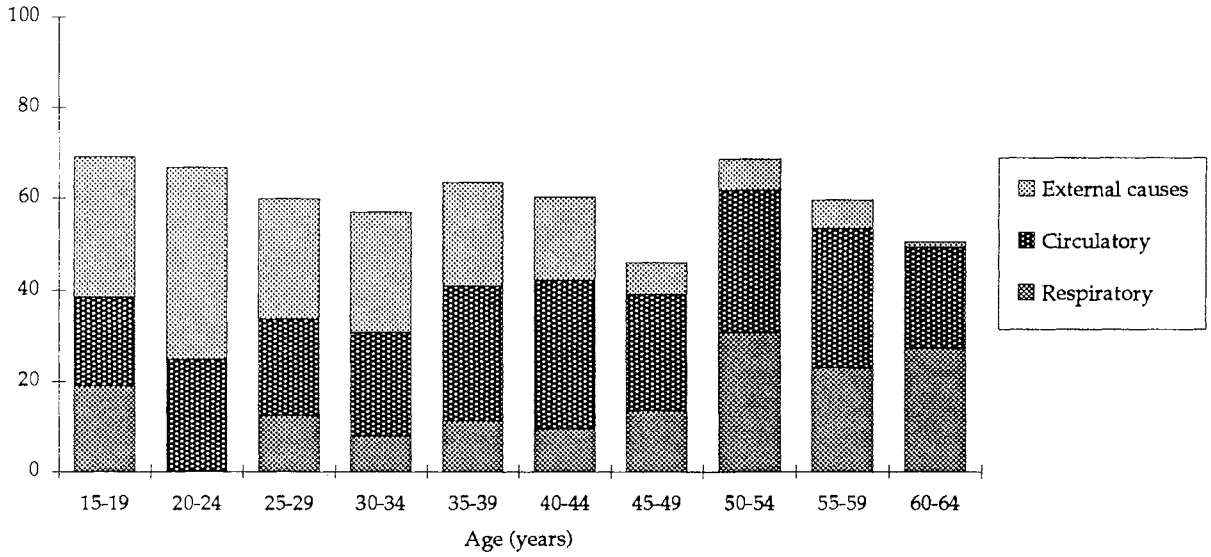


Figure 9 Percentage of excess mortality due to different causes in Northern Territory Aboriginal females, 1979-83.



The excess mortality due to diseases of the circulatory system in each age and sex stratum can be further divided into the proportions attributable to various disease categories. When this is done, it is found that chronic rheumatic fever accounts for the majority of the excess mortality in 15 to 24 year olds, 53 per cent in the case of males and 89 per cent in the case of females, and it remains the largest single category for females at ages 25 to 34 and 35 to 44 as well. Particularly for men, it is quickly replaced by ischaemic heart disease, hypertensive heart disease and cerebro-vascular disease in the older age groups. In the case of men, ischaemic heart disease is the largest single category by age group 35 to 44, accounting for 44 per cent of excess

mortality, and it remains the largest category in age group 45 to 54, accounting for 31 per cent of the excess. By age group 55 to 64, cerebro-vascular disease accounts for 24 per cent of the excess and is the largest single category. In the case of women, ischaemic heart disease is the largest category in age group 45 to 54 (25 per cent of the excess), and cerebro-vascular disease is the largest category of the excess for age group 55 to 64 (30 per cent). For both sexes, there is also a substantial proportion of mortality attributed to 'other' forms of heart disease, and it can be surmised that many of these cases are inadequately classified.

The same procedure can be followed for excess mortality due to respiratory disease. For both males and females 60 per cent or more of the excess mortality is due to pneumonia (including influenza) and chronic obstructive airways disease. In the case of males, mortality from pneumonia constitutes the largest category of excess mortality below age 55, rising from less than 40 per cent in age group 15 to 24 to more than 60 per cent in age group 45 to 54, but the largest category for age group 55 to 64 is chronic obstructive airways disease. In the case of women, the largest single category of excess mortality due to respiratory disease is chronic obstructive airways disease at all ages above exact age 25.

Conclusions

Despite the obvious limitations of these data it is possible to determine areas for intervention. The most important limitation is the small numbers on which these data were based. Although the Northern Territory data set included information on a total of 1,651 deaths, by the time this is subdivided by sex, age and cause, some cells are small, thus limiting extrapolations from the data.

Notwithstanding this limitation it is possible to determine that most excess mortality is from causes that are preventable or treatable, at least theoretically. Most ischaemic heart disease, hypertensive disease and cerebro-vascular disease can be minimised by interventions, including preventive measures, that we already know about. They do not require extensive and expensive new technology but they do require resources to encourage and implement remedies and preventions that are already well-known. Similarly deaths from pneumonia and influenza are theoretically preventable and/or curable. Deaths from chronic obstructive airways disease may be more complicated, because we have little knowledge of the long-term effects of repeated childhood chest infections from which so many Aborigines have suffered. We do, however, know something about the dangers of smoking if lung damage has already occurred and it is possible to determine areas for intervention on data that are already available.

9 LEVELS AND TRENDS OF ABORIGINAL MORTALITY IN CENTRAL AUSTRALIA

Noor A Khalidi

This study covers major Aboriginal population centres in the area of Central Australia, from the twentieth parallel in the north to the South Australian border in the South. This is the area served by the District Registrar of Births, Deaths and Marriages in Alice Springs and also approximately the area served by the Northern Territory Department of Health Regional Office in Alice Springs.

There are many reasons for undertaking mortality analysis of Central Australian Aborigines. The first reason is to gain an understanding of the population's mortality regime and its trend which, in conjunction with fertility behaviour, directly affects the size and age distribution of the population and reflects the health status and well-being of the people. Second, a comprehensive mortality analysis of the Aboriginal people living in the vast areas of Central Australia has not been conducted up till now; most of the studies have been concentrated on the State level. The mortality conditions of the total Aboriginal population of the Northern Territory may not necessarily completely reflect the mortality regime which prevails in Central Australia. Third, a longitudinal study of Aboriginal mortality in the region will enable us to assess the trends in mortality indices and thus will provide necessary tools for evaluation of the health intervention programs.

This is the first analysis of Aboriginal mortality data for a long period from the same geographic area using the same data source. Previous studies on Aboriginal mortality, for example Hicks (1985b) in Western Australia, and Thomson and Smith (1985) in New South Wales country regions, mainly focussed on mortality conditions in a particular year or period. A very recent work on Aboriginal mortality, covering the total Northern Territory (Plant 1988) did not study the trends in the causes of death and how they affect the survivorship of the Aboriginal people.

Finally, the study of Aboriginal mortality in Central Australia is a research priority for those organisations responsible for the delivery of the health care services for the Aboriginal people in the region, especially in the case of the health services provided by the Central Australian Aboriginal Congress. A comprehensive mortality study will define the health status of the people in terms of mortality measurements and provide a guide to intervention strategies.

Results

Analysis of Aboriginal death records from 1975 to 1986 in Central Australia suggests that Aboriginal mortality is steadily declining. The decline is exhibited in the gain in expectation of life at birth from 1975-77 to 1984-86 for Aboriginal males which was found to be more than five calendar years, and for Aboriginal females during the same period which was about four years. The study also shows that the gap between

male and female expectation of life at birth, which was recorded as about 14 years during the period 1975-77, is perhaps closing; during 1984-86 the gap was about 13 years. The effects of various causes of death in the improvement in Aboriginal mortality are examined in the following discussion.

Figure 1 Trend of expectation of life at birth for Aborigines in Central Australia by sex, 1975-85.

In spite of these improvements over the last 10 years, the level of Aboriginal mortality in Central Australia is still much higher than that of the total population of the Northern Territory or the total population of Australia. The Aboriginal crude death rates are found to be up to three times higher than the standardised rates for the total population of Australia in 1986, as shown in Tables 1 and 2. The rates shown are deaths per 1,000 population. For 1975-77, Central Australia includes eight major Aboriginal population centres: Alice Springs, Amoonguna, Areyonga, Hermannsburg, Papunya, Santa Teresa, Yuendumu and Docker River. For the periods 1979-83 and 1984-86 the data also cover Ti Tree, Warrabri, Willowra and Utopia. Comparative data for the total Aboriginal population of the Northern Territory are taken from Plant (1988, 69-70) for the 1979-83 period, and for 1984-86 from Gray (1988b). For the total Australian population the data are from the Australian Bureau of Statistics (1987a). Indirectly standardised crude death rates for the total populations of the Northern Territory and Australia are based on the age structure of the Aboriginal population.

Compared to the total Northern Territory population in 1986, the Central Australian Aboriginal mortality rates are higher by more than 2.6 times for males and 2.0 times for females. Similarly, the expectation of life at birth for the Aborigines of Central Australia is up to 17 years less than that of the population of the Northern Territory as a whole and up to 20 years less than that of the total population of Australia.

The relative risk of mortality for Aborigines of Central Australia, as Table 3 and Figure 2 show, is substantially higher at all ages than in the non-Aboriginal population of the Territory. For example, the relative risk of mortality for Aboriginal males aged zero to four is 4.5 times and for Aboriginal females 4.6 times the rates exhibited by the non-Aboriginal population of the Territory. The table also reveals that the relative mortality risks in the middle adult ages of 25 to 49 for Aborigines of the study area are up to 17.5 times higher than those of the non-Aboriginal population of the Territory. In this comparison, the data for non-Aborigines in the Northern Territory are adapted from Devanesen et al (1986, 23).

Table 1 Crude death rates and life expectancy for Aborigines in Central Australia by sex, 1975-86.

		1975-77	1979-83	1984-86
Crude death rates (per 1,000 population)				
	Persons	13.0	11.0	9.0
	Males	16.8	13.2	11.2
	Females	9.6	8.9	6.9
	Difference	7.2	4.3	4.3
Expectations of life at birth (years)				
	Males	47.26	51.21 (51.32)	52.39 (54.7)
	Females	61.32	62.99 (58.16)	65.08 (62.2)

NOTE: Comparative figures for Northern Territory Aborigines are given in brackets where available.

Table 2 Standardised death rates, mortality ratios and life expectancy for Australian and Northern Territory populations by sex, 1986.

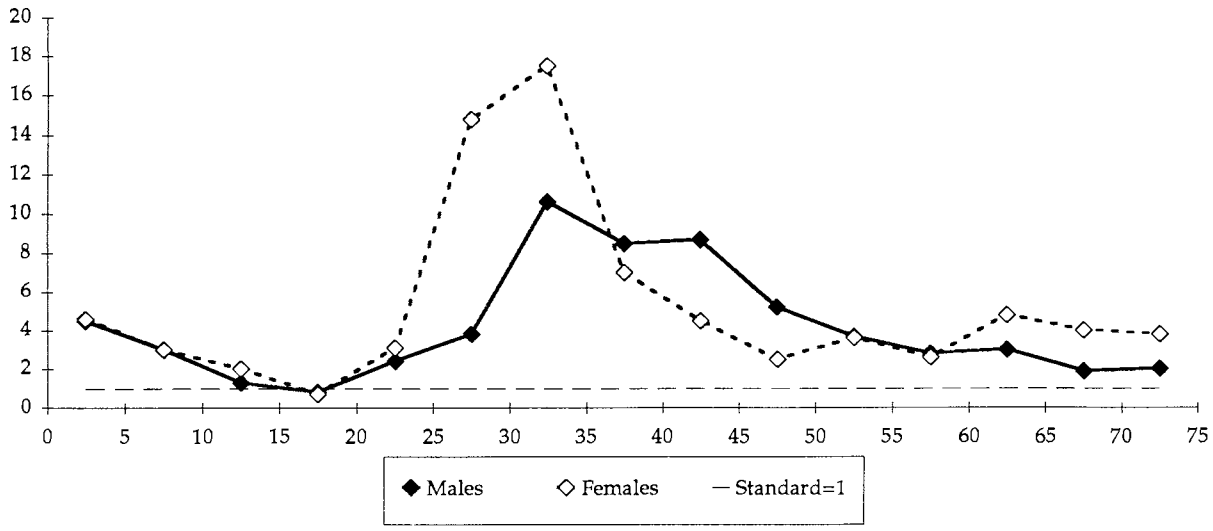
		Australia	Northern Territory
Standardised death rates			
	Males	3.9	4.1
	Females	2.8	3.2
	Difference	1.1	0.9
Standardised mortality ratio			
	Males	287	273
	Females	246	216
Expectations of life at birth (years)			
	Males	72.77	69.29
	Females	79.13	76.37

Table 3 Age-specific mortality rates and relative risk for Aborigines in Central Australia and total Northern Territory non-Aborigines by sex, 1979-83.

Age group	Males			Females		
	Aboriginal	Non-Aboriginal	Relative risk	Aboriginal	Non-Aboriginal	Relative risk
0-4	13.1	2.9	4.5	11.0	2.4	4.6
5-9	0.9	0.3	3.0	1.2	0.4	3.0
10-14	0.9	0.7	1.3	0.6	0.3	2.0
15-19	1.6	2.0	0.8	0.6	0.9	0.7
20-24	5.2	2.2	2.4	2.2	0.7	3.1
25-29	11.1	2.9	3.8	5.9	0.4	14.8
30-34	18.1	1.7	10.6	7.0	0.4	17.5
35-39	23.7	2.8	8.5	5.6	0.8	7.0
40-44	27.0	3.1	8.7	4.9	1.1	4.5
45-49	30.5	5.9	5.2	6.1	2.4	2.5
50-54	32.1	8.6	3.7	9.6	2.7	3.6
55-59	35.1	12.7	2.8	15.9	6.2	2.6
60-64	40.1	13.0	3.0	26.5	5.5	4.8
65-69	50.3	27.0	1.9	44.1	11.1	4.0
70-74	64.6	32.5	2.0	72.7	19.1	3.8
75+	155.6	-	-	163.3	-	-

NOTE: Relative risk is the ratio of the observed rate to the total Northern Territory non-Aboriginal rate.

Figure 2 Relative mortality risk for Aborigines in Central Australia, compared with non-Aboriginal Northern Territory population, 1979-83.



During the entire period 1975-77 to 1984-86, the leading causes of Aboriginal male deaths in Central Australia were found to be, in order, diseases of the circulatory system, diseases of the respiratory system, external causes (accidents, poisoning and violence) and infectious and parasitic diseases. These causes alone contributed more than 70 per cent of the Aboriginal male crude death rate in 1984-1986, as shown in Table 4. For females during the same period the leading causes of mortality were found to be, in order, diseases of the respiratory system, diseases of the circulatory

system, neoplasms, diseases of the genito-urinary system, external causes and infectious and parasitic diseases. These causes contributed 63 per cent of the Aboriginal female death rate during the period 1984-86 (see Table 5). Importantly, the tables also reveal that the rates are steadily declining, except for neoplasms and diseases of the genito-urinary system among females. For example, as Tables 5 and 6 show, the leading cause of death for Aboriginal males, diseases of the circulatory system, dropped from 7.7 per 1,000 population in 1975-77 to 4.6 during 1978-80 and to 3.3 during 1981-83, then slightly increased to 3.7 per 1,000 population during the period 1984-86. For Aboriginal females, the leading cause of death, diseases of the respiratory system, declined from 3.5 per 1,000 population in the period 1975-77 to 2.6 during 1978-80 and to 0.8 during 1981-83, then slightly increased to 1.0 per 1,000 population during 1984-86.

Figure 3 Relative mortality risk for Aborigines in Central Australia, compared with total Northern Territory population by sex, 1984-86.

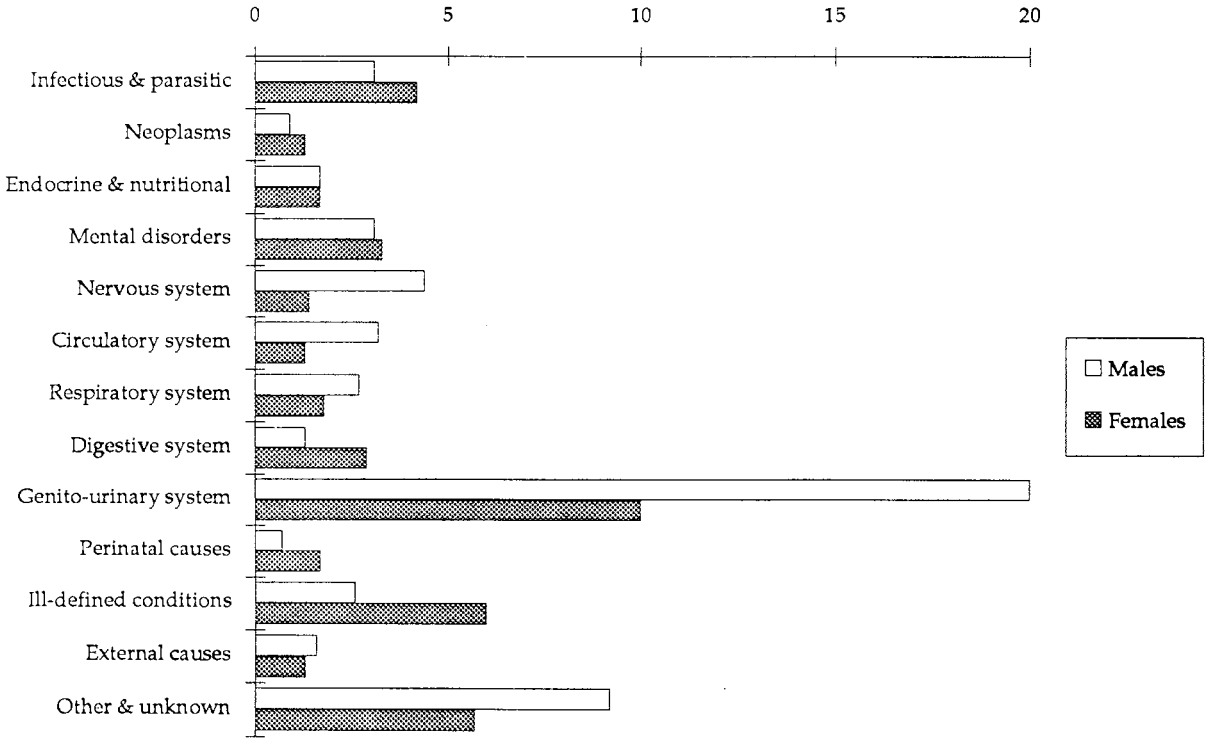


Table 4 Aboriginal male standardised[1] cause-specific death rates in Central Australia, 1975-77 to 1984-86.

Cause of death (ICD-9 code)[2]	1975-77	1978-80	1981-83	1984-86
Infectious and parasitic (I 001-139)	1.8	0.9	0.9	0.5
Neoplasms (II 140-239)	0.4	0.7	0.6	0.4
Endocrine, nutritional and metabolic (III 240-279)	0.9	0.6	0.5	0.2
Mental disorders (V 290-319)	1.0	0.2	0.5	0.4
Nervous system and sense organs (VI 320-389)	0.5	0.9	0.2	0.4
Circulatory system (VII 390-459)	7.7	4.6	3.3	3.7
Respiratory system (VIII 460-519)	4.2	3.4	1.4	1.6
Digestive system (IX 520-579)	0.3	1.3	0.2	0.2
Genito-urinary system (X 580-629)	1.3	0.3	0.2	0.6
Perinatal causes (XV 760-779)	0.9	0.4	0.5	0.1
Symptoms and ill-defined conditions (XVI 780-799)	3.3	1.8	1.0	0.5
Accidents, poisoning and violence (XVII E800-E999)	3.7	4.3	1.6	1.5
Other classes and causes not stated	0.2	2.5	0.6	1.0
All causes	26.2	21.9	11.5	11.2

NOTES: 1. Age distribution of Central Australian Aboriginal males in the period 1984-86 as standard.
2. ICD-9 (1975) by the World Health Organization (1977).

Table 6 shows that, compared to the total Northern Territory population, 39 per cent of the excess mortality risk for Aboriginal males in 1984-86 is due to diseases of the circulatory system. For females external causes constitute three per cent of the excess risk, compared with eight per cent of the excess risk for males, and diseases of the genitourinary system make up about 15 per cent of the excess risk. The relative risks are also shown in Figure 3.

Decline in Aboriginal mortality in Central Australia from 1975-77 to 1984-86 resulted in gains in complete expectations of life for both Aboriginal males and females, though the improvement in the male life expectancy was slightly greater. Tables 7 and 8 show the contribution of the various causes of death and age groups to the improvements in Aboriginal male and female expectation of life at birth in Central Australia from 1975-77 to 1984-86. A quick look at the tables reveals that while over the study period, mortality due to some of the diseases was reduced, these reductions were to some extent balanced by the increase in mortality from some other diseases. The combined effects of these two processes resulted in gains in expectations of life of 6.77 years for males and 5.76 years for females.

For men the following observations may be noted: the contribution of diseases of the circulatory system is dominant in the improvement in expectation of life at birth (3.5 years or 51 per cent of the improvement); a contribution of one year or 15 per cent of the improvement is due to reduction in mortality from symptoms, signs and ill-defined conditions; a contribution of one year or 14 per cent is due to the reduction in mortality from diseases of the respiratory system; reduction in mortality from endocrine, nutritional and metabolic diseases and immunity disorders

Table 5 Aboriginal female standardised cause-specific death rates in Central Australia, 1975-77 to 1984-86.

Cause of death (ICD-9 code)	1975-77	1978-80	1981-83	1984-86
Infectious and parasitic (I 001-139)	1.3	0.4	0.8	0.5
Neoplasms (II 140-239)	0.9	0.7	0.9	0.9
Endocrine, nutritional and metabolic (III 240-279)	0.4	1.9	0.4	0.3
Mental disorders (V 290-319)	0.0	0.6	0.4	0.2
Nervous system and sense organs (VI 320-389)	0.6	0.4	0.5	0.1
Circulatory system (VII 390-459)	2.3	2.3	1.9	0.9
Respiratory system (VIII 460-519)	3.5	2.6	0.8	1.0
Digestive system (IX 520-579)	0.7	0.0	0.4	0.2
Genito-urinary system (X 580-629)	0.5	0.8	0.2	0.6
Perinatal causes (XV 760-779)	0.6	0.2	0.3	0.2
Symptoms and ill-defined conditions (XVI 780-799)	1.5	0.9	0.8	0.6
Accidents, poisonings and violence (XVII E800-E999)	1.7	1.6	0.8	0.5
Other classes and causes not stated	1.2	1.8	2.2	0.8
All causes	15.2	14.2	10.4	7.0

NOTE: See notes to Table 4.

Table 6 Aboriginal cause-specific death rates, relative risk,[1] and proportion of excess risk[2] in Central Australia, 1984-86 compared with the total Northern Territory population, 1986.

Cause of death (ICD-9 code)	Aboriginal death rate (per 1,00)	Total NT rate	Relative risk	Excess risk (%)
Males				
Infectious and parasitic (I)	0.5	0.16	3.1	4.8
Neoplasms (II)	0.4	0.47	0.9	-1.0
Endocrine, nutritional and metabolic (III)	0.2	0.12	1.7	1.0
Mental disorders (V)	0.4	0.13	3.1	3.8
Nervous system and sense organs (VI)	0.4	0.09	4.4	4.3
Circulatory system (VII)	3.7	0.96	3.2	38.8
Respiratory system (VIII)	1.6	0.60	2.7	14.2
Digestive system (IX)	0.2	0.15	1.3	0.7
Genito-urinary system (X)	0.6	0.03	20.0	8.1
Perinatal causes (XV)	0.1	0.15	0.7	-0.7
Ill-defined conditions (XVI)	0.5	0.19	2.6	4.4
External causes (XVII)	1.5	0.96	1.6	7.6
Other causes and unknown	1.1	0.12	9.2	13.9
All causes	11.2	4.11	2.7	100.0
Females				
Infectious and parasitic (I)	0.5	0.12	4.2	10.5
Neoplasms (II)	0.9	0.67	1.3	6.4
Endocrine, nutritional and metabolic (III)	0.3	0.18	1.7	3.3
Mental disorders (V)	0.2	0.06	3.3	3.9
Nervous and sense organs (VI)	0.1	0.07	1.4	0.8
Circulatory system (VII)	0.9	0.66	1.3	6.6
Respiratory system (VIII)	1.0	0.55	1.8	12.5
Digestive system (IX)	0.2	0.07	2.9	3.6
Genito-urinary system (X)	0.6	0.06	10.0	15.0
Perinatal causes (XV)	0.2	0.12	1.7	2.2
Symptoms and ill-defined conditions (XVI)	0.6	0.10	6.0	13.9
External causes (XVII)	0.5	0.39	1.3	3.0
Other causes and unknown	0.8	0.14	5.7	18.3
All causes	6.8	3.19	2.1	100.0

NOTES:

1. Relative risk is the ratio of the observed Aboriginal rate to the standardised total Northern Territory rate. For the total Northern Territory, the cause-specific rates are indirectly standardised based on the Central Australian Aboriginal age structure, 1984-86.
2. Excess risk is derived by subtracting the indirectly standardised cause-specific death rates for the total Northern Territory (1986) from Aboriginal rates (1984-86).

contributed one year or 12 per cent; a contribution of about one-quarter of a year or four per cent is due to reduction in mortality from certain conditions originating in the perinatal period. Diseases of the nervous system and sense organs and diseases of the digestive system also each contributed about three months or four per cent in the expectation of life improvement.

Increase in the occurrence of certain diseases made a negative contribution: increased incidence of neoplasms reduced male expectation of life by 0.6 years or eight per cent, and complications of childbirth and the puerperium, diseases of the genito-urinary system, diseases of the skin and subcutaneous tissue and congenital anomalies also made negative contributions.

It is worth mentioning the large contribution of mortality improvement in age group 15 to 29 from all causes (except from diseases of the respiratory system which made a negative contribution) in the increase in expectation of life of four years. Smaller positive contributions were also observed in all other age groups except age zero and age group 30 to 49, which each had negative contributions of 0.2 years. At age zero, this was due to increase in infectious and parasitic diseases, neoplasms, respiratory system, digestive system and childbirth complications; in ages 30 to 49 the increase in mortality was mainly due to increase from external causes and violence.

For Aboriginal women in particular, the following may be noted: reduction in mortality from diseases of the respiratory system contributed about three years or 52 per cent in the expectation of life improvement, and a contribution of two years or 31 per cent was due to reduction of mortality from diseases of the circulatory system; a contribution of about one and a half years or 26 per cent of the improvement was due to reduction of mortality from symptoms, signs and ill-defined conditions. There is nearly a year improvement in expectation of life due to reduction in mortality from accidents, violence and other external causes. This reduction occurred mostly in the 15

Table 7 Contribution of the various causes of death to the improvement in expectation of life between 1975-77 and 1984-86, for male Central Australian Aborigines.

Cause of death (ICD-9 code)	Age in years 0	1-4	5-14	15-29	30-49	50-69	70+	Total
Infectious and parasitic (I)	-0.25	0.23	0.25	010	-0.08	-0.10	0.07	0.21
Neoplasms (II)	-0.16	-	-	0.18	-0.04	-0.52	-0.02	-0.56
Endocrine, nutritional and metabolic (III)	-	0.23	-	0.25	0.40	-0.10	0.02	0.80
Blood and blood organs (IV)	-	-	-	-	-	0.11	-	0.11
Mental disorders (V) -	-	-	0.10	-0.08	-	-0.02	-	
Nervous system and sense organs (VI)	0.20	-	-	0.25	-0.32	0.11	-	0.24
Circulatory system (VII)	-	-	0.16	1.73	0.02	1.11	0.42	3.45
Respiratory system (VIII)	-0.10	0.23	-	-0.23	0.19	0.65	0.22	0.96
Digestive system (IX)	-0.16	-	-	0.51	0.20	-0.32	-	0.24
Genito-urinary system (X)	-	-	-	0.18	0.12	-0.42	-0.02	-0.14
Pregnancy and childbirth (XI)	-0.16	-	-	-	-	-	-	-0.16
Skin and subcutaneous tissue (XII)	-	-	-	-	-	-	-0.02	-0.02
Musculoskeletal system (XIII)	-	-	-	-	0.20	-	-	0.20
Congenital anomalies (XIV)	-	-	-	-	-	-	-0.02	-0.02
Perinatal causes (XV)	0.35	-	-0.09	-	-	-	-	0.26
Symptoms and ill-defined conditions (XVI)	-0.01	-	-	0.10	-0.08	0.54	0.45	1.00
External causes (XVII)	0.07	0.23	-0.28	1.17	-0.69	-0.41	0.13	0.22
All causes	-0.21	0.90	0.04	4.36	-0.17	0.64	1.21	6.77

Table 8 Contribution of the various causes of death to the improvement in expectation of life between 1975-77 and 1984-86, for female Central Australian Aborigines.

Cause of death (ICD-9 code)	Age in years 0	1-4	5-14	15-29	30-49	50-69	70+	Total
Infectious and parasitic (I)	0.41	0.10	-	-0.09	-0.34	0.10	-0.07	0.11
Neoplasms (II)	-	-	-	-	0.01	-0.83	0.53	-0.29
Endocrine, nutritional and metabolic (III)	-0.12	0.26	-	-	-0.17	0.10	0.05	0.13
Blood and blood organs (IV)	-	-	-	-	-	-	-	-
Mental disorders (V) -	-	-	-	-	-0.34	-	-0.06	-0.40
Nervous system and sense organs (VI)	0.31	-	-	-	-0.17	-0.56	-0.06	-0.48
Circulatory system (VII)	-0.01	-	0.57	0.21	-0.28	1.52	-0.26	1.75
Respiratory system (VIII)	-0.34	0.26	-0.14	0.39	0.59	1.42	0.80	2.99
Digestive system (IX)	-	0.13	-	-	-0.15	0.28	0.11	0.67
Genito-urinary system (X)	-	-	-	-	-0.34	-0.37	-0.12	-0.83
Pregnancy and childbirth (XI)	-	-	-	-	-	-	-	-
Skin and subcutaneous tissue (XII)	-	-	-	-	0.18	-0.37	-	-0.19
Musculoskeletal system (XIII)	0.10	-	-	-	-	-	-	0.10
Congenital anomalies (XIV)	-0.33	-	-	-	-	-	-	-0.33
Perinatal causes (XV)	0.09	-	-	-	-	-	-	0.09
Symptoms and ill-defined conditions (XVI)	-0.01	-	-	-0.18	-0.01	-0.09	1.75	1.49
External causes (XVII)	0.10	-0.32	-	0.80	-0.11	0.28	0.18	0.93
All causes	0.23	0.44	0.43	1.41	-1.08	1.48	2.85	5.76

to 29 age group. For women, increase in diseases of the genito-urinary system had a major negative effect of 0.8 years or 14 per cent, mainly at age 30 and over. Mental disorders, diseases of the nervous system and congenital anomalies also made a noticeable negative contribution.

Mortality in all age groups, except ages 30 to 49 which had a negative contribution of slightly more than a year, declined for women during the study period. The contribution of the reduction in mortality at older ages of 50 and over was more than four years.

In addition to the use of the complete expectation of life for comparative study of mortality or longevity over time or across populations, it is useful to compare the length of time Aborigines of Central Australia survive in various important phases of life to those of the total Northern Territory and total population of Australia. This is done by calculating the 'temporary' expectations of life for such ages as early childhood (zero to four years), school age (five to 14 years), early adulthood (15 to 24 years), middle adulthood (25 to 44 years), late adulthood (45 to 59 years) and old age (60 to 79 years).

Table 9 Difference in temporary expectations of life (nEx) between Aborigines of Central Australia, 1984-86 and the total Northern Territory population, 1986, by sex and age.

Age in years	Females nEx		Difference	Males nEx		Difference
	NT	Central Australian Aborigines		NT	Central Australian Aborigines	
0-4	4.90	4.84	0.06	4.90	4.87	0.08
5-14	10.00	9.99	0.01	9.98	9.94	0.04
15-24	9.97	9.97	0.00	9.91	9.91	0.00
25-44	19.72	19.17	0.55	19.23	17.73	1.50
45-59	14.47	13.66	0.81	14.08	11.36	2.72
60-79	17.27	14.32	2.95	15.64	12.13	3.51

In Table 9, the temporary expectations of life of Aboriginal males and females in Central Australia in these six phases of life are compared to those of the total Northern Territory population. The following observations could be drawn: the pattern of differences between the Aboriginal temporary expectations of life and those of the total Northern Territory populations is the same, the differences for the three lowest age groups are marginal and differences gradually increase over the subsequent phases of life. At ages 60 to 79 Aboriginal men live up to 3.5 years less than their counterparts making up the total Northern Territory or total Australian population. Aboriginal women at ages 60 to 79 live up to three years less than the total Northern Territory female population. It may be noted that the pattern of difference here is not the same as for relative mortality risks—the reason is that death rates are highest at the higher ages in all mortality schedules.

10 ABORIGINAL MORTALITY IN SOUTH AUSTRALIA

Ceilia Divakaran-Brown and Morteza Honari

Analysis of mortality levels and characteristics of mortality in the Aboriginal population of South Australia must be approached with some caution because of the inherent diversity of Aboriginal groups and their corresponding lifestyles found in this State. Data collection and registration systems are still far from optimal and thus do not provide information which would adequately reflect these differences. Issues of procedures for appropriate identification of Aborigines, and extent of coverage of official sources of registration still require refinement for the currently perceived under-enumeration to be corrected.

Determination of the extent and accuracy of Aboriginal death records for 1985 was carried out as part of a larger study undertaken by the AHO of South Australia in 1986. Statistics of deaths in the communities were obtained from the following sources: Information Statements of Death Registration; medical certificates of Cause of Death; Coroners reports; Aboriginal Health Worker records; and community information. Information included the underlying cause of death, age at death and sex. The number of cases recorded by the Registrar of Births, Deaths and Marriages are set out below. The figures for 1983 and 1984 are clearly under-enumerated. The 1985 figure of 122 deaths for an estimated Aboriginal population of South Australia in 1985 of 14,012 forms the basis of analysis in this chapter.

Table 1 Cases of Aboriginal deaths in South Australia by sex from the Registrar of Births, Deaths and Marriages, 1983-85.

	Male	Female	Total
1983	36	26	62
1984	55	31	86
1985	76	46	122
Total	167	103	270

Through a process of validation by community members and Aboriginal health personnel, a register of known Aboriginal deceased persons for 1985 was compiled. In the course of this work, the AHO collected statistics on mortality of Aboriginal people in 1983, 1984 and 1985. The total Aboriginal population of South Australia in 1985 was estimated to be 14,012 comprising 6.3 per cent of the estimated total 223,183 Aboriginal population in Australia and 1.0 per cent of the total population of South Australia.

Results

An analysis of the total number of deaths collected within these three years gives a general picture of mortality as an indicator of health status of the South Australian Aboriginal population. From the total of 270 cases collected, the most frequent cause of death is ischaemic heart disease (60 cases or 22.2 per cent), other forms of heart disease (18 cases or 6.7 per cent) followed by pneumonia and influenza (16 cases or 5.9 per cent) and the same number for accidental deaths. Mortality related to alcohol is high (16 cases or 5.9 per cent) of which five cases have been identified as alcohol dependent syndrome, 10 cases as renal failure, and one case as poisoning by alcohol. There were 15 deaths caused by chronic obstructive pulmonary disease and allied conditions. Diabetes mellitus was identified as the cause of 10 deaths. The number of infants who died between 1983 and 1985 was 13, and this was the highest number of deaths for a single age. Two-thirds of the deaths occurred after the age of 40.

The expectation of life as an index of health is a measure reflecting both infant and adult mortality. Table 2 shows the life table of the South Australian Aboriginal population in 1985. Life expectancy at birth for Aboriginal males is 55.5 years and for females is 65.0 years. This is 17.4 years less than the figure for total Australian males of 72.9 years and 13.6 years less than the figure for total Australian females of 78.6 years. Generally, females expect to live an average of five years longer than males. The sex difference in the South Australian Aboriginal population is 9.5 years and for the total Australian population is 5.7 years.

The life expectancy at selected ages denotes the number of years remaining of a person's life at these ages if the prevailing pattern of mortality does not change. The main finding from an analysis of the differences in the life expectancy at selected ages is that Aboriginal people, as they grow older, expect to live to ages closer to those attained by the total Australian population.

As shown in Figure 1, for all Australian males the expectation of life at birth (72.9 years) increases gradually to 82.0 at 70 years. In Aboriginal males, on the other hand, life expectancy at birth which is 55.5 years increases sharply to 78.5 at age 70 years (3.5 years less than total Australian males). The improvement in expected years of total life from the time of birth to the age of 70 for Aboriginal males is 23.0 years and for Australian males in general only 9.1 years.

For females also the picture is similar, as shown in Figure 2. The expectation of life at birth for Aborigines is 65.0 and for all Australian females 78.6. At age 70 this increases to 80.8 years for Aborigines and to 84.3 years for all Australians, the gap narrowing from 13.6 to 3.5 years. The improvement in expected years of total life from the time of birth to the age of 70 for female Aborigines is 19.4 and for total Australian females 5.7.

Table 2 Life table of the South Australian Aboriginal population by sex, 1985.

Age	Px	Dx	Mx	qx	lx	dx	Lx	Tx	ex
Males									
0	186	2	0.01198	0.01075	100000	1075	99247	5548181	55.5
1-4	789	3	0.00380	0.01498	98925	1 482	392587	5448934	55.1
5-9	845	1	0.00118	0.00588	97443	573	485781	5056347	51.9
10-14	871	1	0.00115	0.00571	96870	553	482965	4570567	47.2
15-19	850	4	0.00471	0.02299	96317	2214	476048	4087601	42.4
20-24	797	2	0.00251	0.01239	94102	1166	467597	3611554	38.4
25-29	616	6	0.00974	0.04644	92936	4316	453892	3143957	33.8
30-34	486	3	0.00617	0.02994	88620	2653	436469	2690065	30.4
35-39	359	8	0.02228	0.10025	85967	8618	408290	2253596	26.2
40-44	272	0	0.00000	0.00000	77349	0000	386744	1845306	23.9
45-49	201	8	0.03980	0.16598	77349	12838	354649	1458562	18.9
50-54	185	7	0.03784	0.15909	64511	10263	296897	103912	17.1
55-59	117	3	0.02564	0.11364	54248	6 16525	5828	807016	14.9
60-64	85	6	0.07059	0.26087	48083	12543	209058	551188	11.5
65-69	64	7	0.10938	0.35354	35540	12565	146288	342130	9.6
70-74	40	5	0.12500	0.38462	22975	88379	2785	195843	8.5
75-79	29	3	0.10345	0.34091	14139	48205	8643	103058	7.3
80-84	15	5	0.33333	0.62500	9319	58243	2033	44415	4.8
85+	13	2	0.15385	1.00000	3494	3494	12382	12382	3.5
	6820	46							
Females									
0	202	1	0.00562	0.00495	100000	495	99653	6497492	65.0
1-4	818	1	0.00122	0.00487	99505	484	397003	6397839	64.3
5-9	829	1	0.00121	0.00600	99021	594	493620	6000836	60.6
10-14	893	1	0.00112	0.00557	98427	548	490765	5507216	56.0
15-19	881	2	0.00227	0.01122	97879	1099	486649	5016451	51.3
20-24	796	3	0.00377	0.01850	96781	1790	479428	4529802	46.8
25-29	688	3	0.00436	0.02134	94991	2027	469885	4050375	42.6
30-34	497	0	0.00000	0.00000	92964	0	464818	3580489	38.5
35-39	400	3	0.00750	0.03614	92964	3360	456418	3115671	33.5
40-44	278	2	0.00719	0.03472	89604	3111	440240	2659253	29.7
45-49	244	5	0.02049	0.09294	86492	8038	412366	2219013	25.7
50-54	191	3	0.01571	0.07282	78454	5713	377988	1806647	23.0
55-59	134	3	0.02239	0.10067	72741	7323	345399	1428659	19.6
60-64	109	3	0.02752	0.12097	65418	7914	307308	1083260	16.6
65-69	97	4	0.04124	0.17094	57505	9830	262950	775952	13.5
70-74	58	6	0.10345	0.34091	47675	1625	3197743	513002	10.8
75-79	36	2	0.05556	0.21739	31422	6831	140033	315259	10.0
80-84	22	2	0.09091	0.31250	24591	7685	103744	175226	7.1
85+	18	1	0.05556	1.00000	16906	16906	71481	71481	4.2
	7191	46							

Figure 1 South Australian Aboriginal life expectancies at selected ages compared with the total Australian population, 1985

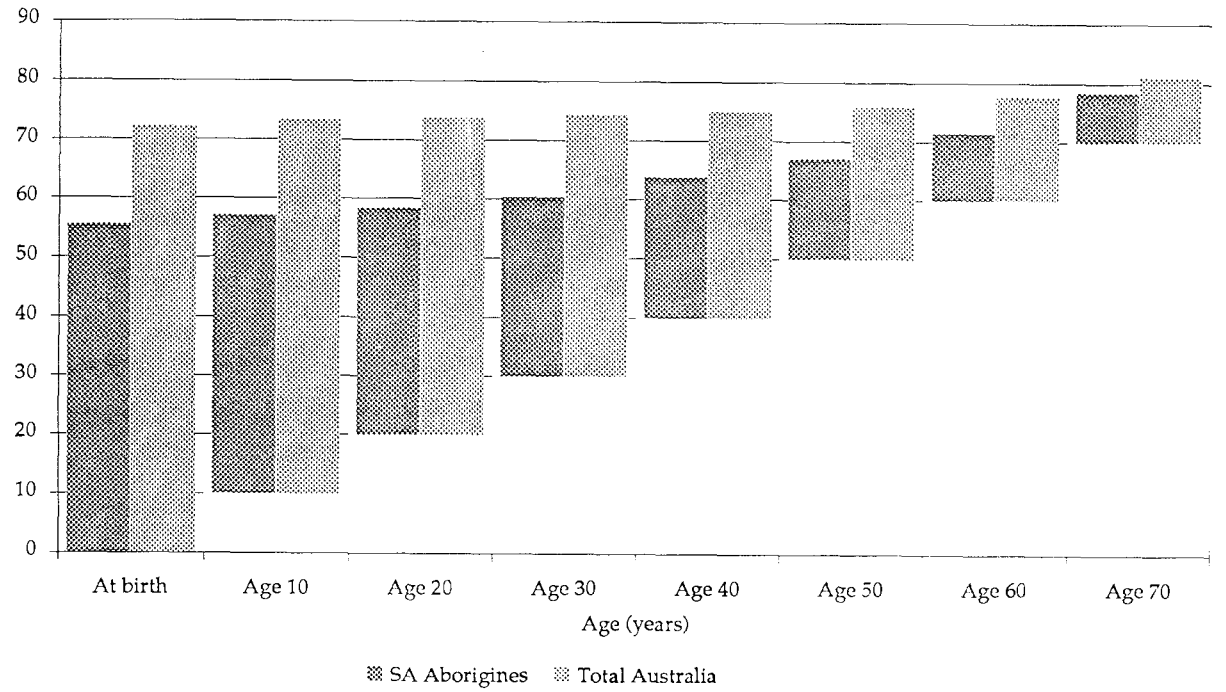
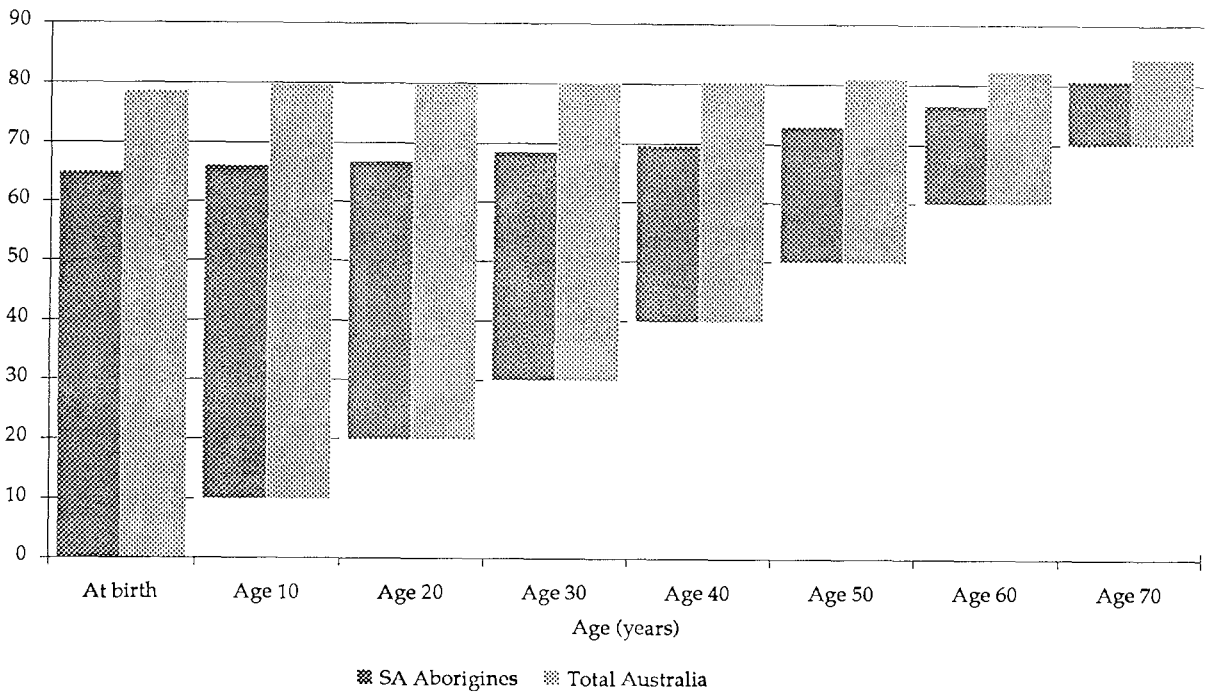


Figure 2 South Australian Aboriginal life expectancies for females at selected ages, compared with the total Australian population, 1985.



Aboriginal mortality is characterised by early to middle adulthood mortality. Sixteen of 46 female deaths occurred in age group 35 to 59 and 26 of 76 male deaths occurred in this same age bracket. Causes of Aboriginal deaths are expressed in observed and expected rates per 1,000 population of the same sex. Expected numbers are the number of deaths that would have occurred in the South Australian Aboriginal population if they had experienced the same death rates as the total Australian population in 1985 within each age group. The causes are grouped according to the class headings of the ICD-9.

The leading cause of death for both sexes was diseases of the circulatory system (ICD-9: 390-459). The expected number for this category was 8.3 for males and 6.7 for females, but the observed numbers are 29 (3.5 times) and 19 (2.8 times) the expected numbers respectively. This class includes causes such as hypertension, rheumatic fever and rheumatic heart disease, disease of pulmonary circulation and cerebro-vascular disease and ischaemic heart disease.

External causes of injury and poisoning (ICD-9: E800-E999) rank as the second most frequent cause of death. The expected number of deaths caused by injury and poisoning is 4.2 for males and 1.5 for females; the observed number (14 deaths) for males is 3.3 times the expected number, and for females (eight deaths) it is 5.2 times the expected number. Deaths caused by motor vehicle accidents, fire, drowning, poisoning and violence are included under this heading.

Table 3 Observed and expected numbers, standardised mortality ratios and proportion of excess risk for causes of death in South Australian Aborigines by sex, 1985.

Cause of death (ICD-9 code)	Observed N	Expected N	SMR	% Excess risk
Males				
Circulatory system (VII 390-459)	29	8.33	3.5	38.5
External causes(XVII E800-E999)	14	4.21	3.3	15.4
Respiratory system (VIII 460-519)	10	1.71	5.9	15.4
Neoplasms (II 140-239)	7	4.78	1.5	7.7
Mental disorders (V 290-319)	4	0.40	0.1	7.7
Infectious and parasitic (I 001-139)	3	0.14	21.4	7.7
Genito-urinary system (X 580-629)	3	0.25	11.9	7.7
Digestive system (IX 520-579)	2	0.69	2.9	-
Endocrine, nutritional and metabolic (III 240-279)	1	0.40	2.5	-
Nervous system and sense organs (VI 320-389)	1	0.40	2.5	-
Congenital anomalies (XIV 740-759)	1	0.77	1.3	-
Symptoms and ill-defined conditions (XVI 780-799)	1	0.61	1.6	-
All causes	76	23.68	3.2	100.0
Females				
Circulatory system (VII 390-459)	19	6.70	2.8	42.9
External causes (XVII E800-E999)	8	1.54	5.2	28.6
Digestive system (IX 520-579)	5	0.52	9.7	14.3
Respiratory system (VIII 460-519)	4	0.98	4.1	14.3
Genito-urinary system (X 580-629)	3	0.25	11.8	14.3
Neoplasms (II 140-239)	2	3.95	0.5	-
Infectious and parasitic (I 001-139)	1	0.13	7.5	-
Endocrine, nutritional and metabolic (III 240-279)	1	0.39	2.5	-
Nervous system and sense organs (VI 320-389)	1	0.34	3.0	-
Skin and subcutaneous tissue (XII 680-709)	1	0.01	-	-
Symptoms and ill-defined conditions (XVI 780-799)	1	0.41	2.4	-
All causes	46	17.1	2.7	100.0

Diseases of the respiratory system (ICD-9: 460-519) are the fourth most frequent cause of death for Aboriginal males and the third for Aboriginal females. For males the observed number of 10 deaths is 5.9 times the expected number and for females the observed number of deaths is 4.0 times the expected number. Pneumonia and asthma are the major causes in this category.

Figures 3 and 4 illustrate the rank order of expected and observed numbers which again highlights the wide gaps between the Aboriginal pattern of mortality in comparison to the total Australian population. A general picture of observed and

Figure 3 South Australian Aboriginal death rates for males by cause, observed rates per 1,000 population and expected rates, 1985.

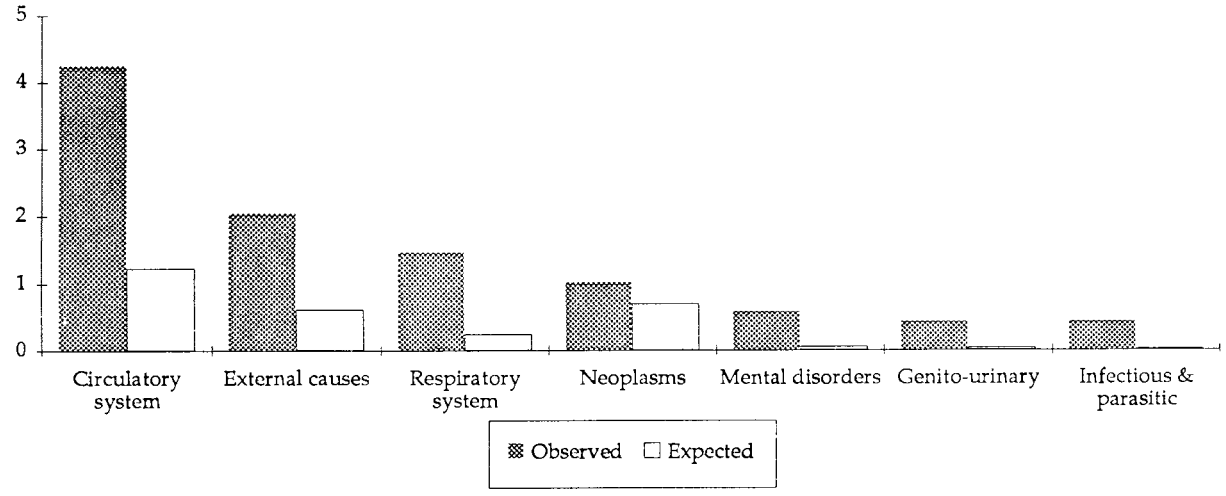
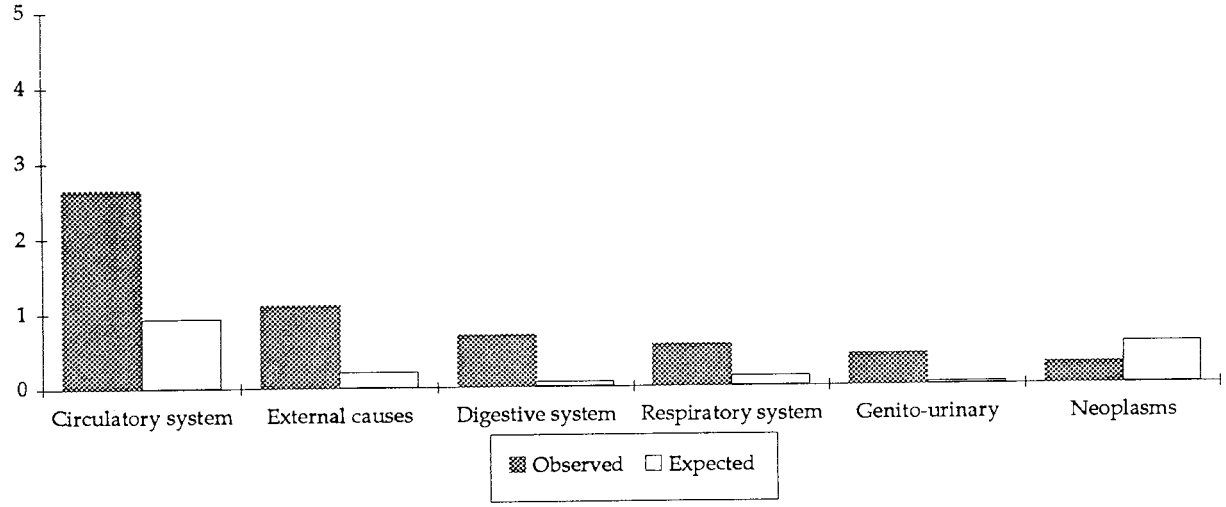


Figure 4 South Australian Aboriginal death rates for females by cause, observed rates per 1,000 population and expected rates, 1985.



expected numbers is shown in Table 3. Table 4 shows age-specific mortality rates and rate ratios. For all age groups age-specific mortality is greater in the Aboriginal population than in the total Australian population; the gap closes slightly around age 20 for males and at ages 30 and 70 for females. Ratios of South Australian Aboriginal rates over those of the total Australian population peak at age 50 for males where the loss of Aboriginal life is greatest. In the female population the ratios have two peaks at ages 15 and 40.

Table 5 shows the proportion of persons who died in South Australian hospitals compared to the total number admitted to hospitals for different causes. The Aboriginal population has been compared to the non-Aboriginal population of the

Table 4 Age-specific mortality rates and rate ratios for South Australian Aborigines and total Australian population by sex, 1985.

Age	N		Rates		Rate
	SA Aboriginal	Australia	SA Aboriginal	Australia	ratio
Males					
0-4	5	1,675	5.1	2.7	1.9
5-9	1	178	1.2	0.3	4.0
10-14	1	198	1.1	0.3	4.0
15-24	6	1,833	3.6	1.4	2.7
25-34	9	1,703	8.2	1.3	6.2
35-44	8	2,001	12.7	1.8	7.1
45-54	15	4,090	38.9	5.1	7.6
55-64	9	10,937	44.6	5.0	3.0
65+	2	41,541	136.6	61.0	2.2
Total	76	64,156	11.1	8.1	1.4
Females					
0-4	2	1,264	2.0	2.2	0.9
5-9	1	114	1.2	0.2	6.1
10-14	1	105	1.1	0.2	7.0
15-24	5	620	3.0	0.5	6.3
25-34	3	710	2.5	0.6	4.6
35-44	5	1,102	7.4	1.0	7.2
45-54	8	2,300	18.4	3.0	6.0
55-64	6	5,620	24.7	7.6	3.2
65+	15	42,817	64.9	45.6	1.4
Total	46	54,652	6.4	6.9	0.9

Table 5 Case fatality in South Australian hospitals by cause of death and sex for Aborigines and non-Aborigines, 1985.

Cause of death (ICD-9 code)	Males		Females	
	Aboriginal	Non-Aboriginal	Aboriginal	Non-Aboriginal
Infectious and parasitic (I 001-139)	0.9	1.9	0.0	1.3
Neoplasms (II 140-239)	7.4	10.2	12.3	7.4
Endocrine, nutritional and metabolic (III 240-279)	1.8	1.8	0.0	1.5
Circulatory system (V 390-459)	8.7	6.9	6.5	8.5
Respiratory system system (VIII 460-519)	0.6	2.0	1.5	1.4
Digestive system (IX 520-579)	1.3	1.2	1.0	1.0
Genito-urinary system (X 580-629)	0.0	1.2	0.4	0.4
Perinatal conditions (XV 760-779)	5.0	1.0	6.9	0.9
Symptoms and ill-defined conditions (XVI 780-799)	1.9	0.9	0.4	0.8
External causes (XV E800-E999)	1.3	0.8	0.3	1.0
All causes	1.3	2.2	0.8	1.4

State. Generally, the non-Aboriginal case fatality rate is almost twice that of the Aboriginal population. The reason for these differences may be less utilisation of services by Aboriginal people and lack of awareness of services, a reticence to use mainstream services because of previous negative experiences as well as the place of illness in everyday life. For males 1.3 per cent of Aborigines admitted to hospitals died there compared to 2.2 per cent of non-Aboriginal patients. For females also the Aboriginal proportion is 0.8 per cent compared with the non-Aboriginal proportion of 1.4 per cent.

Conclusions

In a recently published work on Aboriginal mortality (Gray 1987b), some important clues to the possible changes in the shape of Aboriginal mortality are reviewed. Two key concepts highlighted are the phrase 'what opportunity people have to avoid behaviour which carries health risks' and the notion that 'a lack of social roles and hence the power to change the social environment causes health problems and early death'. In many ways these aspects are implicit within the homelands movement. It is obviously too early to confirm the causal association between geographical location and adult mortality but there is strong suggestion that the homelands movement will be the mercury of this hypothesis. Significant improvements in morbidity have already become evident in these groups.

In examining the underlying causes of death in Aboriginal people it is overwhelmingly evident that this observed early mortality can be avoided through relatively simple measures: appropriate diet and good nutrition to reduce circulatory diseases. The spiritual significance of Aboriginal people settling down in their own country will no doubt impact on Aboriginal quality of life and thus greater longevity. It will thus be of significance to start analysing Aboriginal mortality not just by the conventional categories of infant, adult, male and female but by urban, community and homeland grouping which could potentially reveal the opportunity that Aboriginal people are availing to themselves through cultural revival activities intrinsic in the homelands movement, avoiding behaviour that carries health risks and instead adopting behaviour that prolongs life.

11 REVIEW OF DATA QUALITY FOR ABORIGINAL DEATH RECORDS IN SOUTH AUSTRALIA[1]

Martin Yard, Brian Moody, Ceilia Divakaran-Brown and Eileen Butler

As a result of representations by the Task Force on Aboriginal Health Statistics most State and Territory governments have now included statistical questions concerning Aboriginality on Birth and Death Information Statements. There have been several reviews of the data quality of Aboriginal details obtained from Birth and Death registration documents and the general consensus has been that the data are inadequate. As a result, the ABS and the AIH decided to conduct a pilot study to investigate data quality in one of the States which sought details of Aboriginality on births and deaths registration forms.

Because the South Australian Office of the ABS had undertaken a preliminary study of Aboriginal births and deaths data in 1987, it offered and was subsequently selected to carry out the proposed review. The AHO which has a major role in the preparation and analysis of Aboriginal health statistics accepted an invitation to participate in the review. The major objectives of the review were:

1. to assess the data quality of the information on Aboriginal births and deaths obtainable from the ABS Demographic Statistics System (DEMOSS);
2. to make recommendations to overcome or improve the deficiencies in registration of Aboriginal births and deaths recorded in South Australia;
3. to prepare a quality control methodology which could be adopted by other States and Territories; and
4. to prepare a report, possibly an ABS Occasional Paper, detailing the findings and methodology used in the review.

Although not one of the prime objectives, some analysis of the augmented data was considered to be a possibility, but this was to depend on the successful identification of a sufficient number of vital events to make the study feasible.

This chapter provides a brief summary of the review and its preliminary findings regarding death records. A final report covering births and deaths and addressing the aforementioned objectives has now been issued separately by the ABS and the AHO (Australian Bureau of Statistics 1990).

Methods

The methodology involved several stages. These included analysis of census data, investigating alternative sources of births and deaths information, and

with the assistance of the AHO, a comprehensive scrutiny of all births and deaths registration records for Aboriginality indicators. Details of births to Aboriginal mothers and Aboriginal infant deaths were sought from the Pregnancy Outcome Unit of the South Australian Health Commission (SAHC). The SAHC was also asked to supply data on Aboriginal deaths in hospitals.

The AHO obtained some details of Aboriginal deaths from lists provided by health workers in several locations, particularly communities outside the Adelaide Statistical Division. This information was used as an independent source to check how the events were recorded through the registration system. With the agreement of the Registrar's Office, staff members from the AHO conducted a careful scrutiny of all Births and Deaths Information Statements, and also the relevant indexes at the Registrar's Office in an effort to identify all Aboriginal vital events that had been registered. The following definitions were used in the review:

1. state of registration—the State or Territory in which the death was registered and would normally be the State or Territory where the death actually occurred;
2. state of usual residence—the State or Territory where the deceased normally lived;
3. date of occurrence—the date when the death actually occurred; and
4. date of registration—the date when the death was registered by the Registrar of Births, Deaths and Marriages.

In addition to standard details about death and Aboriginal origin, information was extracted and listed for:

1. 1989 registrations identified from AHO information for future use;
2. deaths noted by AHO but not matched against the registrations;
3. death registrations overlooked in the initial scrutiny but subsequently identified, mainly from AHO information; and
4. interstate deaths reported by AHO and matched against the ABS DEMOSS file for usual residents of South Australia who died in other States (mainly the Northern Territory).

Confidentiality and privacy

Confidentiality and privacy considerations restricted the number and type of cross-checks that could be undertaken as part of this review. Administrative by-product collections were the source of the information for persons identifying themselves as Aboriginal and each of the agencies involved had confidentiality and privacy constraints.

The Principal Registrar of Births, Deaths and Marriages is the legal owner of Births and Deaths registration information. The ABS is provided access to this information for statistical purposes, but under the Census and Statistics Act the ABS cannot release information so obtained about individual persons unless returning it to

the source (the Registrar). The Registrar approved AHO accessing the Birth and Death Information Statements and the Indexes for the purpose of this review. The AHO allowed ABS officers to work with their officers to cross-check their information concerning deaths advised to them by Aboriginal Health Workers.

Sources of information on Aboriginal deaths

An initial listing from the 1988 ABS DEMOSS file identified 85 Aboriginal deaths registered in that year. Subsequently it was found that three of these were most unlikely to have been Aboriginal (despite the Aboriginality box having been ticked) because of country of birth, and other information in the Death Information Statements.

With the assistance of health workers located in the various Aboriginal communities and at hospitals, the AHO provided several lists of known Aboriginal people who had died in 1987 or 1988. An attempt was made to match these names with registered information statements and an additional 25 Aboriginal deaths were quickly identified. However there were some names which were not easily matched. Some of the deaths were found to be 1989 registrations even though a large number had occurred well before the end of 1988. Also a number of the reported deaths had not been registered pending the outcome of coronial enquiries.

The matching process proved to be an involved task for a variety of factors, including reports for which names had to be deleted and instances where women's names had changed (referred to in other research literature as female invisibility). There were several reported deaths for which no registration details or coroners' files could be located. Also, a number of the reported deaths were found to be of South Australian residents whose deaths had occurred and been registered in the Northern Territory.[2]

Of the 115 Aboriginal deaths registered in South Australia in 1988, 16 actually occurred in 1987, and two in 1986. There were 18 deaths that involved usual residents of other States or Territories (13 from the Northern Territory, three from New South Wales, one from Western Australia and one from Queensland). An interrogation of DEMOSS showed that in 1988, the deaths of nine Aborigines who were usual residents of South Australia were registered in the Northern Territory. It should be noted that the recording of deaths of South Australian residents occurring in other States and Territories is affected by the differing registration procedures across States and Territories. Queensland, for example, does not record Aboriginality on its registration forms. The gap between date of occurrence and date of registration for deaths registered in South Australia during 1988 is summarised in Table 1. Registrations were recorded for 86 (75 per cent) of the deaths in the month the death occurred or the month after the death occurred.

Table 1 Months between date of occurrence and date of registration of deaths in South Australia, 1988.

Period in months	N
<3	99
3-6	10
7-9	1
9+	5
	115

The scrutiny of the Death Information Statements identified an additional 30 Aboriginal deaths that were not recorded as such by DEMOSS. Of these there were six cases on old forms that did not include the Aboriginality question, six cases on new forms not ticked as Aboriginal, and 18 cases on new forms ticked as Aboriginal but which had been inadvertently overlooked in loading the computer system.

The AHO provided details of 81 deaths that occurred in 1988. Of these 47 were picked up in the scrutiny of the 1988 Death Information Statements; 11 were 1989 registrations, and nine were registered in the Northern Territory. No record in either the Registrar's system or the Coroner's Office was located for four of the deaths advised by AHO. The advices from AHO resulted in three cases being detected that would not have otherwise been found. One of these involved a death that had been recorded as Aboriginal, but had been inadvertently missed in the scrutiny. In the other two cases, there was insufficient information available on the Death Information Statement to enable the deceased to be identified as Aboriginal.

Included in the above were 26 deaths reported from the North West Statistical Division. They did not include names for privacy reasons and this made the matching exercise extremely difficult, although it is believed that approximately 20 deaths were accounted for, including nine which were registered in the Northern Territory. There were 16 Aboriginal deaths reported using the old Death Information Statement which did not include the Aboriginality question. Nine of these deaths were originally recorded on DEMOSS as Aboriginal. Of the 99 Aboriginal deaths reported on the new form, there were 82 cases where the Aboriginality question was ticked 'Yes'. In addition there were three cases where the 'Yes' box was ticked, but country of birth and other information on the Death Information Statement clearly indicated that the deceased was not Aboriginal.

Staff in the Registrar's Office highlighted with a red biro approximately half of the Aboriginal deaths registered in 1988 and did not punch the Aboriginal identifier for 19 deaths where the Aboriginal 'Yes' box had been ticked. Of the deaths omitted, 14 occurred in the period January-April 1988. After the problem was discussed with the Registrar, a system was introduced for the Registrar's staff to highlight any Aboriginal responses on the Birth and Death Information Statements to ensure correct punching. The proposed scrutiny of all Death Information Statements by ABS or AHO staff would also help overcome any problems in this area.

A search of the Registrar's Death Index was made for common and tribal Aboriginal names. The registration numbers were listed and checked against the Aboriginal deaths already recorded. This check did not disclose any death not already identified. Details of all deaths in the Port Augusta, Unincorporated Flinders Ranges

and the Far North Subdivision were listed from DEMOSS and scrutinised. No additional Aboriginal deaths were identified from this check.

Funeral directors have a key role in the recording of Aboriginal deaths, as they usually complete the Death Information Statement. The examination of the Death Information Statements did not reveal any pattern of reporting errors involving funeral directors, either individually or as a group. However, the cross-checking indicated that the deceased was Aboriginal in six cases where the 'No' box was ticked for the Aboriginality question on the Death Information Statement, and one case where neither the 'Yes' or 'No' box was completed. It is suggested that the Registrar, ABS and AHO jointly approach funeral directors at each of their locations to stress the importance of the Aboriginality question and request them to destroy any old forms they may still have in stock. The inclusion of some of the Aboriginal death statistics may be helpful in such an approach to funeral directors, to emphasise the need for an accurate response.

The South Australian Health Commission Inpatient Separation Information system has recorded 58 Aboriginal deaths in participating hospitals (all recognised hospitals plus a small number of private hospitals) in 1987-88, which is roughly comparable with the number of Aboriginal deaths recorded by DEMOSS in 1988 as having occurred in hospitals. No attempt was made to obtain the names of the Aboriginal people involved as the system does not record the individuals names and it would therefore be necessary to write to hospitals to obtain them. It is likely that problems may be experienced with privacy issues when attempting to obtain the names from hospitals.

Conclusions

The number of identifiable Aboriginal deaths registered in South Australia in 1988 was 115. Of these only 82 were correctly recorded in the ABS demographic statistics processing system prior to the review. Table 2 below summarises the outcome of the scrutiny and cross-checking exercises that comprised the review. The majority of the additional Aboriginal deaths identified in the review were revealed through the scrutiny of the Death Information Statements. An ongoing scrutiny of all Death Information Statements will greatly improve the recording of Aboriginal deaths which are ultimately processed in the ABS. Some further benefit may be gained by negotiation with the AHO to allow their staff to conduct the scrutiny, as they have further information from their own sources to supplement the examination.

Table 2 Aboriginal deaths registered in South Australia, 1988.

	Old forms	New forms	Total
Deaths originally recorded	9	76	85
Less deaths with Aboriginality incorrectly recorded	-	3	3
Sub total	9	73	82
Deaths identified in the review	7	26	33
Total Aboriginal deaths identified	16	99	115

The use of old forms that do not include the Aboriginality question continues to decline but it is recommended that the Registrar be approached to seek his cooperation in eliminating the use of old forms quickly. The abandoning of old forms would lead to an improvement in the quality of data in the recording system.

A small number of additional deaths (three) were identified through the examination of independent records from the AHO. In addition there were details of four deaths provided by AHO which could not be matched with registrations. Despite the fact that the officers involved in the review feel that the 115 Aboriginal deaths identified understate the true figure by an estimated 15 to 20 deaths, the Registrar's data are believed to be the best available source of Aboriginal deaths information, particularly when enhanced by a scrutiny of Death Information Statements. The knowledge and assistance of the AHO is needed to facilitate the improvement of Aboriginal deaths data.

NOTES

1. This chapter was presented at the Workshop on behalf of the authors from the ABS and the AHO of South Australia, by Mr Dan Black, also from the ABS.
2. Cultural taboos do not allow easy access to information relating to deceased persons, nor for this information to be released outside the community concerned.

12 INSIGHTS INTO ABORIGINAL MORTALITY IN WESTERN NEW SOUTH WALES

Robert Hogg

A recent report written by Gray and Hogg (1989) shows contemporary Aboriginal mortality in the western region of New South Wales to be at unacceptably high levels. In particular, estimates drawn from this research show that in comparison with the total New South Wales population, Aboriginal men and women have higher levels of mortality at all ages, but especially during early and middle adulthood; much lower life expectancies; and a greater incidence of deaths due to accidents, poisonings, and violence, circulatory and respiratory system diseases. Overall, this study shows that the pattern of Aboriginal mortality is quite distinct from the New South Wales one and this distinctiveness is in part a product of the social and environmental context in which Aboriginal people live.

The purpose of this chapter is to further highlight contemporary Aboriginal mortality in western New South Wales by expanding upon and reviewing features outlined in the previous report. It will be argued that: the Aboriginal mortality regime is significantly different from that of the total New South Wales population; mortality levels experienced by Aboriginal people are not homogeneous and are sometimes influenced by local socio-cultural conditions; and the association between various causes of death provides some insight into the relationship between Aboriginal lifestyle and mortality. Because of the nature of this argument, the chapter examines Aboriginal mortality trends at three different levels. First, at a regional level, a comparison between Aboriginal and New South Wales mortality rates and causes of death is made, and second, at a community level, a comparison between community mortality levels. Third, at an individual level, the association between various causes of death is categorised and statistically explored. Finally, the goal of this work is to leave the reader with an insightful glimpse into Aboriginal mortality in one region of Australia and with an interpretation of why Aboriginal mortality is so distinct from that of other Australians.

Methods

This enquiry was the first in a series of regional studies conducted by the Aboriginal Health Unit and The Australian National University to update estimates of Aboriginal mortality in the country regions of New South Wales. As the first study, this project concentrated on gathering information on Aboriginal mortality for the years 1984 to 1987 in communities located in the Far West, New England, and Orana regions—the western regions of the State. Overall, the methodological approach taken was patterned after the last Aboriginal Health Unit mortality study in 1983 (Julienne et

al 1983; Smith et al 1983; Thomson and Smith 1985). However, this approach was often modified to meet the operational requirements of the project.

In this study three main sources of mortality data were used. All suffered from serious drawbacks or limitations, but together provided reliable information. The first and the most substantial source of data was the records of registration of death held by local registry offices throughout the State. Information was gathered by visiting and searching through registries located in small to medium-sized communities in the western region or in larger centres such as Broken Hill and Sydney. Aboriginal informants were used to identify deaths and to supplement these records with additional background information. The informants were asked about the deceased's marital status and whether the person was pre-deceased by parents, spouse, and/or children; owned or rented his or her house or flat; had a job in the previous 12 months; and lived in town or on a mission (former reserve). The main problem with this source of data is that local registries are no longer required to contain information on the deaths of people who died locally or were buried locally after dying elsewhere.

The other two sources of mortality data were used to supplement listings of Aboriginal deaths obtained from local registry records. The first source was the yearly listings of deaths produced by the Principal Registrar from notifications for registrations. These yearly listings have been produced since 1980 and are generated by including a question on the death record notification form about whether the deceased was considered to be Aboriginal or not. The main drawback of this source is that these listings are incomplete (Gray 1983b). The second supplementary source was the system put in place by the Aboriginal Health Unit in 1978. In this case health workers are responsible for completing notification forms for all Aboriginal deaths that occur in their health district. The basic details found on these notifications are then forwarded to regional and central offices. Unfortunately, for this period and area of study, these records were also incomplete.

In total, information on 394 Aboriginal deaths was obtained from these three sources of data. The data set included deaths which occurred outside the study area or in communities in which the enumeration of death was believed to be incomplete. Therefore, instead of basing estimates of Aboriginal mortality on the total number of deaths, calculations were restricted to 315 deaths (205 male and 110 female) from the communities of Bourke, Brewarrina, Collarenebri, Condobolin, Coonamble, Dubbo, Gilgandra, Gingie, Goodooga, Gulargambone, Lightning Ridge, Menindee, Moree, Murrin Bridge, Lake Cargellico, Narrabri, Walgett, Wellington, and Wilcannia, in which the enumeration of deaths was believed to be complete.

Estimates of the population living in and around these 19 communities on 31 December 1985, the mid-point of the four year study period, were obtained by applying a limited cohort-component method of reverse survival to 1986 Census figures (Shryock and Siegel 1971, 740-41). The technique projected the Aboriginal population in these communities in the period 31 June 1986 backwards to 31 December 1985, using constructed life table survivor ratios. The Aboriginal population on 31 December 1985 was estimated to be 9,357 of whom 4,629 were male and 4,728 were female.

Rates of mortality at regional level

This is the first of three levels at which mortality data were analysed. Aboriginal mortality rates and causes of death were examined and compared with those of the New South Wales population. In particular, mortality rates were produced in the form of sex-specific crude and age-specific death measures. and abridged life tables; and causes of death were examined by comparing age and sex-specific differences in the major classes of death. Finally, the main purpose behind this comparison was to show that the Aboriginal mortality regime is significantly different from that of the New South Wales population.

Overall, the level of Aboriginal mortality in western New South Wales is markedly higher than that of the New South Wales population in 1986 (see Table 1). For all age groups, the Aboriginal crude death rate was 8.4 per 1,000, or 11.1 per 1,000 for males and 5.8 per 1,000 for females. This compares with a New South Wales crude death rate of 7.6 per 1,000, or 8.2 per 1,000 for males and 7.1 per 1000 for females.

The problem with this comparison is that the above rates have been seriously affected by their populations' age structure. As shown in Table 2, the New South Wales population has lower dependency ratios and an older age structure than the Aboriginal population. Approximately 21 per cent of the New South Wales population are less than 15 years of age and more than 10 per cent are 65 years and over. This compares with the Aboriginal population in which about 45 per cent are less than 15 years of age and less than five per cent are 65 years and over. Therefore, the New South Wales population, with a larger proportion of old people, will generally have higher sex-specific crude death rates, because older people are at a much greater risk of death than younger people. If the Aboriginal population had the same age structure as the New South Wales population then its directly standardised crude death rates[1] would have been 19.5 per 1,000 or 24.0 per 1,000 for males and 15.2 per 1,000 for females. Put in these terms, the Aboriginal mortality rate for males is about three times and for females is about twice the rate for the New South Wales population.

Another way of examining the relationship between age structure and mortality levels is by looking at the component differences between Aboriginal and New South Wales crude death rates. Using a component analysis the differences between these two crude death rates can be broken down into the differences due to age-specific mortality rates and age composition (Kitagawa 1955). Also, the interaction or residue around each component can be measured. This uncertainty can be interpreted as meaning that each component could vary by plus or minus that particular increment from the calculated value. Results from this analysis demonstrate that the level of Aboriginal mortality is higher than the New South Wales one, and that the lower Aboriginal crude death rate for females is a product of the different age distributions (see Table 2). The measure of residue or interaction shows that these two components, for either sex, are highly variable in magnitude but not in degree.

Table 1 Crude and standardised death rates[1], standardised mortality ratios and component differences for Aborigines in western New South Wales, 1984-87, and the New South Wales population, 1986.

	Males	Females	Persons
Aboriginal, 1984-87			
Total number of deaths	210	105	315
Crude death rate	11.1	5.8	8.4
Standardised rate[2]	24.0	15.2	19.5
New South Wales, 1986[3]			
Total number of deaths	22,571	19,596	42,167
Crude death rate	8.2	7.1	7.6
Standardised mortality ratio[4]	2.9	2.2	2.6
Components of difference[5]			
Due to mortality	11.8	5.9	8.8
Due to population composition	-8.9	-7.2	-8.0
Interaction/residual	-4.0	-2.2	-3.1
Difference between the two crude rates	2.9	-1.2	0.8

NOTES:
1. Rates are deaths per 1,000 population.
2. Directly standardised Aboriginal crude death rate based on the New South Wales age structure, 31 June 1986.
3. Adapted from ABS publication *Deaths in New South Wales 1986*.
4. The standardised mortality ratio is a ratio of the Aboriginal standardised crude death rate to the New South Wales population crude death rate.
5. Component differences between Aboriginal and New South Wales crude death rate.

Table 2 Estimated Aboriginal[1] and New South Wales[2] population by age group, 1986.

	Aboriginal		New South Wales	
Age in years	Males	Females	Males	Females
0–4	696	669	211,978	202,032
5–9	609	600	205,612	196,028
10–14	639	641	227,319	216,825
15–19	627	630	225,854	215,706
20–24	450	488	230,126	220,572
25–29	355	391	232,913	228,009
30–34	271	273	217,404	216,414
35–39	253	233	220,857	213,278
40–44	193	207	181,363	171,925
45–49	162	172	152,164	143,907
50–54	133	134	133,614	127,858
55–59	80	98	137,862	134,028
60–64	47	76	126,988	134,181
65–69	49	48	96,881	112,026
70–74	33	41	75,136	95,771
75+	32	27	80,919	145,976
All ages	4,969	4,728	2,756,990	2,774,536
Dependency ratio[3]	80.0	75.0	48.3	53.6

NOTES:
1. Based on the estimated resident population of Aborigines living in and around 19 western New South Wales communities, 31 December 1985.
2. Based on the preliminary estimated resident population of New South Wales, 30 June 1986. For further details, see the ABS publication *Estimated Resident Population by Sex and Age: States and Territories of Australia June 1981 to June 1987.*
3. The dependency ratio is a ratio of the population in the age group 0-14 and 65 and over, to the population in the age group 15-64 years.

These death rates have little meaning unless they are related to specific age groups or compared with those of other populations. In this case, to make these measures more meaningful, Aboriginal death rates in this region were compared to rates of the New South Wales population (Australian Bureau of Statistics 1988b, 1988c). The calculations involved in this comparison took place in three stages. First, age intervals were grouped so as to limit the effects of small numbers on the calculation of age-specific rates. Second, age-specific relative risks, a ratio of the Aboriginal rate to the New South Wales rate, were determined. Third, confidence intervals, at a 95 per cent

level of confidence, were constructed around each ratio of relative risk (Bailar and Ederer 1964).

A comparison between Aboriginal and New South Wales age-specific death rates reveals a consistently higher relative risk of death experienced by Aboriginal men and women in most age groups, but especially between the ages of 25 and 45 years (see Table 3). The lower confidence intervals of relative risk range from about one to over four, and the upper intervals range from two to over 14. Overall, these relative risks suggest a pattern of gradually increasing relative risk until the age of 45, then gradually declining to old age.

Another important descriptive measure of overall mortality is based on a life table function. This type of analysis provides a way of linking observable events to the consequences these events would have on a hypothetical population, free of the effects caused by demographic disturbances (Howell 1979, 74). Life tables can provide a range of descriptive functions, but the expectation of life is the most commonly used. As shown in the last column of Tables 4 and 5, the life expectancy at birth for Aboriginal men was about 53 years and women about 64 years. However, Aboriginal

Table 3 Age–specific mortality rates (ASMR)[1] and relative risks[2] for Aborigines in western New South Wales, 1984-87, and New South Wales,[3] 1986.

Age in years	Aborigines		New South Wales		Relative
	Deaths	ASMR	Deaths[4]	ASMR	risk range
Males					
0–4	27	9.7	514	2.4	(2.7-6.1)
5–14	2	0.4	120	0.3	(0.4–11.9)
15–24	16	3.7	645	1.4	(1.6–4.6)
25–34	22	8.8	570	1.3	(4.6–11.1)
35–44	21	11.8	768	1.9	(4.0–10.0)
45–54	32	27.1	1,428	5.0	(3.8–7.9)
55–64	37	72.8	3,843	14.5	(3.6–7.1)
65+	48	105.3	14,671	58.0	(1.4–2.5)
Females					
0–4	11	4.1	389	1.9	(1.2–4.3)
5–14	3	0.6	75	0.2	(1.1–14.1)
15–24	8	1.8	209	0.5	(1.9–8.7)
25–34	9	3.4	229	0.5	(3.5–14.3)
35–44	10	5.7	395	1.0	(3.0–11.5)
45–54	17	13.9	810	3.0	(2.9–8.0)
55–64	18	25.9	2,079	7.8	(2.1–5.6)
65+	34	73.3	15,408	43.6	(1.2–2.4)
Persons					
0–4	38	7.0	903	2.2	(2.3–4.5)
5–14	5	0.5	195	0.2	(0.9–6.7)
15–24	24	2.7	854	1.0	(1.9–4.5)
25–34	31	6.0	799	0.9	(4.7–9.9)
35–44	31	8.7	1,163	1.5	(4.2–8.7)
45–54	49	20.4	2,238	4.0	(3.8–6.9)
55–64	55	45.7	5,922	11.1	(3.2–5.5)
65+	82	89.1	30,079	49.6	(1.4–2.3)

NOTES:
1. Rates are deaths per 1,000 population.
2. Relative risk is the ratio of the observed Aboriginal rate to the New South Wales rate. The relative risk range represents confidence intervals at a 95 per cent level of probability around each ratio.
3. Adapted from ABS publication *Deaths in New South Wales 1986.*
4. Excluding age not stated deaths.

Table 4 Aboriginal male abridged life table for western New South Wales, 1984-87.

x	n	Males	Deaths	nMx	nqx	lx	ndx	nLx	Tx	ex0
0	1	132	21	0.03977	0.03862	100,000	3,862	97,204	5,346,530	53.5
1	4	564	6	0.00266	0.01057	96,138	1,016	382,154	5,249,326	54.6
5	5	609	1	0.00041	0.00205	95,122	195	475,122	4,867,172	51.2
10	5	639	1	0.00039	0.00195	94,927	186	474,170	4,392,050	46.3
15	5	627	5	0.00199	0.00992	94,741	940	471,353	3,917,880	41.3
20	5	450	11	0.00611	0.03010	93,802	2,823	461,950	3,446,523	36.7
25	5	355	12	0.00845	0.04138	90,979	3,765	445,481	2,984,573	32.8
30	5	271	10	0.00923	0.04509	87,214	3,932	426,239	2,539,091	29.1
35	5	253	10	0.00988	0.04822	83,282	4,016	406,370	2,122,852	25.4
40	5	193	11	0.01425	0.06879	79,266	5,453	382,699	1,706,482	21.5
45	5	162	10	0.01543	0.07429	73,813	5,484	355,357	1,323,783	17.9
50	5	133	22	0.04135	0.18739	68,329	12,804	309,636	968,426	14.2
55	5	80	23	0.07188	0.30464	55,525	16,915	235,337	658,790	11.9
60	5	47	14	0.07447	0.31390	38,610	12,120	162,751	423,452	11.0
65	5	49	8	0.04082	0.18519	26,490	4,906	120,188	260,701	9.8
70	5	33	20	0.15252	0.54945	21,585	11,860	78,274	140,514	6.5
75	+	32	20	0.15625	1.00000	9,725	9,725	62,240	62,240	6.4
Total	4,629		205							

Table 5 Aboriginal female abridged life table for western New South Wales, 1984-87.

x	n	Females	Deaths	nMx	nqx	lx	ndx	nLx	Tx	ex0
0	1	168	10	0.01488	0.01472	100,000	1,472	98,935	6,478,048	64.8
1	4	501	1	0.00050	0.00199	98,528	196	393,650	6,379,113	64.7
5	5	600	1	0.00042	0.00208	98,332	205	491,148	5,985,463	60.9
10	5	641	2	0.00078	0.00389	98,127	382	489,682	5,494,315	56.0
15	5	630	5	0.00198	0.00987	97,745	965	486,314	5,004,634	51.2
20	5	488	3	0.00154	0.00766	96,780	741	482,050	4,518,319	46.7
25	5	391	4	0.00256	0.01271	96,040	1,220	477,147	4,036,270	42.0
30	5	273	5	0.00458	0.02263	94,819	2,146	468,731	3,559,123	37.5
35	5	233	6	0.00644	0.03168	92,673	2,936	456,026	3,090,392	33.3
40	5	207	4	0.00483	0.02387	89,737	2,142	443,332	2,634,366	29.4
45	5	172	5	0.00727	0.03569	86,596	3,126	430,162	2,191,034	25.0
50	5	134	12	0.02239	0.10601	82,469	8,954	399,961	1,760,872	20.9
55	5	98	10	0.02551	0.11990	75,515	9,055	354,939	1,360,911	18.0
60	5	76	8	0.02630	0.12346	66,460	8,205	311,790	1,005,972	15.1
65	5	48	8	0.04167	0.18868	58,255	10,992	263,798	694,183	11.9
70	5	41	7	0.04268	0.19284	47,264	9,114	213,534	430,384	9.1
75	+	27	19	0.17593	1.00000	38,150	38,150	216,850	216,850	5.7
Total	4,728		110							

men and women still live approximately 15 to 20 years less than in the total New South Wales population. In 1986 the life expectancies at birth for men and women of the New South Wales population were 73 and 79 years respectively. It is clear from this comparison that the gap between the Aboriginal and New South Wales populations is alarmingly large.

Causes of death were coded using ICD-9 (World Health Organization 1977). The coding of the underlying cause of death was the code obtained from the yearly listings of Aboriginal deaths produced by the Principal Registrar, except in cases where the deaths were not listed. In these cases codes were assigned according to standard procedures. These codes were then used to calculate age and sex-specific death rates for the major classes of death.

As before, a comparison with the New South Wales population was done by directly standardising Aboriginal cause-specific rates to the New South Wales age structure, grouping age intervals, and constructing measures of relative and excess risk. Excess risks were derived by subtracting cause-specific death rates for the New South Wales population from the rates for the Aboriginal population. The proportion

of excess risk was then determined by producing a ratio of cause-specific excess risk to the excess risk for all causes.

In comparing Aboriginal and New South Wales age and sex–specific rates, major differences in cause-specific death rates can be found (see Tables 6 to 9). The most apparent difference between these two populations is the incidence of circulatory system diseases. In comparison with the New South Wales population, diseases of the circulatory system made up over 40 per cent of the excess risk experienced by Aboriginal men and women with the relative risk of death being the highest between the ages of 25 and 65 years.

Table 6 Male cause–specific mortality rates (CSMR),[1] relative risks[2] and proportion of excess[3] risk for Aborigines in western New South Wales, 1984-87, as compared with the New South Wales population,[4] 1986.

Causes of death (ICD-9 code)	Aboriginal Deaths	CSMR	NSW CSMR	Relative risk range	Proportion of excess risk
All causes	205	24.0	11.1	(2.6–3.4)	100.0
Infectious and parasitic (I)	4	0.2	0.0	(1.5–13.8)	0.8
Neoplasms (II)	21	3.1	2.1	(1.0–2.4)	6.6
Endocrine, nutritional and metabolic (III)	2	0.1	0.2	(0.3–7.6)	0.0
Mental disorders (V)	3	0.3	0.1	(1.3–18.7)	1.6
Nervous system and sense organs (VI)	2	0.2	0.1	(0.4–11.3)	0.3
Circulatory system (VII)	84	13.0	3.8	(2.8–4.3)	57.6
Respiratory system (VIII)	10	1.4	0.6	(1.2–4.7)	5.1
Digestive system (IX)	17	1.8	0.1	(10.0–27.6)	10.4
Genito–urinary system (X)	3	0.5	0.1	(1.5–21.6)	2.4
Congenital anomalies (XIV)	5	0.2	0.0	(1.2–8.8)	0.6
Perinatal conditions (XV)	6	0.2	0.1	(1.3–7.7)	0.7
Symptoms and ill-defined conditions (XVI)	18	1.0	0.1	(10.0–26.6)	5.6
External causes (XVII)	30	2.0	0.7	(2.1–4.5)	8.6

NOTE: See Notes to Table 7.

Table 7 Female cause-specific mortality rates (CSMR),[1] relative risks[2] and proportion of excess risk[3] for Aborigines in western New South Wales, 1984-87 compared with the New South Wales population,[4] 1986.

Causes of death (ICD-9 code)	Aboriginal Deaths	CSMR	NSW CSMR	Relative risk range	Proportion of excess risk
All causes	110	15.1	7.1	(1.8–2.6)	100.0
Infectious and parasitic (I)	6	0.2	0.0	(3.5–20.7)	2.3
Neoplasms (II)	10	1.9	1.6	(0.6–2.4)	0.2
Endocrine, nutritional and metabolic (III)	8	1.4	0.1	(4.8–21.8)	15.2
Mental disorders (V)	2	0.1	0.1	(0.4–10.7)	0.4
Nervous system and sense organs (VI)	3	0.1	0.2	(0.4–6.2)	0.4
Circulatory system (VII)	41	7.5	3.7	(1.5–2.8)	45.3
Respiratory system (VIII)	9	1.5	0.5	(2.2–9.0)	14.2
Digestive system (IX)	4	0.5	0.3	(0.7–6.3)	2.5
Genito–urinary system (X)	5	0.7	0.1	(2.7–19.4)	7.0
Congenital anomalies (XIV)	2	0.0	0.0	(0.3–7.6)	0.0
Perinatal conditions ((XV)	2	0.0	0.0	(0.2–7.3)	0.1
Symptoms and ill–defined conditions (XVI)	3	0.2	0.0	(1.4–20.3)	1.6
External causes (XVII)	15	1.0	0.3	(2.0–5.8)	8.3

NOTES:
1. Rates are deaths per 1,000 population. Aboriginal cause-specific rates were calculated by directly standardising Aboriginal cause-specific rates to the New South Wales age structure.
2. Relative risk is the ratio of the directly standardised Aboriginal rate to the New South Wales rate. The relative risk range represents confidence intervals at a 95 per cent level of probability around each ratio.
3. The proportion of excess risk was derived by subtracting cause-specific death rates for the New South Wales population from directly standardised Aboriginal rates.
4. Adapted from ABS publication *Causes of Death, New South Wales 1986.*

Table 8 The major causes of male Aboriginal mortality (CSMR)[1] in western New South Wales, 1984-87, by age category compared[2] with the New South Wales population,[3] 1986.

Causes of death (ICD-9 code)	Aboriginal Deaths	CSMR	NSW CSMR	Relative risk range	Proportion of excess risk
Under 1 year					
All causes	21	39.8	10.3	(2.5–6.2)	100.0
Symptoms and ill-defined conditions (XVI)	9	17.0	2.9	(3.1–12.8)	48.1
Perinatal causes (XV)	6	11.4	4.0	(1.3–7.7)	25.0
Congenital anomalies (XIV)	4	7.6	2.4	(1.2–11.5)	17.5
1 to 24 years					
All causes	24	2.1	0.8	(1.8–4.1)	100.0
External causes (XVII)	14	1.2	0.6	(1.3–4.0)	51.5
25 to 44 years					
All causes	43	10.0	1.6	(4.7–8.8)	100.0
Circulatory system (VII)	15	3.5	0.3	(7.4–21.8)	38.0
External causes (XVII)	9	2.1	0.7	(1.6–6.5)	16.5
Digestive system (IX)	7	1.6	0.3	(2.7–13.9)	15.8
45 to 64 years					
All causes	69	18.2	6.1	(2.4–3.9)	100.0
Circulatory system (VII)	36	8.9	2.0	(3.2–6.4)	56.5
Neoplasms (II)	11	2.6	0.1	(10.3–37.0)	20.3
Digestive system (IX)	6	1.6	2.8	(0.3–1.5)	-9.8
Respiratory system (VIII)	5	1.6	0.3	(2.1–14.8)	10.2
65 years and over					
All causes	48	105.3	58.0	(1.2–2.4)	100.0
Circulatory system (VII)	32	70.2	30.7	(1.6–3.3)	83.5
Neoplasms (II)	7	15.4	14.6	(0.5–2.6)	1.7

NOTE: See Notes to Table 7.

Table 9 The major causes of female Aboriginal mortality (CSMR)[1] in western New South Wales, 1984-87, by age category as compared[2] with the New South Wales population,[3] 1986.

Causes of death (ICD-9 code)	Aboriginal Deaths	CSMR	NSW CSMR	Relative risk range	Proportion of excess risk
Under 1 year					
All causes	10	14.9	8.0	(1.0–3.9)	100.0
Infectious and parasitic (I)	4	6.0	0.1	(31.3–293.4)	85.6
Congenital anomalies (XIV)	2	3.0	2.1	(0.4–11.7)	12.6
Perinatal conditions (XV)	2	3.0	3.4	(0.2–7.3)	-5.5
1 to 24 years					
All causes	12	1.0	0.3	(1.7–5.9)	100.0
External causes (XVII)	9	0.8	0.2	(2.0–9.0)	84.9
25 to 44 years					
All causes	19	4.3	0.8	(3.7–9.5)	100.0
Circulatory system (VII)	4	0.9	0.1	(3.2–30.3)	22.4
External causes (XVII)	3	0.7	0.2	(1.2–16.8)	13.6
45 to 64 years					
All causes	35	18.2	6.1	(2.2–4.3)	100.0
Circulatory system (VII)	17	8.9	2.0	(2.8–7.7)	56.5
Endocrine, nutritional and metabolic (III)	5	2.6	0.1	(7.9–57.2)	20.3
Respiratory system (VIII)	3	1.6	0.3	(1.6–23.3)	10.2
Neoplasms (II)	3	1.6	2.8	(0.2–2.8)	-9.8
65 years and over					
All causes	34	73.3	43.6	(1.2–2.4)	100.0
Circulatory system (VII)	19	40.9	26.3	(1.0–2.6)	49.9
Neoplasms (II)	5	10.8	8.2	(0.6–4.1)	8.6
Respiratory system (VIII)	4	8.6	2.4	(1.4–13.4)	21.1
Endocrine, nutritional and metabolic (III)	3	6.5	0.9	(2.4–33.5)	18.6

NOTE: See Notes to Table 7.

After the age of 65 years the relative risk range suggests that Aboriginal sex-specific circulatory system death rates are probably approaching New South Wales levels. The greatest proportion of the 125 deaths due to circulatory system disease were

from ischaemic heart disease (70 deaths or 56 per cent) and cerebro-vascular disease (32 deaths or 26 per cent). Overall, in this region Aboriginal people have a high risk of heart disease especially during middle-age. In fact, an effective reduction of circulatory system diseases through health education would be one of the best ways of reducing Aboriginal death rates in this region to the levels exhibited by the New South Wales population.

External causes were also a major cause of death. In general, accidents, poisonings and violence accounted for approximately eight per cent of the excess risk to Aboriginal people. The relative risk of death from these causes for both sexes was at least twice the rate experienced by the New South Wales population. The major proportion of these 45 deaths, which disproportionately occur between the ages of 15 and 29 years, were from motor vehicle accidents (22 deaths or 49 per cent) and violence (nine deaths or 20 per cent). The specific causes of violent deaths include stabbing, shooting, manual strangulation and inflicted head injuries. Other deaths due to violent acts include a suicide by hanging and a death in police custody. Finally, this category of deaths includes a small proportion of deaths due to several separate house fires (five deaths or 11 per cent).

Among men other important categories of death include diseases of the digestive system, malignant neoplasms (cancer), and respiratory system diseases. In total, these diseases made up over 20 per cent of the excess risk. Overall, the major proportion of the 17 deaths caused by digestive system diseases were from chronic liver disease and alcoholic cirrhosis (13 deaths or 76 per cent). Nearly half (nine deaths or 42 per cent) of the 21 deaths from cancer were from lung, pancreas and prostate cancers. Finally, seven of the nine deaths from respiratory system disease were from chronic obstructive lung disease (respiratory disease due to smoking).

Among women other important causes include diseases of the endocrine and respiratory systems. These causes of death made up approximately 30 per cent of the excess risk. Seven of the eight deaths from endocrine disorders were from diabetes. Chronic obstructive lung disease was responsible for six of the nine deaths due to respiratory system diseases.

Additionally, infant deaths (those under one year) were generally from four groups of causes—congenital anomalies, infectious diseases, perinatal causes, and sudden infant death syndrome. However, sudden infant death syndrome is not actually a cause of death, but rather is a category used by practitioners to indicate that despite exhausting all possibilities a cause could not be found. Of the seven reported cases of this condition all were baby boys. This high incidence of deaths due to unknown causes should be seen as a major health concern in this region. There are also some notable causes of death in this data set which should be mentioned. These are deaths caused by tuberculosis (one active case), epilepsy (six deaths), narcotics dependency (three deaths), acquired immune deficiency syndrome (one case from blood transfusion), and complications of measles (one death).

Rates of mortality at community level

Community mortality patterns were compared in order to highlight the mortality differentials between communities; and to examine the relationship between community mortality patterns and two socioeconomic variables—household size and employment. Specifically, this section has attempted to show that community mortality levels are not homogeneous and are sometimes influenced by local sociocultural conditions.

As mentioned previously (Gray and Hogg 1989, 41), differences in the social environments of Aboriginal people living in various communities in western New South Wales are evident even to the most casual observer. In the largest communities, like Dubbo and Moree, Aboriginal people make up a small proportion of the total population and are generally urban residents. In other smaller country towns, like Brewarrina, Bourke, Walgett, and Wilcannia, Aboriginal people make up a substantial proportion of the total population and often live in former reserves or fringe settlements.

Somewhat less obvious are the differences in mortality rates between communities in this region. After standardising for different age-structures, communities in this study have average death rates (standardised mortality ratios) ranging from over half up to twice the regional average crude death rate (see Table 10). Additionally, after a simple chi-square test was employed, the observed number of deaths was shown to be significantly different from the expected number in half of these communities. These standard mortality ratios fall into three categories: a very high level in Bourke, Brewarrina, and Coonamble; a moderately high level in Walgett and Wilcannia; and a below average level in other communities.

Table 10 Standardised mortality ratios[1] for Aboriginal people living in selected western New South Wales communities, 1984-87.

	Observed deaths[2]	Expected deaths[3]	Standardised mortality ratio
Bourke	37	19.7	1.9[8]
Brewarrina	36	21.0	1.7[8]
Condobolin[4]	25	29.0	0.9
Coonamble[5]	27	13.1	2.1[8]
Dubbo	25	44.7	0.6[8]
Moree	45	53.3	0.8
Walgett[6]	49	35.8	1.4[8]
Wellington	14	22.2	0.6
Wilcannia	22	16.7	1.3
Other communities[7]	35	46.5	0.7

NOTES:

1. The standardised mortality ratio is the ratio of the observed to the expected number of Aboriginal deaths for both sexes if Aboriginal age-specific mortality for western New South Wales applied.
2. Observed deaths are the number of deaths which occurred in the community between 1984 and 1987.
3. Expected deaths are the actual number of deaths expected between 1984 and 1987 if age-specific death rates for Aboriginals in Western New South Wales applied.
4. Includes deaths from Lake Cargellico and Murrin Bridge.
5. Includes deaths from Gulargambone.
6. Includes deaths from Gingie.
7. Includes deaths from Collarenebri, Gilgandra, Goodooga, Lightning Ridge, Menindee and Narrabri.
8. A significant difference between the observed and expected number of deaths at a probability level of 0.05.

The question is why do some communities in this study have lower mortality rates than other communities? A rank correlation statistical test shows that two social variables can help explain these mortality differentials (see Table 11). The first variable is household size, where community mortality rates are negatively ($p<0.1$) correlated with the proportion of Aboriginal households with less than five members. The second is employment, where community mortality rates are negatively ($p<0.05$) correlated with the proportion of the Aboriginal labour force employed. Both these correlations are much stronger if only the communities which have significant differences in observed and expected number of deaths are ranked. In this case, the correlation between the standard mortality ratio and household size is -0.61 and employment is -0.82. Aboriginal mortality is lower in communities where households are smaller and where there is more employment.

Table 11 Rank correlation of Aboriginal standardised mortality ratios (SMR),[1] household size and employment[2] for communities in western New South Wales, 1984-87.

	SMR	Percentage of households <5 persons	Percentage of labour force employed
Bourke	1.9	27.5	34.1
Brewarrina	1.7	25.7	52.1
Condobolin[3]	0.9	30.8	41.6
Coonamble[4]	2.1	31.4	39.5
Dubbo	0.6	42.0	52.8
Moree	0.8	25.5	41.1
Walgett[5]	1.4	24.3	41.5
Wellington	0.6	31.4	53.9
Wilcannia	1.3	19.9	38.4
Other communities[6]	0.7	33.9	47.8
Rank correlation with SMR		-0.49	-0.68

NOTES:
1. The standardised mortality ratio is the ratio of the observed to the expected number of Aboriginal deaths for both sexes if Aboriginal age-specific mortality for western New South Wales applied.
2. Community-specific data on these two variables were obtained from the 1986 Australian census of population and housing.
3. Includes deaths from Lake Cargellico and Murrin Bridge.
4. Includes deaths from Gulargambone.
5. Includes deaths from Gingie.
6. Includes deaths from Collarenebri, Gilgandra, Goodooga, Lightning Ridge, Menindee and Narrabri.

Rates of mortality at individual level

The third and final dimension, at the individual level, employed two statistical tests to examine the sex-specific association between various causes of death. First, a chi-square test was used to determine if the relationship between causes of death was significant; and second, a measure of association between causes was calculated to determine the correlation for each significant relationship. Finally, these relationships were categorised under major classes of causes of death. The goal here was to provide some insight into the relationship between Aboriginal lifestyle and mortality.

This statistical procedure was restricted to standardised versions of variables and to records of deaths from the total data set for persons aged 15 years and over. The standardised versions of variables used in this exercise were derived by determining whether a particular condition was present or absent in a complete death record. These variables measured the occurrence of ischaemic heart disease, other forms of heart disease, liver disease, kidney disease, diabetes, other pancreatic disease, alcohol-related conditions, chronic obstructive lung disease, other airways disease, cancer, accidents, poisoning or violence, infections, epilepsy, other digestive disease, and being elderly or senile.

The relationships exhibited by these variables were then statistically explored and categorised, by major classes of death, into five separate tables. In the first, the sex-specific relationships between accidental and violent conditions and other causes of deaths is explored (see Table 12). Generally, the table shows that accidental and violent deaths occur to the exclusion of other causes. These relationships are not surprising considering most accidental and violent deaths occur disproportionately at younger ages and the other disease conditions predominantly at older ages.

Table 12 The measure of association[1] between accidental and violent conditions and other causes of death among Aborigines in western New South Wales, 1984-87.

Disease condition	Accidental and violent conditions	
	Males[2]	Females[3]
Ischaemic heart disease	-0.45	-0.37
Other forms of heart disease	-0.27	-0.26
Liver disease disorders	-0.19	
Diabetes	-	-0.22
Alcohol-related	-0.17	-
Other respiratory system diseases	-0.27	-0.22
Infectious diseases	-0.20	-

NOTES: 1.Associations between causes of death were determined by using chi-square and phi statistical tests. The associations shown in this table are all significant at a 0.05 probability level and can range in value from -1.0 to 1.0.
2. Based on 206 observations.
3. Based on 119 observations.

Table 13 The measure of association[1] between alcohol-related conditions and other causes of death among Australian Aborigines in western New South Wales, 1984-87.

Disease condition	Alcohol-related conditions	
	Males[2]	Females[3]
Liver disease	0.49	0.58
Diabetes	-	-2.10
Other pancreatic disorders	0.22	-
Cancer	-0.18	-
Accidental and violent conditions	-0.17	-
Infectious diseases	-	0.28
Epilepsy	-	0.48

NOTE: See notes to Table 12.

In Table 13, the association between alcohol-related conditions and other conditions is explored. For both males and females there is a strong relationship with liver disease. This strong association is not surprising considering most deaths due to liver disease in this data set are from alcoholic cirrhosis of the liver. A relatively strong relationship is also found with other pancreatic disorders among males and with infectious diseases and epilepsy among females. Overall, a strong association between alcohol-related conditions and liver disease for both sexes, and possibly a distinct sex-specific patterning of association between alcohol-related conditions and other causes of death, is shown. Yet, ischaemic and other forms of heart disease are not associated with the high incidence of alcohol-related conditions mentioned among causes of death. These conditions are frequent among Aboriginal deaths and occur together in many descriptions of deaths. However, what is being established here is that these conditions are not significantly associated with each other. Additionally, in this region circulatory system diseases would most probably retain their importance among Aboriginal people without the existence of alcohol-related conditions. This statement can also be supported by pointing to other measures of association previously employed on this data set which also show no relationship (Gray and Hogg 1989, 23-31) and to a large number of Aboriginal deaths in the study area from cardiac disease at quite young ages which are clearly not associated with alcohol consumption.

Table 14 Association[1] between ischaemic heart disease and other causes of death among Aborigines in western New South Wales, 1984-87.

Disease condition	Ischaemic heart disease Males[2]	Females[3]
Diabetes	-	0.27
Cancer	-0.26	-
Accidental and violent conditions	-0.45	-0.37

NOTE: See notes to Table 12.

Table 15 The measure of association[1] between sex-specific differences in pancreatic disorder and other causes of death among Aborigines in western New South Wales, 1984-87.

Disease condition	Pancreatic disorders Males[2]	Females[3]
Ischaemic heart disease	-	0.27
Other forms of heart disease	-	0.28
Diabetes	0.27	-
Alcohol-related conditions	0.22	-0.21
Chronic obstructive lung disease	-	-0.21
Accidental and violent conditions	-	-0.22
Other digestive diseases	0.26	-

NOTE: See notes to Table 12.

Table 16 The measure of association[1] between other respiratory system diseases and other causes of death among Aborigines in western New South Wales, 1984-87.

Disease condition	Other respiratory system diseases	
	Males[2]	Females[3]
Chronic obstructive lung disease	0.22	0.22
Accidental and violent conditions	-0.27	-
Infectious diseases	0.51	0.61
Elderly/senility	0.21	-

NOTE: See notes to Table 12.

In Table 14, the association between ischaemic heart disease and other causes of death is examined for both males and females. Principally, ischaemic heart disease is not positively associated with other causes of death in the data set, except among females where ischaemic heart disease is moderately associated with diabetes.

Table 15 displays the association between sex-specific differences in pancreatic disorders and other causes of death. Among males other pancreatic disorders are moderately associated with diabetes, alcohol-related conditions and other digestive diseases. Among females, diabetes is associated with various forms of heart disease, and not associated with alcohol-related conditions, chronic obstructive lung disease, and accidental and violent conditions. Although these relationships are relatively weak they show a strikingly sex-specific patterning.

In Table 16 the association between other respiratory system diseases and other disease conditions is set out. For both sexes, there is a strong association with infectious diseases and moderate association with chronic obstructive lung disease. Among males other respiratory system diseases are also related to being elderly or senile and not dying from accidental or violent causes. These relationships suggest that deaths from other respiratory diseases often occur later in life and are often associated with a type of infection or chronic obstructive lung disease.

These results reveal significant differences between Aboriginal and New South Wales mortality rates, but how do Aborigines in this region compare with other ethnic groups in Australia? Are Aboriginal rates significantly higher or lower? In this case, a comparison can be made by comparing the standardised mortality ratios (a ratio of the observed to the expected number of deaths for persons aged 15 to 74 years if Australian age-specific mortality applied) of Aborigines with those born in Australia and overseas. As shown in Table 17 Aborigines have sex-specific standardised mortality ratios significantly higher than those exhibited by the Australian-born or any ethnic group. This table provides us with a striking reminder of how different Aboriginal mortality levels in this region are from those exhibited by other Australians.

Table 17 Standardised mortality ratios[1] for Aborigines in western New South Wales, 1984-87 and other ethnic groups in Australia by birthplace,[2] 1984-86.

	Males	Females
Australian-born	1.05	1.04
Western New South Wales Aborigines	4.10	4.05
Africa	0.80	0.85
America	0.84	0.78
Canada and USA	0.96	0.91
Other	0.59	0.62
Asia	0.68	0.72
Lebanon	0.58	0.69
Vietnam	0.46	0.71
Europe	0.91	0.90
Greece	0.56	0.67
Italy	0.68	0.69
UK and Ireland	0.97	0.99
Yugoslavia	0.88	0.76
Other	1.00	0.92
Oceania	0.95	0.88
New Zealand	0.92	0.84
Other	1.10	1.08

NOTES: 1. In this case the standard mortality ratio is the ratio of the observed to the expected number of deaths for persons aged 15 to 74 years if Australian age-specific mortality applied.
2. The standardised mortality ratios shown in this table for ethnic groups other than Aboriginal Australians are based on ratios shown in Table 2.4 of the Australian Bureau of Statistics publication *Overseas Born Australians 1988*.

Conclusions

Results from this study show significant differences in mortality levels and associations between causes of death. At a regional level, Aboriginal mortality has been shown to be significantly different from the New South Wales regime. Mortality estimates drawn from this level show that: the relative risk of death for males and females is at least twice that of the New South Wales population; life expectancy at birth is about 53 years for males and 65 years for females; males and females can expect to live 15 to 20 years less than the total population; for most causes of death Aboriginal and New South Wales rates are significantly different; and deaths from circulatory system diseases account for the greatest proportion of excess risk between Aboriginal and New South Wales cause-specific rates.

At a community level, significant differences have been found between mortality levels of a select sample of communities. Mortality levels in these communities fall into three groups, very high, moderately high, and low. Additionally, mortality has been shown to be lower in communities with higher levels of employment and smaller households.

Finally, at an individual level the association between various disease conditions has provided some insight into the relationship between Aboriginal lifestyle and mortality. In particular, circulatory system diseases have been shown not to be associated with alcohol-related conditions. This is important because this lack of association suggests that alcohol-related conditions are not a major cause of early onset heart disease. Other important associations include the link between diabetes and heart disease in females and the sex-specific patterning of associations between pancreatic disorders and other disease conditions.

The distinctiveness of Aboriginal mortality levels compared to those exhibited by other Australians is in part due to the social and environmental context in which Aboriginal people live. Historically, Aboriginal access to and relationship with the land and its resources have been drastically altered by changing economic and living conditions caused by white colonisation and settlement. This rapid transition from a nomadic existence to a sedentary one has had a profound effect on traditional cultural values and relationships. More specifically, after white settlement Aboriginal institutions were no longer created *in situ*, but evolved from a complex interrelationship with the larger non-Aboriginal society. These newly-created social institutions often altered the significance of traditional social roles within Aboriginal society. Finally, these changing social institutions and social roles had a profound effect on Aboriginal health.

In western New South Wales this effect on Aboriginal health can be seen in the high level of mortality discovered by this research exercise. These high levels of mortality have reduced the integrity of Aboriginal society by affecting the security of family and household structures (Gray 1984). In this society, social and familial institutions often reflect the society's great concern with allocation of social roles from the dead to surviving relations (Blauner 1966). In this sense a society can be seen as a role structure through which people pass at various stages of their life-cycles, being assigned or claiming for themselves various social roles. Social roles which an individual may acquire influence the extent of exposure to particular risks of injury and the way in which people react to physical illness. Ultimately, these differences in risks, attitudes and health care opportunities lead to different rates of health service use, illness and mortality (Verbrugge 1983).

This interpretative framework is just one way of explaining the mortality patterns of Australian Aboriginal people. However, in the future such frameworks will become increasingly important, because they will allow researchers to make broad-based inferences without overlooking demographic or institutional anomalies which occur at a regional, community or individual level. Taking this framework into account, future research efforts should be directed at examining institutional relationships and demographic regimes at a national, regional, community and individual level; and comparing institutional relationships and demographic trends of Aborigines with other indigenous groups outside Australia.

Current research in these areas does exist. In Australia, many studies have explored the link between institutional relationships and demographic regimes. One of the more comprehensive studies was the Aboriginal Family Demography study undertaken by Gray (1989b) on the north coast of New South Wales. This study had three aims: to investigate the relationship between Aboriginal family structure and household occupancy and generate a meaningful typology of household and family types; to examine the economic significance of family and household structure and links with demographic outcomes in the areas of family formation, births, deaths and health status; and to investigate the extent to which government programs in areas such as health services, housing and employment addressed micro-economic and

demographic realities (Gray 1989b, 17). Overall, the project found familial and household relationships and structures to be important to the understanding of health and social disadvantage within Aboriginal society (Gray 1989b, 29).

Other research efforts, not all of them demographic, have explored and compared the institutional relationships of Aboriginal Australians with other indigenous groups around the world. Certainly, where institutional relationships between aboriginal peoples and non-aboriginal societies have followed similar paths, as for example in Australia, Canada, New Zealand and the United States, comparative frameworks have been appropriate and beneficial to researchers. For example, the work by the anthropologist Weaver (1985) compares the representativeness of Indian and Aboriginal political organisations in Australia and Canada. Also, research by historians like Bienvenue (1983), Fisher (1980), and Jacobs (1971) examines the impact of European settlement on indigenous peoples in Australia, Canada, New Zealand and the United States. However, from a demographic perspective, research based on this type of comparative framework has only begun recently. There is the research review article by Johansson (1982) in which she attempts to integrate a selective bibliography of research papers into a demographic history of indigenous peoples in North America since contact. Other research efforts have not been so historically comprehensive. Kenen (1987) notes the similarities in the recent morbidity and mortality patterns of Australian Aborigines and Native Americans and Gray (1989a, 3) briefly discusses the similarities between recent Australian Aboriginal and Canadian Indian fertility and mortality regimes. These types of comparative studies in demography, as Romaniuc (1987, 86) points out, are important because they allow researchers to gain constructive insights into the changes in fertility and mortality regimes experienced by Australian Aborigines, New Zealand Maoris, and North American Indians and Inuit. In the long-run both research areas will probably play an important role in the understanding of how the relationships between Aboriginal institutions and non-Aboriginal society influence Aboriginal demographic regimes.

NOTE

1. In this report Aboriginal crude and cause-specific death rates have been directly standardised to the New South Wales age structure. These rates will therefore differ in magnitude from the indirectly standardised rates previously published in the report to the New South Wales Department of Health (Gray and Hogg 1989).

DISCUSSION

Dr A Gray I think that what is very obvious from the presentations this morning is related to what Bob Douglas asked before: are these mortality patterns similar in different parts of Australia? The answer, that they are extremely similar, really screams out from the presentations that we have had this morning. We see this common pattern of high levels of mortality in middle adulthood, high levels of circulatory system disease, diabetes and heart disease.

There was another theme which came through very strongly today and that was the theme of difficulties with the data collections that we have to examine Aboriginal mortality in Australia, and the lack of official statistics which has allowed this situation to continue for so long. The occasional studies which have been done in different parts of the country are published generally by State governments and then not really noticed at a national level. The awareness of this pattern of Aboriginal mortality and these extremely high relative risks of mortality in middle adulthood are not addressed by governments.

There was another minor theme to which I would like to draw your attention. It was raised by Ceilia Brown and by Robert Hogg. It is the differences in mortality levels in different types of communities and some of the things that are connected with those differences. I think that it is very important for us to place Aboriginal mortality within this context of the social institutions which operate in Aboriginal communities and which operate in interactions between Aboriginal families and households and general Australian society. Now there were three substantial questions that were raised. The first one was Aileen Plant's question to Noor Khalidi about the comparability of his denominator data in concluding that Aboriginal life expectation was actually increasing in Central Australia. Would you like to comment on that?

Mr N Khalidi The population data which I have used for 1976 comprises eight Aboriginal communities, including Alice Springs, and I can say that they were fairly good data. By that I mean that in comparisons with Alice Springs data in various censuses, 1981 and 1986, I got a fairly consistent set of data. For 1981 and 1986 the population comprises 12 communities and they are large enough communities, more than 100 in population, so to my judgement they were not under-enumerated to an extent which distorts the data. There were fluctuations from community to community, but on the aggregate, they were a fairly good set of data.

Dr A Plant What methodology was used in coding the causes of death to determine what contributions different things have made to the increase of life expectancy?

Mr N Khalidi As I said in my presentation I was amazed by the details of the causes of death noted on the death certificates. At least 70 per cent of the death certificates were quite extensively recorded. By that I mean it was recorded exactly as was instructed by the ICD-9. I also had some data from the Department of Health, so I coded all of the causes reported. It took at least three months to do so. Then, I constructed the underlying cause of death. For instance, if there was one cause of death reported I took

that because it was the only one available. If there were more than one, I went through the definition as specified by the WHO and picked up the one which represents the underlying cause of death. So I made sure that the coding was as accurate as possible with the decimal points showing in the classification of cause of death. I have them in detail if anybody is interested.

Dr A Plant One of the other things that concerned me was that over the time period there has been a fall in mortality, probably even more marked in Central Australia than the rest of the Northern Territory, and yet the male infant mortality contributed a negative effect in improvement of life expectancy. Have you got any understanding of why that might be so?

Mr N Khalidi In 1983 according to my life table, the Aboriginal male infant mortality rate was about 32 to 35 and for Aboriginal females it was about 29, and I do not remember the figure for 1975. I cannot give you a definite answer for that, but it might have something to do with fluctuations in small numbers.

Dr A Plant One last thing. Decrease in mortality from circulatory disease contributed quite a lot of your improvement in life expectancy. Now that to me seems to be at variance when you look at Moody's data, earlier than yours, which showed a very small proportion of deaths due to circulatory disease and compared to my own data, that showed I think about roughly 30 per cent of deaths from circulatory disease. You would anticipate that what the Northern Territory is actually going through is a transition phase and in fact probably what would happen is over the next 10 years that the proportion of deaths due to circulatory disease would increase till it reaches the same levels reported by Len Smith, Alan Gray and Neil Thomson, getting up to around 50 per cent of deaths. It just doesn't seem to add up. I would think deaths from circulatory disease are increasing, and yet you found a decrease. Do you have any feeling of where that decrease has occurred within the category of circulatory disease?

Mr N Khalidi We should be aware that I am talking about a rate, not the number. You might have an increase in the number which does not necessarily mean an increase in the rate. What I am talking is about the rate of death from circulatory system which is obviously decreasing. I do not see a reason why I should doubt the data, because I made sure that the coding is consistent. I did it myself from 1975 to 1986, and I did not rely on data supplied by the Northern Territory Department of Health because I found some cases of very inaccurate coding. I checked the same events coded in Darwin with the coding I did myself. Because they were not matching, I checked with the manuals, I thought I might have overlooked something there. I see no reason to doubt the result I obtained.

Dr A Gray I would like to comment. If you look carefully at Noor [Khalidi's] data you will see that actually in the 30 to 49 age group there is a negative influence on expectation of life for females, and it is about zero for males. The improvement appears to be at younger ages. It is possible that it is rheumatic heart disease that has

decreased. When you raised the question of Moody's data, were you referring to the south coast of New South Wales?

Dr A Plant No. Northern Territory.

Dr A Gray Right, I am not familiar with it.

Dr N Thomson In Noor [Khalidi's] calculations the major improvement actually was in the age group 50 to 69. This is not explicable by rheumatic heart disease deaths, which are likely, if they are after the initial young years, to be in the 40s rather than higher. But I think the other thing is that the numbers are very, very small. If you look at the detailed numbers, there is only one for age group 15 to 29, which is one of the groups he looked at. There is only a total of 20 deaths in 1975 to 1977 and in 1984 to 1986 the total is 15 deaths. You are talking about a small number of deaths there, and I think one of the problems we are falling into is that you can have fluctuations just because of the very small numbers. Making general conclusions, I think, is a bit risky.

Mr G Briscoe One of the problems that I wanted to raise was the patterns of consistency about the loss of Aboriginal women at their most productive period of life. There are probably a number of reasons. My concern is that this is the factor that Aboriginal society is not conscious about. It is certainly conscious and has been made conscious about deaths in custody over the past five years, and yet there is no consciousness of the level of deaths of Aboriginal women at their most productive phase of life.

You hear this comparison made all the time, that if white babies were dying at the same rate as Aboriginal babies then there would be an uproar. If white women would die at the same rate as Aboriginal women at that particular age area, there would be more than a commotion about it. Bearing in mind these other issues like deaths in custody, these other important issues tend to get a back seat.

I don't want to denigrate deaths in custody. Those people who are responsible for legal services have done their job, they have highlighted deaths in custody. Now it should be those people who are conscious about statistics, those people who are conscious about the need for gathering data, who should start to raise their voice a bit more. I don't necessarily believe that problem then, like the ABS suggested, should be a problem of heaping the problem back on top of Aboriginal people themselves. I mean it is a question of costs, on the one hand, but it is a question of ignorance on the other.

You know Aboriginal society is burdened in many ways with being asked to express a consciousness about all sort of things. Well this one, I would say, is impossible because they simply do not know that their women are dying in their most productive years of life. I have never heard one Aborigine mention to me a concern about the fact that their women are dying at their most productive ages. Now I do not care what anyone says, they are ignorant about this fact, totally ignorant. It's a great problem and there is only one way of overcoming this. That is by bringing Aborigines together and raising their awareness about the importance of data gathering, and we know we can put all sorts of connotations on this problem.

I have this thing in my mind about calling anthropologists the CIA of Aboriginal affairs. Well perhaps surveillance is a problem that Aborigines have to cope with too. But it's still important intelligence gathering. Vital statistics are important and perhaps one recommendation coming from this conference could be that we bring either interested organisations or certainly interested Aborigines together and have conferences about these sorts of problems. Perhaps I am not expressing myself properly and I do not know where the resources are going to come from, but it might be one recommendation that the ABS can contribute some money. It is just not a matter of propaganda, it is a matter of systematic education, not just putting up posters in Aboriginal organisations. They are short-cuts, they are political solutions. What we want is epistemological solutions which are related to knowledge rather than political propaganda.

Dr A Gray There was a specific question that you asked earlier, as well, which was about the issue of surveys. Before we go on to that, could I address Ric Streatfield's question about the apparent conflict between health services provision and people returning to homelands. Maybe Ceilia Brown would like to comment on this as well after me. I think that there is a real conflict there. It is also there in that birthing centre in Congress in Alice Springs. There are problems with these experiments because the health service system is not flexible enough to deal with changes in the way which antenatal services can be delivered. Maybe there need to be two things done: one of them is very close monitoring of what actually happens in a community; and there also has to be more flexibility in delivery of services.

Ms C Brown You referred to conflict, that in fact a lot of the women that were having babies seemed to be at-risk because of various conditions such as diabetes and high blood pressure, so the nurses and midwives were concerned about not evacuating them to Alice Springs Hospital because that is where they would have the medical back-up, but the women have been deciding for themselves and disappearing just before the delivery date and having the babies on the land. So the staff had to face that situation and make some decisions about it by supporting the women in their decision to have the babies on the land and that just confirms what Alan has been saying.

If I may make just one other point about the homelands movement, which is that we are observing that cardiovascular disease is one of the main killers. I know a lot of us know about how x number of women went back to Derby from Melbourne and they went without medication for their diabetes for six weeks and everything was fine till they went back to Melbourne and they were back on medication. Once again, with the increased exercise and improved diet when Aboriginal people move back on their homelands, then the chances of reducing diabetes, blood pressure problems and obesity, which are the precipitating factors to cardiovascular disease, are reduced. So there is some hope of us expecting reduced mortality.

Dr R Streatfield I fully agree with that. We are actually following where Kerin [O'Dea] led the way. We have had several camps at Yarrabah, our biggest community, near Cairns. We have over 60 diabetics there and we have got the health team there, who have won the National Prize for Diabetes Education. That is a National Prize

competing with Hospitals and Diabetic Units right across Australia, and our Aboriginal Health Team at Yarrabah have won it twice. Each time they have won it they got six more glucose monitors and they have got most of their diabetics, middle to older age people, actually doing their own blood sugar monitoring at home.

People told us that Aboriginal people just could not do all this sort of thing. But they can and they run their own camps and we monitor their blood sugars and their blood pressures and their weights out on these camps, and they have a whale of a time collecting shellfish and eating traditional fashion. I have got graphs plotting their blood pressure steadily going down and their blood sugar levelling out. We are slowly getting the message across, but what we are doing at Yarrabah we have got to do right through the Cape.

With regards to the outstation movement—when I first came to Queensland from New Guinea, I won't say who it was in the health department in Queensland who asked me what I wanted to talk about. I didn't know much about Aboriginal health and I said: 'I want to talk about the homelands movement. Tell me about that.' He nearly went through the roof. He said: 'They will die like flies. There is no way that we can get services out to them out in these outstations.' But that is high tech medical people saying that sort of thing, and I really think we have got to get low-tech methods, decentralised methods of doing the monitoring, whether it be antenatal monitoring or anything else. You can do it, I know you can do it, it is done in New Guinea and it has been done in other places in the world. Why can't we do it here? We just have to have a tight system of referral so that if anything goes wrong way out at the outstation, there has got to be some way of getting that person to the most appropriate level of service. I fully support the positive aspects of the outstation movement, and since the change in government policies in Queensland now there is strong movement back to the outstations. There always has been in Aurukun because the State has not always had full control over Aurukun. There are now outstation movements at Lockhardt, at Yapoon and at Weipa South.

Mr D McDonald I wish to raise the issue of suicide and self-inflicted injuries. In the State presentations, very tiny numbers of suicides and self-inflicted injuries were reported, and we know of course that there is massive under-reporting of this across society at large. I wonder whether the people who have looked at the State data closely have any feeling about whether there is any differential in the under-reporting rate in this area between Aboriginal people and the society at large, in view of the political reticence in some Aboriginal health organisations to accept that intentional self-inflicted death or suicide actually occurs among a lot of Aboriginal people.

And the related issue to that is a massive and wonderful potential source of valuable mortality data, namely Coroner's Records. The Coroner's Records of Australia are in an absolute mess. They are not usable for anything except for legal enquiries. There is not a State in Australia that has computerised coronial records. I know this is something AIH has a great interest in, and other States may have given some thought to it, but in any summary record from this workshop I wonder if we could indeed note the potential value of coronial records for helping to understand morbidity, to understand mortality and develop appropriate interventions. So my

question to speakers from the three State presentations is whether there is any more knowledge about self-inflicted deaths?

Dr A Plant In five years of my study there were four male suicides and one female suicide. Both of those were substantially less than expected. Of course I don't have any idea of what the under-reporting is. As you said, you don't know the 'true' rate for the general community, you don't know it for the Aboriginal community either. It certainly was not a big feature in five years of Aboriginal deaths.

Dr D Hicks I cannot really comment on the suicide rates. They were a very small percentage of the total deaths and not the most important cause of deaths. I didn't look at them more closely than what was recorded on the death certificate or the Coroner's Report.

Ms C Brown There were no deaths recorded through suicide, but I think one of the points is that when there is a suicide or Coroner's Report filled out it does not have the question of race, so that is probably one of the sources of the problem.

Mr R Hogg Suicides made up a small proportion of deaths and there is only one case that was mentioned in the report that occurred outside of prison or outside of death in custody. That case was identified not by an informant but by the Registrar. The thing that I find very interesting, if you compare that to Canadian statistics, is that suicides among Native Indians are very high, especially during adolescence. I remember that just before I left Canada, there was a case at a reserve out at Edmonton where there was one person who killed herself, and then there were about three or four other cases just after that, so it became a national incident and it got a lot of reporting. When I came here I expected suicides to be much more prevalent and I started to talk to people about it, to informants, and they just really didn't think about it, it was something that occurred. So maybe it is something to do with different attitudes, but that is something that should be examined.

Dr N Thomson There is really more data about it. The most intensive study done about it has been done by Hunter in the Kimberley region. He has looked over a period of 1957 to 1988. He has identified over that period 16 Aboriginal suicides there, but only one of them occurred before the mid-1970s. That was a male in 1962, and only one other had occurred before 1983. So since 1983 suicides are identified in the Kimberley region. He has looked very closely at all the information that was recorded there. More importantly than that too is that all except possibly three, he doesn't give full details unfortunately in his paper, but 11 out of the 14 deaths for which the residence was identified were in Broome Shire. It was actually a very localised thing even within the Kimberley region. He calculated the rate for 1982 and 1986 of deaths in the Kimberley region, that is including Broome of course which is about one-quarter of the region's population, and found it to be about 31 per 100,000 compared to an overall Western Australian rate for males of 20 per 100,000. So it is one and a half times as high. This was for the Kimberley. If you look at the population in the Broome Shire you would really get a lot higher rate. The other thing, I guess, is indirect evidence in relation to

the under-reporting of suicides. I dug up a reference from the National Aboriginal Mental Health Association's Conference, and I think Joe Reser, who has done a lot of work on the subject, is saying that it is a very much under-reported phenomenon but it is also increasing in frequency in Queensland.

Dr D Hicks I didn't have a question so much as a comment. That there is no such thing as a 'right' mortality rate and that a mortality rate will vary from community to community or racial group to racial group. To take the extreme example, I would anticipate that a properly functioning homelands movement may have a maternal mortality rate, from post-partum haemorrhage or something. However, if it is a true homelands movement, it won't have a motor vehicle accident mortality rate. If it is in New Guinea, I understand the motor vehicle accident mortality rate relates to the driver who gets stoned rather than to the person who is hit by the vehicle. So there are different mortality rates in different communities, and I worry about this particularly when we have people from outside Australia who come and look at the mortality rate among Aborigines and say that it is terribly bad. I think mortality rates do vary from community to community and we should not use our baselines as always the right measure of mortality rate. Having said all that, I think it is just something we should keep in mind. There are a lot of things in the Aboriginal mortality figures which are much worse than we would like and we do need to develop some strategies to help to change those.

Prof R Douglas I think I have the distinction of being the least expert and the least knowledgeable about Aboriginal mortality in this room. There is a certain privilege that this group has got at the moment, that it is the first expert group that has come together to look at Aboriginal mortality across the entire Australian continent in 16 years.

I am concerned that we are identifying issues and identifying broad themes, but we are not crystallising in a way that will do more than simply contribute to scholarly knowledge. This follows on from what Gordon Briscoe was saying. There are certain things that are starting to emerge from this analysis that are not known outside this room. How are we going to translate that into both public information for the Aboriginal community, and into policy thinking within the various avenues that can make use of it? Because there will not be another opportunity to do precisely what we can do. What I am really asking is: how can we ensure that these things such as the possible domestic violence theme that is emerging in the reproductive age group for Aboriginal women, such as the consistency and inconsistency that seems to occur across the various State collections, such as the need for clearer action by the ABS, how can we ensure that these things get to become public knowledge? I am throwing up as a suggestion that as these things become consensus concerns within this group, that somebody writes them up on the board so that at the end of the session tomorrow we might have 10 or 15 statements, that we believe should be made clear to policy-makers. I am concerned that we are one day into the workshop, we have got another day to go, that we ensure that this becomes a productive exercise both in terms of future research agenda and in terms of where we are now.

Dr A Gray I think that during the lunch break we will try to draw up some of those on the board, both from yesterday afternoon's session and this morning's session and then maybe immediately after lunch we can have a brief discussion about putting those issues in some kind of order. It is of concern to me, this being a closed workshop, that we did not really intend to issue any kind of communiqué or press statement at the end. But I think that we can reach some kind of broad agreement which we can eventually include in the report of the workshop, and with the consent of everybody here, that we can forward to appropriate authorities.

Mr R Hogg To get to community level takes much more action than merely issuing a press communiqué at the end of this workshop.

Dr J Stuart I would like to ask Robert Hogg a question about his infant mortality rates in western New South Wales. He states that, apart from congenital disease, the greatest proportion of deaths is from sudden infant death syndrome, and it is 23 per cent of infant deaths. This is the first time that I have heard sudden infant death syndrome mentioned in regard to Aboriginal death rates and I have personally never been involved with a case. My question is whether it is a real diagnosis or is it a rag bag when people did not really know why the child died? Would you like to comment?

Mr R Hogg There are more details on that analysis in the report that Alan and I wrote but sudden death syndrome is really a category more than a cause of death, and it does not refer to one type of death. If you look at the total death records that we collected, we collected 394 for the region of which we only used 310 in this paper and also the other paper. If you include all of them there are even more sudden infant death syndrome cases, especially for male infants.

Dr J Stuart Did you look at post-mortem evidence?

Mr R Hogg I think there was none. We do not have post-mortem information. I have been told that the deceased in these cases usually go down to one of the hospitals to get examined in Sydney. There was no such thing in the community that I was in.

Dr R Streatfield I have forgotten exactly what Don Hicks said, but it was something about having to put up with more maternal mortality on the outstations. What really changed thinking in the health department in Queensland was a trip by Ken Donald, Deputy Director General of Health, around the Aboriginal communities in 1987 talking about AIDS and the threat of AIDS to the communities. I was with him on some of those trips and visits. He talked about AIDS for about an hour and a half or so and tried to impress the importance of AIDS on the communities and the threat it represented to each community he went to. Several people came up to him afterwards and said they did not know much about AIDS but what they wanted was to have their babies on their communities. Finally what we had been saying for years got to the hierarchy of the health department.

They said they were willing to put up with maternal deaths to be able to have their babies in a traditional fashion on their communities, in North Queensland.

It was introduced in 1974, I think, well before I got there, that every woman, black or white regardless, was to be flown into Cairns Base Hospital, to the nearest obstetrician to have her baby. She had no choice and that is why we have got all this data so accurately. That has been carried out and the maternal mortality rate for Far North Queensland has plummeted right down to zero. If the communities are willing, we will have to put up with the higher mortality rates to allow them to have their choice.

The other thing we were talking about was the importance of rheumatic fever in causing cardiovascular disease and deaths in younger to middle age group people in Central Australia. We have had a rheumatic fever prevention program going in Yarrabah for three years. We have had no cases where they had 139 times the rate of the nation in Yarrabah before we started. It is very easily done and it is very successful.

Dr K Streatfield There have been considerable variations in how much depth or how much massaging, to use Dan Black's words, of the limited numbers on which people are doing their analysis. Noor Khalidi's analysis, for example, of contributions to increase or change in life expectation is a good example of pushing fairly small numbers quite a long way. My general feeling is slight unease about a large number of people working in different States using fairly small numbers, quite often for single years only, and producing patterns. You can always produce a pattern but it is hard to know quite how much that fluctuates from year to year unless you have group data or continuous data. So I just put in a plug for regular data collection or for us, as Bob Douglas suggested we do tomorrow sometime, trying to work out what are the minimum requirements for data numbers and so on. And what analytical procedures might be gone through. And then that would be applied to each State every year by some unit that will evolve from all this. That leads on to the subsequent steps that Bob Douglas mentioned, of then putting this information in some kind of form to which the public has access, particularly Aboriginal communities. But my impression of all this is a lot of somewhat isolated activity across the States producing analysis in which we can have varying degrees of confidence.

Mr G Briscoe One of the things that we should be careful to point out is that we really need more data about the homelands movements, as you call them. I call them recolonisation. With a lot of the people actually moving back to the so-called homeland, people are moving to new areas: they were not born there. They are really remigrating. That puts the thing in a different light. In 1788, Britain sent people out here to colonise Australia for the first time. I do not want to talk too deeply about what was happening, but just the general situation. As a result of that it is recorded that the people who came here had health problems different from the ones that they suffered from in England. In fact their health improved in many ways, the people that came and settled Botany Bay, or took over, whatever you like to say.

So the homeland movement is a reaction against centralisation of Aboriginal communities in the 1940s, 1950s, 1960s or earlier. I think we should not necessarily think about land rights so much as land reform because those problems are going to reoccur. Increasing populations on outstations, if you like, which is not necessarily an Aboriginal idea. I mean the pastoralists were the first ones to invent

Aboriginal people living away from homesteads, because if they lived near the homesteads that meant an increased cost and a reduction of the profits of the cattle industry. So we do not want to be hoodwinked into thinking that somehow land rights is some inherent idea in Aboriginal minds. It is not.

Protectionism has been part and parcel of the way whites have treated Aborigines. Since whites have been so influential in the way Aboriginal people think, it is a natural step to examine past white attitudes to determine what Aborigines are going to think about next. The questions of land and protection of Aborigines have been a focal and inherent factor in the way whites have treated Aboriginal people. The fact that today these are interconnected does not necessarily mean that we should then link that to better health and say that health is improving. But what about these other factors, such as maternal deaths? Are we, by definition, purely because we are interested in the improvement of cardiovascular problems, are we then going to be able to accept higher death rates in certain areas? That might be a choice that Aborigines themselves are going to have to make.

Dr G Durling Just brief information. The Northern Territory has embarked on a project of health indicators what we are calling Mark II of the Northern Territory, which will cover a 10 year period 1979 through 1988. It will include morbidity data, mortality data, perinatal data, and probably some social indicators also, and a few other things which have not been decided yet. Publication is geared for probably a year and a half from now, roughly speaking.

Section Three

National and Comparative Studies

13 CAUSES OF ABORIGINAL MORTALITY

Morteza Honari

Data used here to illustrate the patterns of Aboriginal mortality are the combined data for the Queensland communities and Western Australia, South Australia and the Northern Territory in 1985. The Queensland Department of Health provided data for the Queensland communities. The Health Department of Western Australia provided the information on Western Australian Aboriginal deaths; the South Australian data was provided by the AHO of South Australia; and the Northern Territory Department of Health and Community Services supplied the information for the Northern Territory. The combined Aboriginal population in which the deaths occurred was 95,771, comprising 43 per cent of the estimated total Aboriginal population in 1985. Table 1 shows the study population by region and sex. These estimates are based on results from the 1986 Census of Population and Housing.

Table 1 Aboriginal population by region and sex, 1985.

	Males	Females	Total
Queensland communities	5,466	5,187	10,653
Western Australia	18,492	18,556	37,048
South Australia	6,820	7,192	14,012
Northern Territory	16,580	17,478	34,058
Sub-total	47,358	48,413	95,771
Total Aboriginal population	110,446	112,737	223,183

Methods

Deaths have been grouped according to the ICD-9 class headings. Using total Australian age and cause-specific death rates, indirect standardisation was applied to calculate the expected numbers, and hence expected rates, of Aboriginal deaths for each cause. These observed and expected death rates form the basis of the main comparison between Aborigines and the total Australian population. Specific causes of death were compared also for a number of age groups: zero to four years, five to 14, 15 to 24, 25 to 44, 45 to 64 and 65 years and older. These comparisons were based on the age-specific rates for Aborigines and the total Australian population.

Results

Of the 47,358 Aboriginal males in 1985, 572 deaths occurred, which is 3.2 times the expected number. For the female population of 48,413, 395 deaths occurred, 3.4 times the expected number. Expected numbers, standardised mortality ratios and confidence intervals for the ratios are shown in Table 2. The leading cause of death for both sexes of the Aboriginal population was diseases of the circulatory system (ICD-9:

390-459). For males, the total of 157 deaths was 2.4 times the expected number. This number includes 76 deaths from ischaemic heart disease and 47 from other forms of heart disease. For females, the total of 118 deaths was 2.6 times the expected number. This consisted of 43 cases of ischaemic heart disease and 21 deaths from other forms of heart disease.

Table 2 Aboriginal standard mortality ratio compared with the total Australian population, 1985.

	Observed N	Expected N	SMR	95% confidence interval
Males	572	179.3	3.2	2.9-3.5
Females	396	115.7	3.4	3.1-3.8

Table 3 Male Aboriginal observed and expected deaths and proportion of excess risk, 1985.

Cause of death (ICD-9 code)	Observed N		Expected N	SMR	Excess risk
Infectious & parasitic diseases (001-139)	28	1.0	28.0	6.9	
Intestinal infectious (001-009)	7	0.1	63.1	-	
Tuberculosis (010-018)	11	0.2	71.0	-	
Neoplasms (140-239)	49	37.8	1.3	2.9	
Digestive system (150-159)	9	10.3	0.9	-	
Liver (155)	4	0.5	7.5	-	
Respiratory system (160-169)	20	10.7	1.9	-	
Endocrine & nutritional disorders (240-279)	13	3.0	4.3	2.5	
Diabetes mellitus (250)	9	1.9	4.6	-	
Blood & blood organ disorders (280-289)	1	0.5	2.0	0.1	
Mental disorders (290-319)	21	2.4	8.8	4.7	
Nervous system (320-389)	16	3.2	5.0	3.3	
Circulatory system disease (390-459)	157	65.9	2.4	23.2	
Rheumatic fever & heart disease (390-398)	7	0.4	17.5	-	
Hypertensive disease (401-405)	7	1.1	6.7	-	
Ischaemic heart disease (410-414)	76	41.8	1.8	-	
Other forms of heart disease (420-429)	39	6.8	5.8	-	
Cerebrovascular disease (438)	28	11.9	2.4	-	
Respiratory system disease (460-519)	77	13.4	5.7	16.2	
Pneumonia (480-486)	38	2.3	16.2	-	
Obstructive airway disease (490-496)	26	9.5	2.7	-	
Asthma (493)	3	1.2	2.6	-	
Digestive system disease (520-579)	18	5.3	3.4	3.2	
Liver disease (571)	12	2.3	5.3	-	
Genito-urinary system disease (580-629)	13	1.9	6.8	2.8	
Congenital anomalies (740-759)	14	5.3	2.6	2.2	
Perinatal conditions (760-779)	24	6.0	4.0	4.6	
Ill-defined symptoms (780-799)	18	4.2	4.3	3.5	
Sudden death (798)	9	3.9	2.3	-	
Injury and poisoning (E800-E999)	103	28.7	3.6	18.9	
Motor vehicle accidents (E810-819)	34	12.3	2.8	-	
Suicide (E950-959)	13	6.8	2.8	-	
Homicide (E960-969)	15	1.1	14.1	-	
Unknown	20	-	-	-	
All causes	572	179.3	3.2	100.0	

External causes of injury and poisoning (ICD-9: E800-E999) was the second most frequent cause of death for Aboriginal males and the third for females. Deaths caused by motor vehicle accidents, fire, drowning, poisoning and violence are included in this group. For males, the total of 103 deaths occurring in 1985 was 3.6 times the expected number. For females, the total of 46 deaths was 4.4 times the expected. Within this category, 34 males and 13 females died from motor vehicle traffic accidents, and 15 males and 18 females from homicide and injury purposely inflicted by other persons.

Diseases of the respiratory system (ICD-9: 460-519), including both acute and chronic conditions, comprised the third most frequent cause of death for Aboriginal males and the second for Aboriginal females. For males, the total of 77 deaths was 5.4 times the expected number. This comprised 39 cases of pneumonia, 29 cases of obstructive airway disease and three cases of asthma. For females, the total of 48 deaths was 7.3 times the expected. Female deaths in this group included 27 from pneumonia, 10 cases of obstructive airway diseases and four cases of asthma.

Table 4 Female Aboriginal observed and expected deaths and proportion of excess risk, 1985.

	Observed N	Expected N	SMR	Excess risk
Infectious and parasitic diseases (001-139)	16	0.9	17.8	5.5
Intestinal infectious diseases (001-009)	6	0.1	51.3	-
Tuberculosis (010-018)	4	0.1	39.7	-
Neoplasms (140-239)	38	27.4	1.4	3.8
Digestive system (150-159)	8	7.3	1.1	-
Liver (155)	3	0.2	15.0	-
Respiratory system (160-169)	4	2.9	1.4	-
Cervical cancer (180)	5	1.0	5.2	-
Endocrine & nutrition disorders (240-279)	23	2.7	8.5	7.2
Diabetes mellitus (250)	9	1.9	4.6	-
Mental disorders (290-319)	5	1.7	2.9	1.2
Nervous system (320-389)	12	2.3	5.2	3.5
Circulatory system disease (390-459)	118	45	2.6	26.0
Rheumatic fever & heart disease (390-398)	10	0.6	17.4	-
Hypertensive disease (401-405)	8	1.1	7.3	-
Ischaemic heart disease (410-414)	43	22.2	1.9	-
Other forms of heart disease (420-429)	21	5.5	3.8	-
Cerebrovascular disease (430-438)	26	12.7	2.0	-
Respiratory system disease (460-519)	48	6.6	7.3	14.8
Pneumonia (480-486)	27	1.7	15.5	-
Obstructive airway disease (490-496)	11	3.8	2.9	-
Asthma (493)	4	1.2	3.2	-
Digestive system disease (520-579)	27	3.5	7.7	8.4
Liver disease (571)	14	0.9	15.6	-
Genito-urinary system disease (580-629)	16	1.7	9.4	5.1
Pregnancy-related disease (630-676)	1	0.1	10.0	0.3
Skin & subcutaneous tissue (680-709)	1	0.1	10.0	0.3
Musculoskeletal system disease (710-739)	3	0.7	4.3	0.8
Congenital anomalies (740-759)	7	4.4	1.6	0.9
Perinatal conditions (760-779)	16	5.0	3.2	3.9
Ill-defined symptoms (780-799)	14	2.7	5.2	4.0
Sudden death (798)	7	2.4	2.9	-
Injury and poisoning (E800-E999)	46	10.4	4.4	12.7
Motor vehicle accidents (E810-819)	13	4.8	2.7	-
Homicide (E960-969)	18	0.7	25.5	-
Unknown	5	-	-	-
All causes	396	115.7	3.4	100.0

There were 46 deaths coded under the heading neoplasms among males and 38 among females (1.3 and 1.4 times the expected numbers). The main types of cancer among males were of the respiratory system (20 cases), the digestive system (nine cases) and the liver (four cases). The most frequent cancer deaths in females were of the digestive system (eight cases), the cervix (five cases), the respiratory system (four cases) and the liver (three cases).

For each group of causes for which a significant number of Aboriginal deaths occurred, the observed rate exceeded the rate expected from total Australian rates, as illustrated in Tables 3 and 4 and Figure 1. Even the number of deaths caused by neoplasms, previously thought to occur less frequently among Aborigines than among the total population, was slightly higher than the number expected.

Figure 1 Observed and expected rates for causes of male Aboriginal deaths, 1985.

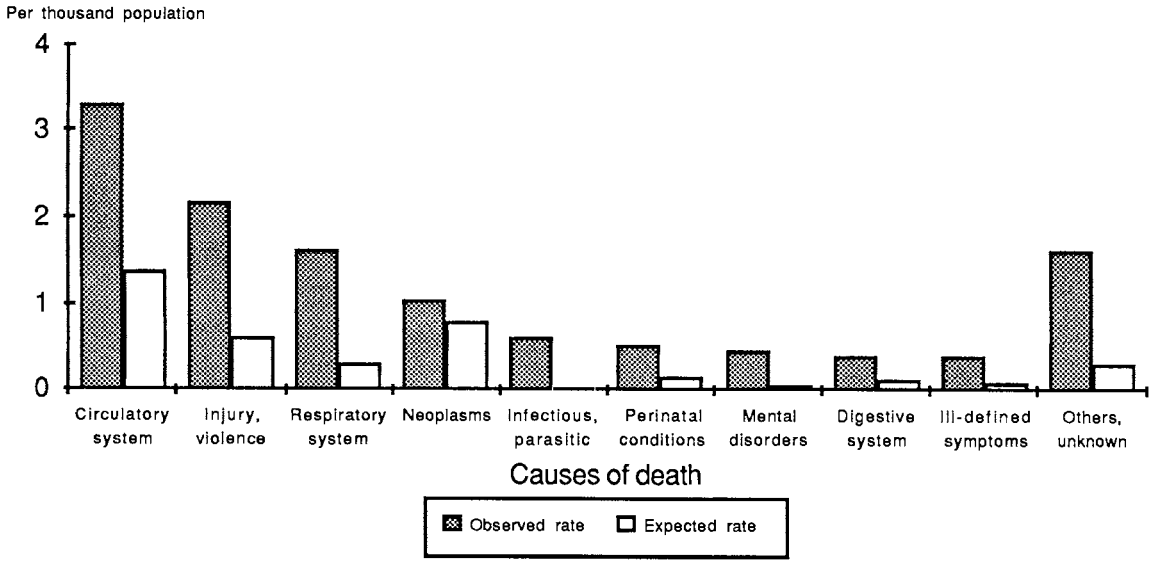
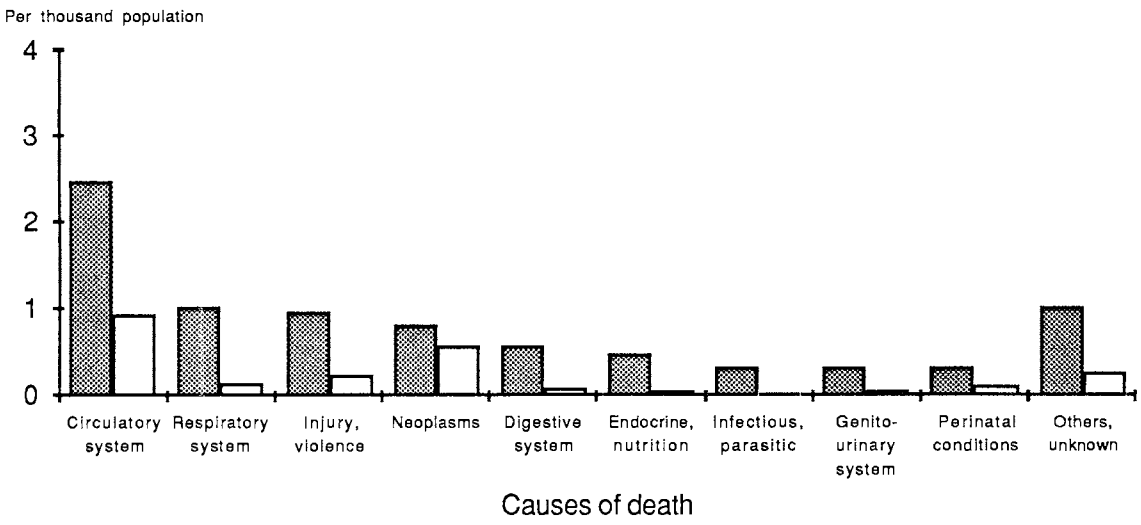


Figure 2 Observed and expected rates for causes of female Aboriginal deaths, 1985.



NOTE: The observed numbers in Figures 1 and 2 represent the combined data for Aborigines of the Queensland communities, Western Australia, South Australia and the Northern Territory. The expected numbers are the mortality rates for the total Australian population applied to the Aboriginal population.

SOURCES: Data provided to the AIH by the Queensland Department of Health, the Health Department of Western Australia, the Aboriginal Health Organisation of South Australia, the Northern Territory Department of Health and Community Services; Australian Bureau of Statistics, *Deaths Australia 1985*, Catalogue No 3302.0.

Some caution needs to be exercised in considering the results presented here, as some are based on small numbers of deaths. For Aborigines in age group zero to four years, there were 60 male deaths and 50 female deaths. For all specific causes of death, the Aboriginal rates exceeded those of the total population. For Aborigines, the ranking of the leading causes is basically the same as that of the total population, certain conditions originating in the perinatal period, congenital anomalies, symptoms, signs and ill-defined conditions, external causes of injury and poisoning and diseases of the respiratory system. Out of 24 deaths in Aboriginal males (4.0 times the expected number) and 16 in females (3.2 times the expected number) relating to conditions originating in the perinatal period, nine and seven cases respectively were from sudden infant death syndrome. The notable feature is the number of Aboriginal deaths attributed to infectious and parasitic diseases, including gastroenteritis. Compared with the five male and six female deaths occurring among Aborigines, there were a total of only 21 male deaths and 20 female deaths for the total Australian population, which includes Aborigines in other States. Given the size of the Aboriginal population in the Northern Territory, Western Australia, South Australia and Queensland communities relative to the total Australian population, this represents massively disproportionate risk.

Figure 3 Components of excess mortality risk of male Aborigines, 1985.

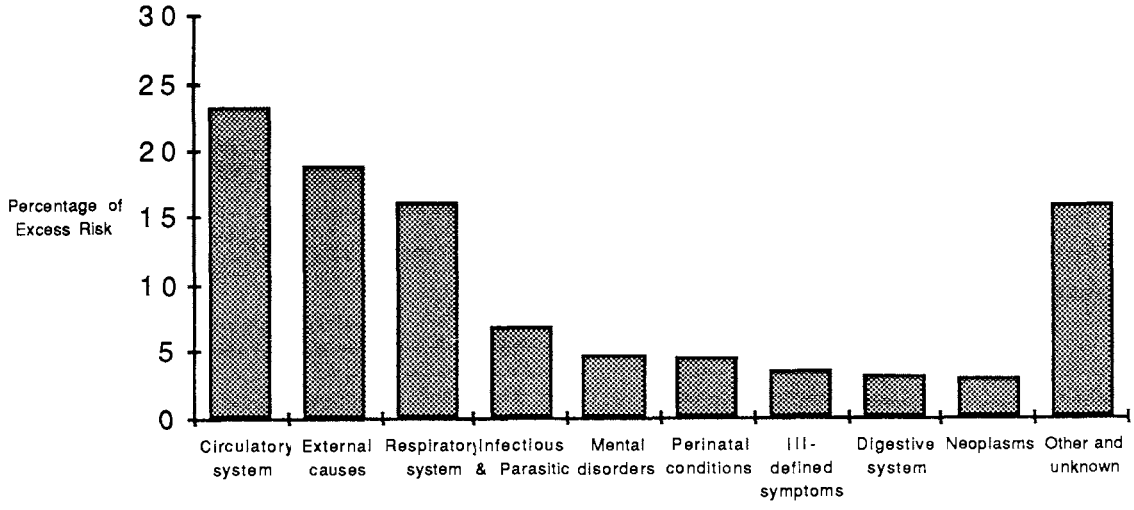
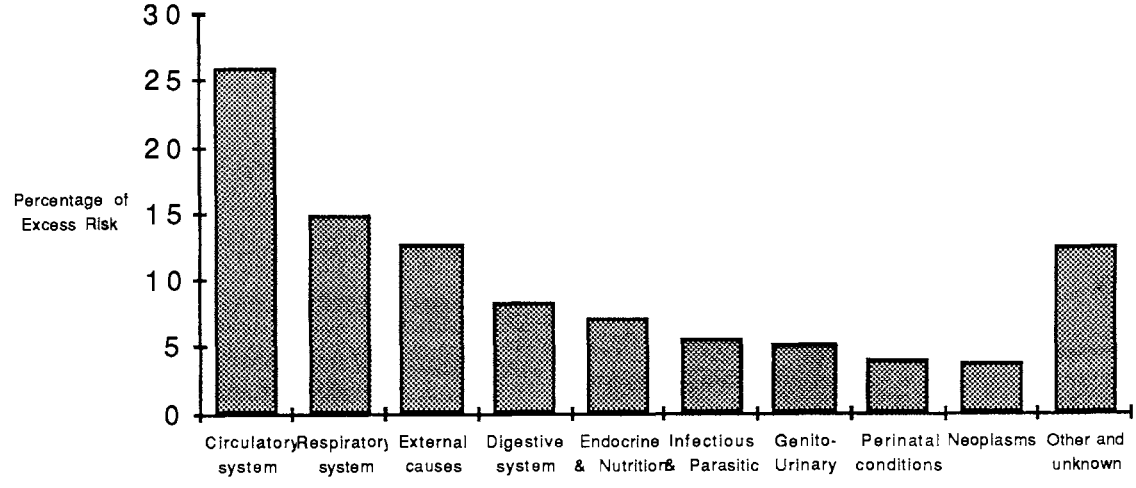


Figure 4 Components of excess mortality risk for female Aborigines, 1985.



SOURCES: Data provided to the AIH by the Queensland Department of Health, the Health Department of Western Australia, the Aboriginal Health Organisation of South Australia, the Northern Territory Department of Health and Community Services; Australian Bureau of Statistics, *Deaths Australia 1985*, Catalogue No. 3302.0.

For Aborigines aged five to 14 years, there were 17 male deaths and five female deaths. From the 12,500 Aboriginal males (less than one per cent of the total Australian male population in this age group) came 4.5 per cent of all deaths in Australia. From the 11,850 Aboriginal females (less than one per cent of the total Australian female population in this age group) came 2.3 per cent of all deaths. Although based on quite small numbers, the most significant cause of death was the ICD-9 group—external causes of injury and poisoning—responsible for 10 male and two female deaths.

For Aborigines in age group 15 to 24 years, there were 36 male deaths and 31 female deaths. The ICD-9 group—external causes of injury and poisoning—was a significant cause, being responsible for 23 male and 19 female deaths. For males, four deaths attributed to 'mental disorders' (ICD-9: 290-319), were due to alcohol- and drug-related causes.

For Aborigines aged 25 to 44 years, there were 146 male deaths and 55 female deaths. For each of the leading specific causes of death, Aboriginal rates exceeded those of the total Australian population. While the ICD-9 group—external causes of injury and poisoning—was responsible for a substantial number of deaths (43 males and 12 females), the group—diseases of the circulatory system—was more notable in its relativity to the total Australian population. For Aboriginal males, the number of deaths attributed to causes within this group (38), was almost 14 times the number expected. For Aboriginal females, the 20 deaths reported were almost 15 times the number expected.

In age group 45 to 64 years there were 170 male Aboriginal deaths and 134 female deaths. For each of the leading specific causes of death, Aboriginal rates exceeded those of the total Australian population. As for the total population, the ICD-9 group—diseases of the circulatory system—was the leading cause of death for Aborigines in this age group. For Aboriginal males, the number of deaths attributed to causes within this group (54), was more than three times the number expected. For Aboriginal females, the number of deaths reported (48), was seven times the number expected. For Aboriginal males and females, the ICD-9 group—diseases of the respiratory system—was the second most frequent cause of death. The age-specific death rate for Aboriginal males, 7.1 deaths per 1,000 population, was almost 12 times that of the total Australian male population. For Aboriginal females, the age-specific rate of 4.1 per 1,000 was almost 14 times that of the total Australian female population.

For Aborigines aged 65 years and over there were 143 male deaths and 120 female deaths. For all except one of the leading specific causes of death, Aboriginal rates exceeded those of the total Australian population. The one exception, and indeed the only exception for any leading specific cause of death for any of the selected age groups, was the ICD-9 group—neoplasms—in the case of Aboriginal males. For this age group, the cause-specific death rates for diseases of the circulatory system were more similar to those of the total Australian population, being 1.3 times higher for males, and 1.1 times for females. The differences in cause-specific death rates for

diseases of the respiratory system were also less marked than for the younger age groups, 2.7 times higher than that of the total Australian population for males, and 2.9 times for females.

As well as comparing the reported and expected numbers and rates of deaths from specific causes, the analysis of cause of death among Aborigines needs to provide some measure of the impact on the population of the various causes of death. This involves an analysis of the proportions attributable to specific causes of the excess mortality experienced by Aborigines compared to the total Australian population, as shown in Figures 3 and 4.

Deaths attributed to the ICD-9 group—diseases of the circulatory system—contributed 23 per cent of the excess deaths reported for Aboriginal males, and 26 per cent of those reported for females. Almost 19 per cent of excess deaths reported for Aboriginal males were caused by the ICD-9 group—external causes of injury and poisoning—as were 13 per cent of excess deaths reported for Aboriginal females. The next most significant cause of excess Aboriginal mortality was the group—diseases of the respiratory system—contributing 16 per cent of excess male deaths and 15 per cent of excess female deaths. The group—infectious and parasitic diseases—contributed seven per cent of the excess deaths for Aboriginal males, and the group—diseases of the digestive system—more than eight per cent of excess deaths for Aboriginal females.

14 NATIONAL ESTIMATES OF ABORIGINAL MORTALITY

Alan Gray

During recent years, a number of studies of the age pattern and level of Aboriginal mortality based on actual details of Aboriginal deaths have been undertaken. Sufficiently good data have been available from New South Wales (Smith et al 1983; Gray and Hogg 1989), South Australia (Australian Institute of Health 1988), Western Australia (Hicks 1985b; Australian Institute of Health 1988) and the Northern Territory (Devanesen et al 1986; Plant 1988; Australian Institute of Health 1988) to permit construction of approximate life tables, although in most cases, and in some States much more than in others, the data have required considerable adjustment for suspected under-enumeration of deaths relative to enumeration of population.

There are many problems with the data. It is only since 1980, when Aboriginal origin was first included as a data item on official death notification forms in New South Wales, that information on Aboriginal deaths has been available from the official death registration system in any State. However, some other collections of data, none of them complete, had been made by the health authorities in some States before then. During the mid-1980s, after urging by the Commonwealth Government Task Force on Aboriginal Health Statistics, most States and both Territories have been moving towards identifying Aboriginal deaths in their official collections. Except those for the Northern Territory, all of the studies mentioned above have been based either wholly or partly on these new data sources. Most of the studies have found some gaps in the information, in the form of deaths not identified as Aboriginal; most have also found difficulty with the population data available to use as denominators for rates.

There were also earlier studies of the levels and age distribution of Aboriginal mortality, using data from other sources. The first was by Jones (1963), who used data from the Northern Territory in 1960 to estimate expectation of life at birth to be 50 years for both sexes. Smith (1975 and 1980) fitted a model life table to intercensal survival data for the period 1961 to 1971, and found Australia-wide mortality levels similar to those that Jones had found for the Northern Territory alone. Moodie (1973) measured age-specific mortality on the south coast of New South Wales during the late 1960s, Boundy (1977) produced life tables for the Kimberley region of Western Australia for 1974 to 1976, while an analysis of mortality in 1978-79 in New South Wales (New South Wales Health Commission 1979) again used model life table methods. It was only the accumulation of evidence that finally prompted the realisation that the age distribution of Aboriginal mortality resembled no model life table pattern.

Intercensal survival methods for estimation of mortality are based on looking at the proportion of people from an age group at one census who survive to the appropriate age group at the next census. Although Smith did manage to fit a model life table to intercensal survival data, indirect estimates of Aboriginal mortality

using intercensal survival methods have been notoriously difficult to derive, because of different levels of enumeration in successive censuses and also because of some characteristic patterns of age mis-statement in data for the Aboriginal population. Both of these data problems create serious difficulties for these methods. Smith was able to overcome the first because the 1961 and 1971 censuses were apparently not seriously different in their level of enumeration of the Aboriginal population, and the second difficulty was overcome by massive smoothing of the intercensal survival ratios using a model life table system. Neither problem can be handled as easily to produce estimates for the 1981-86 period; there is a very large difference in the levels of enumeration of the Aboriginal population in the 1981 and 1986 Censuses, and it is now known that standard model life tables are simply inappropriate to describe the pattern of Aboriginal mortality.

Fortunately, advances in technical demography in recent years permit a different approach to both problems. It has now been possible for some time to estimate the relative levels of enumeration in two censuses using what only need to be approximately accurate information on the pattern of mortality in the intercensal period (Preston and Hill 1980). For the second problem due to age mis-statement in the data, there is a new procedure (Luther and Retherford 1988) for producing consistent from inconsistent estimates which are theoretically related to each other by additive balancing equations. The Luther-Retherford procedure uses methods of functional analysis to locate the 'consistent', that is balancing, set of estimates closest to a preliminary set of estimates.[1]

Methods and results

The new sources of data available for a number of States show the pattern of Aboriginal mortality very well. Data from South Australia, Western Australia and the Northern Territory in 1985 (Australian Institute of Health 1988) and western New South Wales from 1984 to 1987 (Gray and Hogg 1989) were scaled and aggregated to produce death rates for what represents about 42 per cent of the total Aboriginal population. While data for the individual State components contained quite small numbers of deaths, the age pattern and level for each sex seemed to be very similar for each State. The patterns are shown in Figures 1 and 2.

While the patterns lack smoothness, due to the small numbers of deaths that were used in constructing the rates which they represent, the characteristic nature of Aboriginal mortality found in many different studies is replicated extremely well. This pattern is one where mortality rates are extremely high (relative to mortality in other populations) in the ages of middle adulthood, between 30 and 50 years. It would have been possible to smooth the logarithmic rates out to the straight line form implied by a Gompertz model, but not without running the risk of losing what might be a slight convexity in the male curve, and it was felt to be safer to leave the preliminary rates unadjusted in any way. The comparison in Figure 1 with mortality in the total Australian population shows this difference as the large vertical gap between the two lines for each panel, which is accentuated in the ages of middle adulthood, particularly for males.

These preliminary death rates were used to construct guesstimates of the numbers of Aboriginal deaths in each State by age and sex for the 1981 to 1986 intercensal period as if the 1981 and 1986 population enumerations were compatible and complete.[2] Age cohort data were then analysed using the Preston-Hill technique to estimate the difference in level of enumeration in the two censuses. This method uses some constructed measures which when plotted against each other produce a theoretically straight line with two main characteristics: the line's intercept with the vertical axis measures the relative completeness of the first census against the second census; and the line's slope divided by the intercept measures the relative completeness of the guesstimated number of deaths against the second census. This actually works well for the intercept parameter no matter how bad the preliminary estimates of the pattern of mortality are, but it works well for the slope-divided-by-intercept parameter only when the mortality guesstimates are extremely accurate. Figures 3 and 4 show the 'straight lines' obtained for New South Wales, including the Australian Capital Territory, for the 1981-86 period.

Figure 1 Age-specific death rates and logarithmic scale for Aboriginal males in South Australia, Western Australia, Northern Territory and western New South Wales, 1985, compared with total Australian mortality, 1981-86.

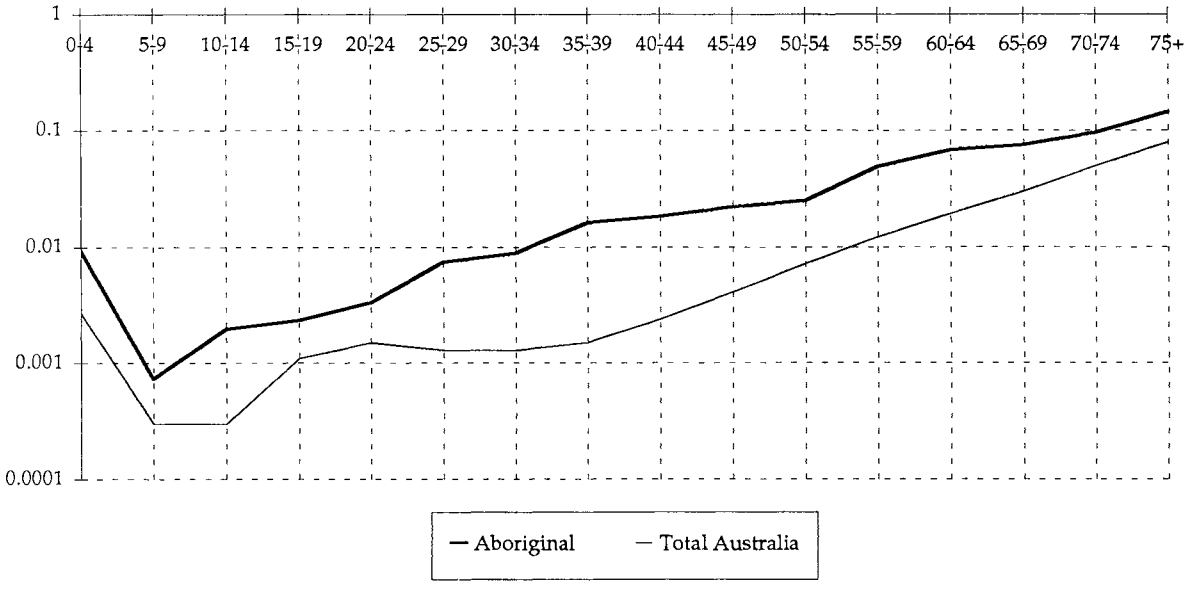


Figure 2 Age-specific death rates and logarithmic scale for Aboriginal females in South Australia, Western Australia, the Northern Territory and western New South Wales, 1985, compared with total Australian mortality, 1981-86.

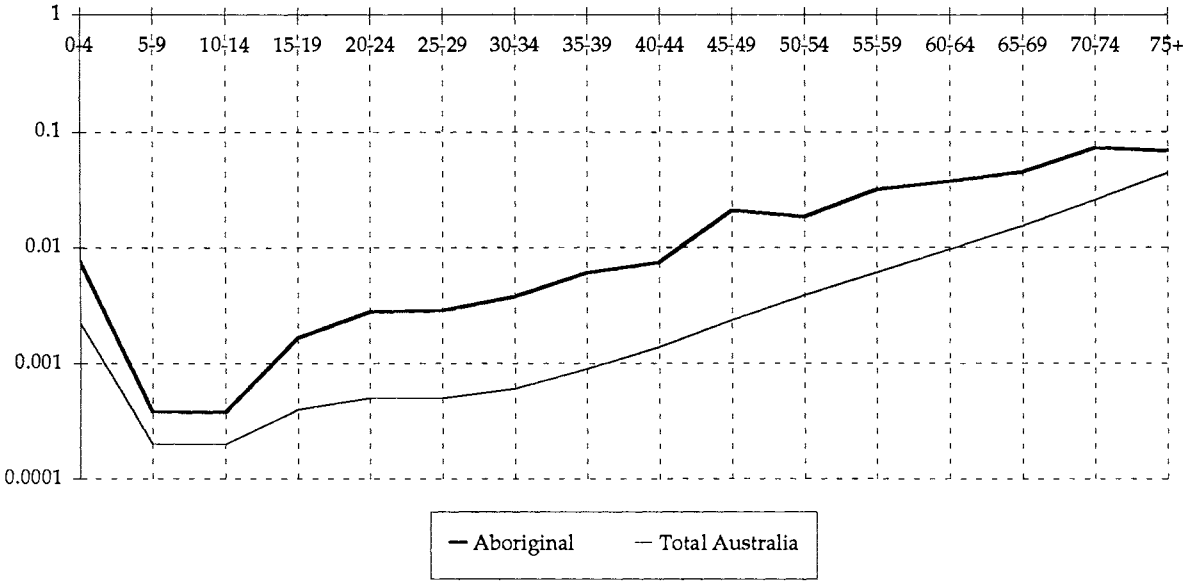


Figure 3 Preston-Hill intercensal analysis for Aboriginal males in New South Wales and the Australian Capital Territory, 1981-86.

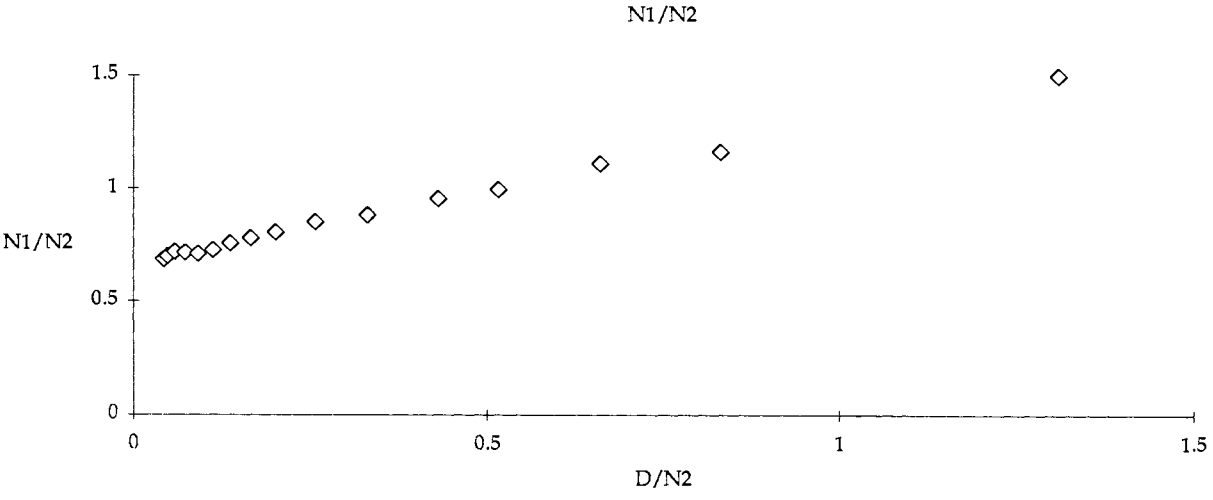
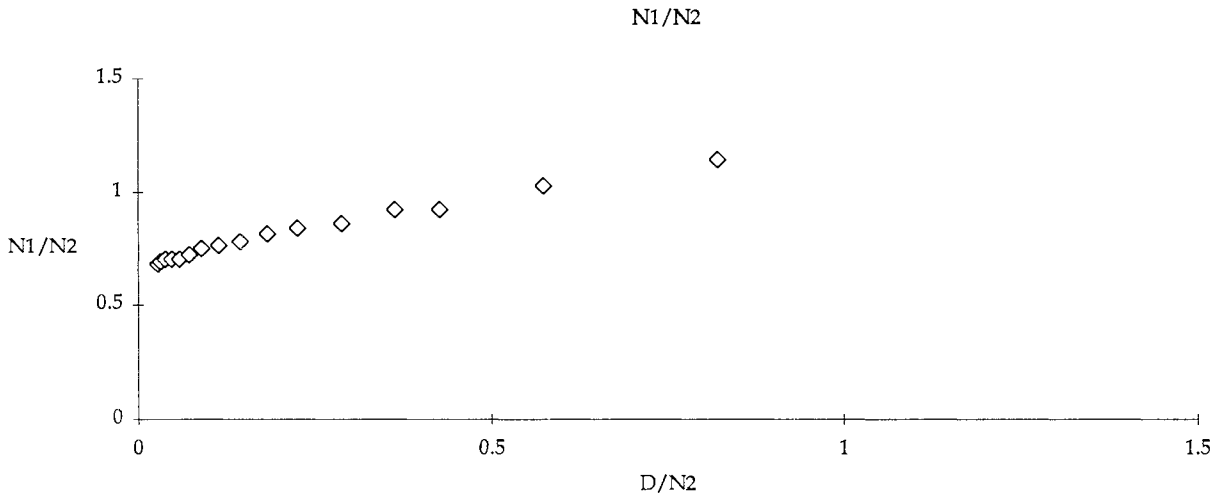


Figure 4 Preston-Hill intercensal analysis for Aboriginal females in New South Wales and the Australian Capital Territory, 1981-86.



The intercepts with the vertical axis in Figures 3 and 4 are at 0.67 for males and 0.68 for females, implying very substantial underenumeration in the 1981 Census compared with the 1986 Census. This is entirely expected, for the 1981 Census recorded far fewer Aboriginal people in the State than in 1986, far fewer than could possibly be accounted for by intercensal deaths or migration between States. The slopes divided by intercepts, at 0.90 for males and 0.81 for females, also show that the guesstimated number of intercensal deaths is slightly too low. This is also expected, because the guesstimate was calculated using an estimate of 1981 population size that was itself too low.

The theoretically straight lines in Figures 3 and 4 are also quite straight in practice, which means that the results can be used with some confidence. Similarly straight lines were generally obtained for data from the other States and Territories, although it was necessary to combine Victoria and Tasmania to obtain even a fair result, and the lines obtained for South Australia, based on small population size as in the case of Victoria and Tasmania, were also somewhat wobbly. The only States where there was fairly close agreement between the levels of enumeration in the two censuses (intercepts between 0.9 and 1.0) were Western Australia and the Northern Territory. The intercepts for the various States and both sexes are shown in Table 1. They represent preliminary estimates of the extent of under-enumeration in the 1981 Census compared with the 1986 Census. Note the fairly close agreement between estimates for the two sexes, which is something that ought to be expected even though the results were obtained independently for each sex.

Table 1 Estimated completeness of the 1981 Census against the 1986 Census for Aborigines by jurisdiction.

	Males	Females
New South Wales and ACT	0.67	0.68
Victoria and Tasmania	0.48	0.49
Queensland	0.82	0.83
South Australia	0.83	0.79
Western Australia	0.97	0.92
Northern Territory	0.90	0.93

NOTE: Completeness is defined as the proportion by which the 1981 Census results would need to be divided to make them comparable in level with the 1986 Census data for the Aboriginal population.

This first stage of the analytical procedure appears to overcome the problem of incompatible levels of census enumeration quite well. The second stage of analysis was to invoke the Luther-Retherford procedure to balance the estimates for population by age in 1981 and 1986 and deaths in the intercensal period, using the inverses of the preliminary factors in Table 1 as first estimates of correction factors for population data for the year 1981 and estimates of one for correction factors for all other quantities in a series of balancing equations. Note that there is an implied acceptance here that the results of the 1986 Census were correct in level of enumeration of the Aboriginal population; if they were not, then all the results in this analysis would be biased accordingly through the action of biased denominators for death rates. The procedure included further adjustments to the population data but importantly produced a final set of estimates of age-sex-specific death rates for each of the geographical areas.

The reason that the second stage is necessary is illustrated very well in Figures 5 and 6, which show three sets of survival ratios for the 1981-86 period for each sex for New South Wales and the Australian Capital Territory. Here a survival ratio is the proportion of people from an age group in 1981, for example the zero to four age group, who are still living five years later in 1986, who for the example would be in the five to nine age group. It is abundantly obvious that a survival ratio cannot be greater than one, that is there cannot be more survivors than were in the age group in 1981. It is also and unfortunately obvious in these figures that many of the estimated survival ratios are in fact greater than one, even when the adjustment for level has been undertaken in the first stage of analysis. The first 'original' set of survival ratios are vastly too high, the second 'level' set of ratios contain impossible values greater than one, and only the third 'model' set which is the output of the second stage of analysis produces a meaningful result. We may characterise the first stage of analysis as a gross adjustment of the level of survival ratios and the second stage as a fine adjustment of the progression of age-specific ratios.

Figure 5 Adjustment of census survival ratios for Aboriginal males in New South Wales and the Australian Capital Territory, 1981-86.

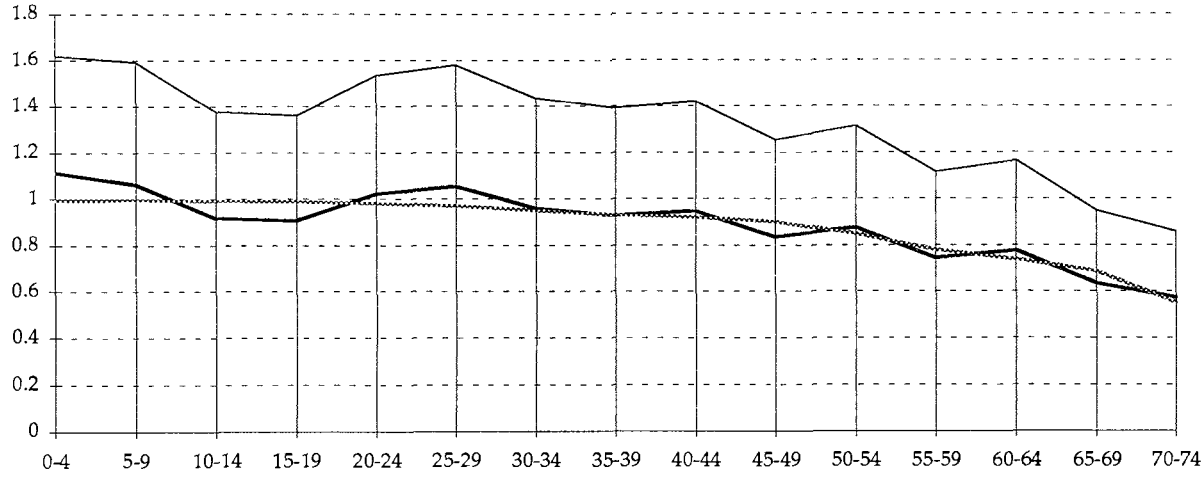
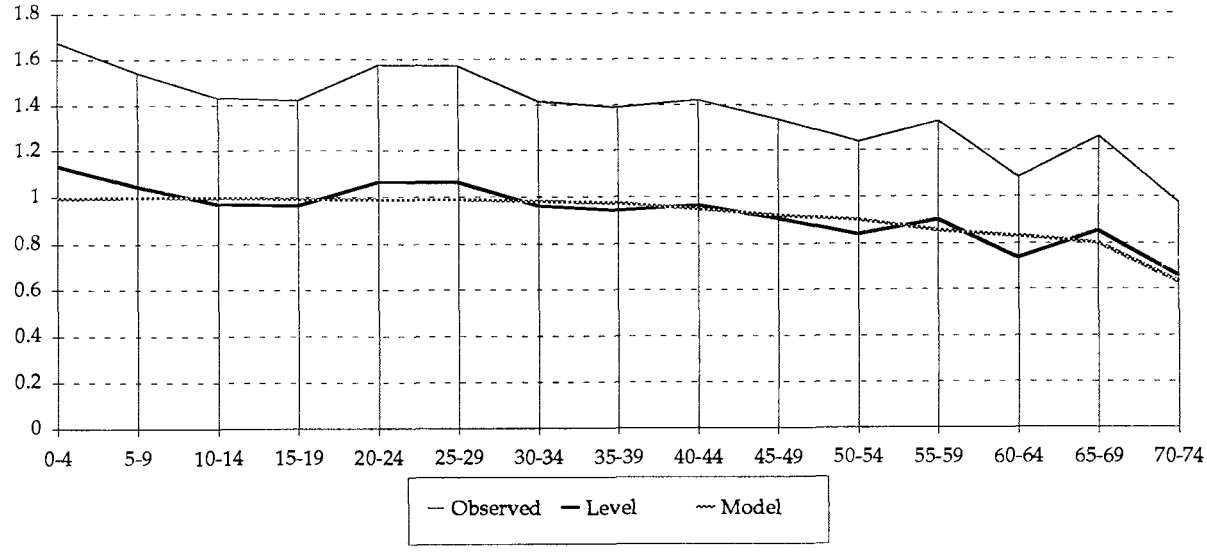


Figure 6 Adjustment of census survival ratios for Aboriginal females in New South Wales and the Australian Capital Territory, 1981-86.



It should be emphasised that the process of constructing a reasonable series of age-sex-specific mortality rates depends completely on successful implementation of both stages of this analytical process. It is also necessary to mention that the results of the second stage are sensitive to the initial estimates of mortality pattern, which for this analysis were set out in Figures 1 and 2. Any change to the initial estimates can result in quite a substantial shift in the results. The extent of this sensitivity can be

illustrated by pointing out that an arbitrary across-the-board alteration of the preliminary age-specific mortality rates by plus or minus 10 per cent resulted in changes almost as large in the final estimates, and in additions or subtractions of 1.3 to 1.5 years to the expectations of life implied by the set of mortality rates for each State.

This aspect of analytical sensitivity means that while the differences found between different States in the analysis are no doubt genuine and in the right directions, they may, but by no means necessarily do, understate the extent of difference between the patterns of Aboriginal mortality in the various States. This sensitivity can be modified slightly in the Luther-Retherford procedure, by altering weightings attached to distance measures in the functional analysis. Luther and Retherford left choice of weighting systems inconclusive, but suggested that the weight attached to the correction factor for any measure could be chosen to be proportional to its size. In the current application this suggestion was modified in the first instance by giving correction factors for deaths a lower weighting corresponding to the scaling necessary to construct mortality rates from intercensal deaths and population estimates for each end-point, and it was found that this lower weighting actually induced slightly greater variation in resultant estimates. Experimentation with greatly lower weightings revealed that it was actually possible to produce negative estimates of deaths in some age groups and survival ratios which closely resembled the impossible 'level' ratios of Figures 5 and 6. But in general, it was found that the procedure was not particularly sensitive to choice of weighting system compared with altering the level of the preliminary mortality estimates.

The main results of the analysis are presented in Table 2, which shows estimates of age-specific mortality rates for each age, sex and geographical grouping, including aggregate estimates for the whole of the Australian Aboriginal population. It can be seen that the extent of variation in the estimates of age-specific mortality is quite considerable between the different States, suggesting that the approach used was quite capable of displaying the directions of variation from the preliminary estimates. The summary measure shown for each sex and geographical area is expectation of life at birth (e0), which is lowest for the Northern Territory for each sex, next lowest in Western Australia, progressing to relatively higher values in Queensland followed by South Australia, New South Wales, Victoria and then Tasmania. This is the type of progression that would be expected, but note that it is less even than this on an age-by-age basis.

For instance, South Australian Aborigines have relatively high early childhood mortality in age group zero to four, compared with their overall ranking, by these estimates. Similarly, the highest male mortality in the two age groups 40 to 44 and 45 to 49 is in Western Australia, not in the Northern Territory which has the highest overall mortality, or equivalently lowest expectation of life. There are many such features in the estimates; all of them can be expected to indicate some peculiarity of the mortality patterns in the different States, even though the method of analysis which has been used here might not be capable of picking up the full extent of the variations effectively. If the aggregate mortality rates are plotted as in Figures 1 and 2, the result is still very similar, although the analysis has resulted in some smoothing of the bumps in the preliminary estimates.

Conclusions

The first aim of this analysis was to demonstrate, through the application of what are admittedly very powerful and somewhat difficult techniques of demographic balancing, that it is entirely possible to produce reasonable estimates of Aboriginal mortality for Australia as a whole from the deficient data that are available currently. The need for this type of analysis may be expected to diminish progressively as the new sources of data from official sources are improved. In the medium-term, especially for the present intercensal period from 1986 to 1991, it remains very likely that similar techniques will remain necessary even if we will become increasingly sure that the results obtained are valid for particular geographical areas simply because there will be at least some validating data from those areas where there are currently none.

Table 2 Estimated age-specific mortality rates for Aboriginal Australians per 1,000 population, 1981-86.

	NSW & ACT	VIC & TAS	QLD	SA	WA	NT	AUST
Males							
0-4	7.0	5.8	7.8	9.1	8.6	9.0	7.9
5-9	0.5	0.5	0.6	0.6	0.6	0.7	0.6
10-14	1.5	1.2	1.6	1.5	1.8	1.8	1.6
15-19	2.1	1.8	2.1	2.1	2.2	2.2	2.1
20-24	3.1	2.9	3.5	3.3	3.5	3.7	3.3
25-29	5.9	5.9	6.7	6.7	7.2	8.0	6.7
30-34	7.3	7.0	7.4	7.9	7.8	8.3	7.6
35-39	13.9	11.1	14.6	14.7	14.4	15.4	14.2
40-44	14.8	16.0	17.0	17.6	18.2	17.7	16.7
45-49	18.9	18.4	20.0	19.1	22.2	20.4	20.0
50-54	22.9	23.9	26.4	24.5	27.8	29.8	26.1
55-59	43.9	40.5	45.5	39.5	42.9	46.8	44.2
60-64	57.4	67.3	60.2	47.6	52.8	73.5	60.4
65-69	64.5	78.1	69.5	57.3	70.7	75.0	69.8
70-74	88.7	95.7	95.4	85.4	90.0	123.2	97.5
75+	127.8	132.2	134.9	132.2	119.8	196.7	138.9
e0	56.9	57.2	55.6	56.2	55.3	53.7	55.7
Females							
0-4	5.2	4.4	6.1	6.6	6.5	6.9	6.0
5-9	0.3	0.2	0.3	0.3	0.4	0.4	0.3
10-14	0.3	0.2	0.4	0.3	0.4	0.4	0.4
15-19	1.5	1.2	1.6	1.4	1.6	1.5	1.5
20-24	2.2	2.0	2.4	2.3	2.7	2.6	2.4
25-29	2.2	2.2	2.5	2.7	2.8	2.8	2.5
30-34	3.3	3.0	3.4	3.7	3.7	3.9	3.5
35-39	5.6	5.3	5.6	6.3	5.8	6.0	5.7
40-44	6.2	6.8	7.1	6.8	6.9	7.1	6.8
45-49	16.3	15.2	18.2	16.6	18.3	19.6	17.7
50-54	17.0	12.3	19.1	17.1	19.1	22.1	18.5
55-59	25.2	22.6	27.1	24.9	23.4	23.2	24.9
60-64	38.9	33.4	37.8	23.0	40.4	43.4	38.4
65-69	35.8	32.4	41.4	29.7	43.5	54.8	41.8
70-74	55.6	55.8	60.3	57.7	64.5	76.1	62.4
75+	100.9	98.2	99.2	101.6	111.1	124.3	106.1
e0	65.0	66.6	63.9	65.1	63.2	61.8	63.9

There were some other aims of the analysis. By demonstrating that Aboriginal mortality levels are indeed slightly higher in those geographical areas of Australia in which there are more remote and rural communities, the analysis conforms with expectations which had been confused by previous production of estimates for different parts of Australia which in some cases actually reversed these

Figure 7 Age-specific death rates and logarithmic scale for Aboriginal males in the Northern Territory, 1964-68 and Australia, 1981-86.

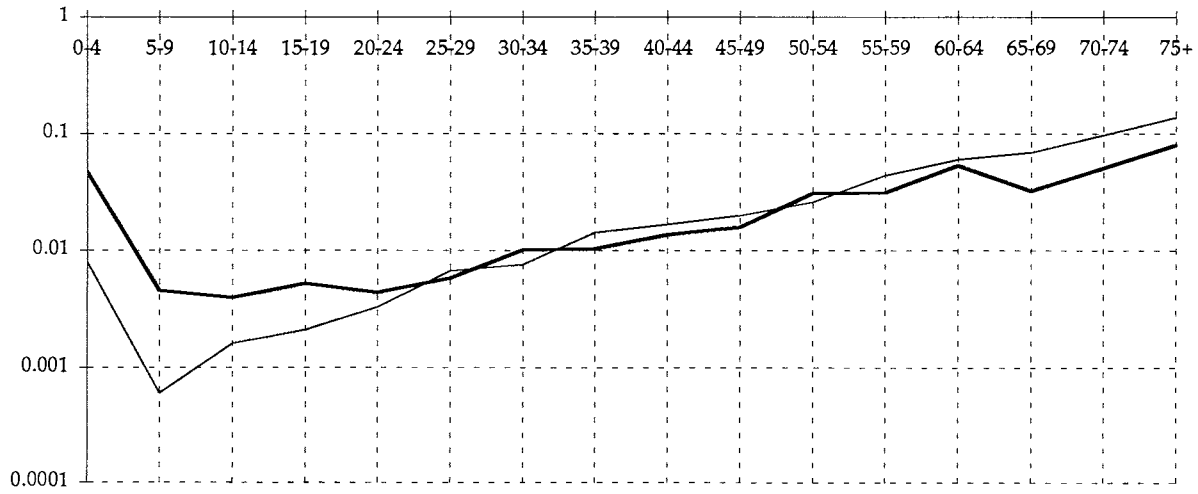
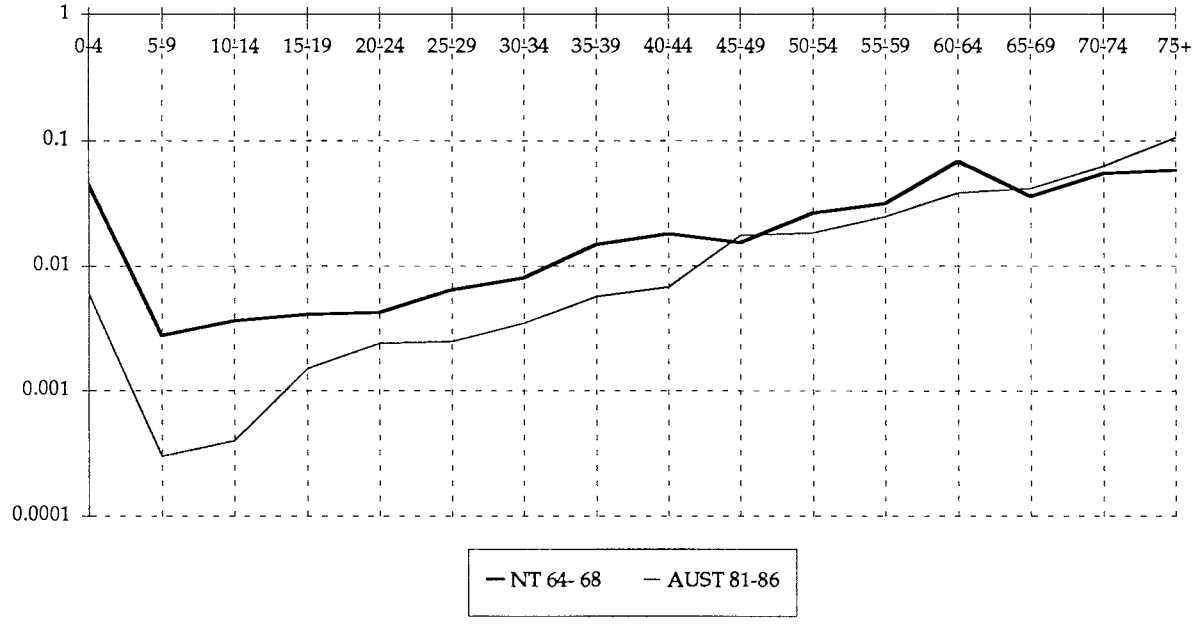


Figure 8 Age-specific death rates and logarithmic scale for Aboriginal females in the Northern Territory, 1964-68 and Australia, 1981-86.



expectations. Yet the differences between States are actually very small, by these findings, compared with differences between the mortality rates of the Aboriginal population and the total population of Australia. As has been noted in the analysis, the rather small geographical differences located could have been artificially created by the method, but on the other hand actual data from a number of States really show very similar levels.

Previous analysis has not drawn much attention to it, but it is very obvious both from the preliminary age-sex-specific mortality rates, drawn from actual data, and the balanced estimates for all geographical areas, that males under the age of five have much higher mortality than females under the age of five. In the data for western New South Wales (Gray and Hogg 1989), a major component of this difference occurred in the form of sudden infant death. At all subsequent ages, as has been noted by all previous analysts, males have very much higher death rates than females, and consequently very much lower expectation of life. It is, by international standards, quite unusual to find such a large gap between male and female expectation of life for a population with life expectancy as low as in the Aboriginal population. This is because low expectation of life is usually associated with very high infant mortality. Aboriginal infant mortality is high by Australian standards but is now low compared with nearly all developing countries. As a result, the differences between male and female expectations of life are largely determined by adult mortality, as in a population with low mortality, and consequently a large male-female difference is found.

The final aim of this analysis was simply to set out baseline estimates of Aboriginal mortality for use in assessing future movements in levels and age distribution, for population projection and for general health assessment. It is in many respects very unsatisfying to work with estimates which have so little content of explanation as the ones which have been set out here, but at the same time it is necessary to work from a more factual basis than has been available before now. What must be added to this analysis will be very obvious from the papers from regional studies which have been presented in this volume: it is necessary to look at patterns of causation of death and beyond to the social environment which produces these patterns and the common outcomes for Aboriginal people in all parts of Australia.

There is, however, one very useful immediate purpose which can be served by the estimates which have been produced here, and that is to compare the pattern of Aboriginal mortality throughout Australia in the early 1980s with the pattern in the Northern Territory in the period 1964 to 1968 (from Gray 1983a), as was also done in a recent paper (Gray 1989b). The relevant part of the comparison is reproduced in Figures 7 and 8. These potently illustrate that male adult mortality rates for Australia in the 1980s have exactly the same pattern and level as those for the Northern Territory in the 1960s. (For infants, children and teenagers, the earlier Northern Territory data have a much higher pattern.) A slightly different picture is conveyed from the equivalent data for females. In this case, the higher pattern in the 1960s extends through the ages of childbearing until the early 40s. The most tangible conclusion from this comparison is that the emphasis on maternal and child health in

expansion of health services during the past 20 years ostensibly coincided with a major impact on improvement of survival chances for infants but also on survival chances of older children and mothers themselves. A causal link must be hypothetical but is certainly likely. Apart from these improvements, we must conclude that the pattern of Aboriginal mortality throughout Australia in the 1980s was not only similar in different parts of Australia but also similar to the Northern Territory in the 1960s.

NOTES

1. The Luther-Retherford procedure is computationally complex and is only feasible as a computer method. The application in this paper is not identical to the example given in the Luther-Retherford paper (1988).
2. It should be mentioned that inter-State migration in the Aboriginal population is quite substantial. While numbers of in-migrants to any State are almost exactly balanced by numbers of out-migrants, for each age and sex, all population data were nevertheless adjusted by subtracting intercensal out-migrants (as measured in 1986) from the corresponding 1981 age cohorts and subtracting intercensal in-migrants from the 1986 age cohorts. The results were not very sensitive to these adjustments but they were included for sake of completeness, because the methods of analysis assume closed populations. Strictly, none of the results can be assumed to apply to inter-State migrants.

15 ABORIGINAL AND NON-ABORIGINAL DEATHS IN PRISON AND POLICE CUSTODY[1]

David Biles, David McDonald and Jillian Fleming

This chapter[2] aims to place the phenomenon of Aboriginal deaths in custody in the broader context of all deaths in prison and police custody over the nine year period from 1 January 1980 to 31 December 1988. The project, informally known as 'Black and White Deaths in Custody', was undertaken with the full cooperation and assistance of all Australian police forces and prison services who supplied the basic information.

The data to be analysed in this chapter can be presented very simply and are shown in Table 1, but several explanatory points must be made before any analysis is attempted. In the first place some explanation is needed as to why the Aboriginal total is lower than the total on the official Royal Commission list, currently 103. This difference is partially due to the removal of juvenile cases from the project, as no attempt was made to obtain details of non-Aboriginal juvenile cases. Also, deaths in sobering-up centres were excluded as it is doubtful whether the term 'custody' applies to these cases. Possibly the most important reason for the difference, however, is the fact that police and prison authorities exercised their own judgement in supplying details of cases for the purposes of this project. Therefore some cases awaiting determination by the Commissioners for inclusion or exclusion from the official list are not a part of this project. It should be pointed out that no attempt was made to apply the precise terms of reference of the Royal Commission to the non-Aboriginal cases in this study.

Table 1 Deaths in custody by custodial authority and race, 1980-88.

	Aboriginal	Non-Aboriginal	Total
Prison	35	245	280
Police	5	123	182
Total	94	368	462

It was intended that the lists of Aboriginal and non-Aboriginal deaths compiled for this study should be based on the same criteria for inclusion, but it is apparent that there has been some degree of under-counting with non-Aboriginal deaths. This seems to be due to the fact that, at least until the establishment of the Royal Commission, some agencies did not maintain central registries of all deaths. It is therefore possible, even likely, that a number of cases where the individual was transferred to hospital before death, may have been missed by this project. Hypothetically, a person could have died in hospital while technically still under

police custody and, if there were no suspicious circumstances, no central record would have been kept.

The fact that some non-Aboriginal deaths may have been overlooked is illustrated by drawing a distinction between the actual location of death and the custodial authority at the time of death. As is shown later in this chapter, proportionately more Aboriginal than non-Aboriginal deaths occurred in hospitals rather than in police or prison facilities. This difference, it is suspected, is due to the fact that the very existence of the Royal Commission has brought to notice more cases of Aboriginal deaths in custody than otherwise would have been the case.

Even though it cannot be claimed that absolutely identical criteria for inclusion in this project were applied to both the Aboriginal and non-Aboriginal groups, considerable effort was made to ensure that the data were as accurate and comprehensive as possible. In late 1988, complete lists of all deaths in all police and prisons administrations over the nine year period were prepared and these were forwarded to the relevant authorities for confirmation or amendment. Each authority subsequently certified the accuracy of the data supplied by each jurisdiction. The lists of names that form the basis of this study were as known on 3 March 1989.

As a further partial check on the accuracy of the data that had been supplied, the statistics of deaths in prisons over the period 1980 to 1985 inclusive were compared with those which formed the basis of a study by the Australian Institute of Criminology (Hatty and Walker 1986). That comparison revealed almost identical numbers of prison deaths over that period for all jurisdictions except Queensland which reported slightly higher numbers for this study than for the Institute study. For all other jurisdictions, any differences between the numbers in the two studies are either extremely small or non-existent, thus increasing confidence in the accuracy of the data.

The data obtained from police and prison sources were not exactly identical as some items were more or less relevant in different custodial situations. Police forces supplied lists which included for each case: name, sex, Aboriginality, age, time and date of death, place of death, cause/manner of death, reason for detention, alcohol level if known, time in custody, time since inspection, and whether or not the deceased person was alone at the time of death. In the case of prison deaths, the correctional agencies completed forms which covered similar items to the police lists but excluded reference to alcohol level and included more detail of the place of death (single cell, dormitory and so on), legal status, time served, time to serve, and made provision for other relevant factors (for example, drugs, psychiatric history, temporary leave) to be noted. Thus, marginally more information was available for prison deaths than was available for police deaths.[3]

A final introductory point should perhaps be made. This project aims to describe and analyse the facts relating to the phenomenon of Aboriginal and non-Aboriginal deaths in custody. The project was not planned as a basis for comparisons to be made between the relative incidence of Aboriginal and non-Aboriginal deaths and the relative numbers of Aboriginal and non-Aboriginal people in prison and police custody. These comparisons can be made, however, by relating the facts in this chapter with those in Research Paper No 6 (Biles 1989) and the results of the national

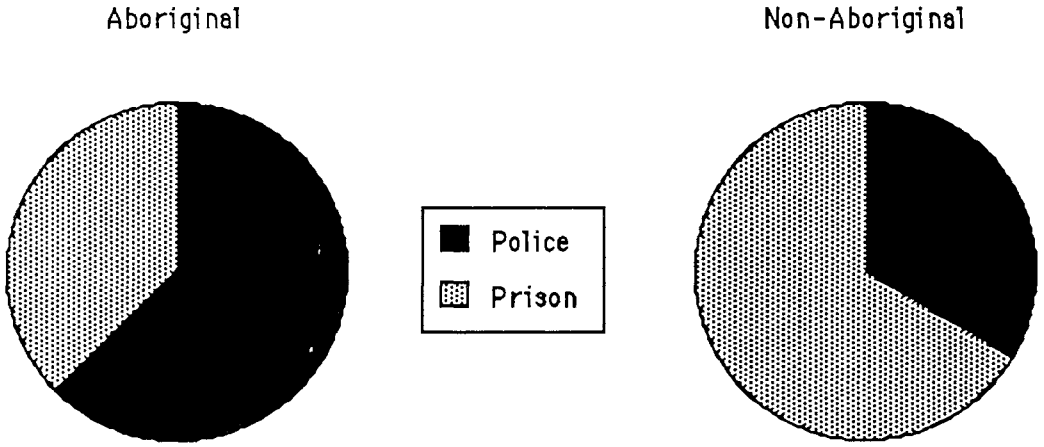
police custody survey which were recently released in preliminary form (McDonald 1989).

It seems that Aborigines in custody constitute a more representative cross-section of the Aboriginal population than non-Aborigines in custody. The fact that Aborigines are so grossly over-represented in custody indicates that different selective factors may be operating with respect to arrest and/or sentencing. Together this suggests that some caution is needed in the interpretation of Aboriginal and non-Aboriginal comparisons presented here.

National overview

As shown in Table 1 above, the basic data set comprises 462 cases of deaths in prison or police custody. It can also be seen that there is a marked difference between the two population groups and the relevant custodial authority. The Aborigines who die in custody are much more likely to die in police custody than in prisons; conversely, non-Aborigines are much more likely to die in prison. This striking difference is illustrated in Figure 1 and discussed in more detail further on.

Figure 1 Aboriginal and non-Aboriginal deaths in prison and police custody as a percentage of deaths in custody.



The basic data set will be examined in many different ways and the first such examination shows any differences that might exist between Australian States and Territories (see Table 2). It can be seen that there are noticeable differences between jurisdictions, with, for example, the Northern Territory having more Aboriginal than non-Aboriginal deaths and Western Australia having nearly equal numbers. In all other jurisdictions non-Aboriginal deaths out-number Aboriginal deaths to a significant degree. There are also differences between jurisdictions in the distribution of police and prison deaths. Most noticeable is the fact that, when Aboriginal and non-Aboriginal deaths are added, Western Australia and the Northern Territory have

more police deaths than prison deaths. The opposite is the case in most other jurisdictions.

Table 2 Aboriginal and non-Aboriginal deaths in prison and police custody by jurisdiction, custodial authority and race, 1980-88.

	Aboriginal			Non-Aboriginal			
	Police	Prison	Total	Police	Prison	Total	All cases
NSW	7	6	13	44	77	121	134
VIC	3	-	3	22	75	97	100
QLD	16	8	24	22	49	71	95
WA	20	13	33	17	18	35	68
SA	6	4	10	9	18	27	3
TAS	1	-	1	4	5	9	10
NT	6	4	10	5	2	7	17
ACT	-	-	-	-	1	1	1
AUST	59	35	94	12	245	36	462

NOTE: In all tables in this chapter, where relevant: some percentages may not total to 100, owing to rounding; cross tabulations (ie variable 1 by variable 2) include only the cases where data are available on each of the variables in the table; and the symbol '-' means nil, or a number rounded to nil.

As detailed in Table 4, over the nine year period under review, 94 of the 462 deaths, or 20 per cent, occurred among Aboriginal people in police or prison custody. This is a ratio of 41 deaths per 100,000 of the Aboriginal population of Australia at the 1986 Census. The comparable non-Aboriginal ratio is two per 100,000 population and for all deaths in custody the ratio is three per 100,000. When we consider only the population aged 15 years and above, we find that the corresponding ratio of total deaths in custody to population is four per 100,000, the non-Aboriginal ratio three and the Aboriginal ratio 69 (see Table 3). Taking the analysis a little further, by including only males aged 15 years and older (since this group makes up 95 per cent of the 1980-88 deaths in custody), we find that the corresponding ratios are seven per 100,000 for all deaths in custody, six per 100,000 for non-Aboriginal deaths and 131 per 100,000 for Aboriginal deaths. This alarmingly high Aboriginal death rate is explained, almost entirely, by the over-representation of Aboriginal people in custody.[4]

Table 3 Deaths to population ratios for deaths in custody, 1980-88.

	Population 1986 Census	Persons 15 years+	Deaths in custody 1980-88	Deaths per 100,000 population over 15 years
Aboriginal	227,645	137,13	94	69
Non-Aboriginal	15,374,511	11,965,311	368	3
Total	15,602,156	12,102,444	462	4

Substantial differences exist between the eight States and Territories regarding numbers of deaths in custody. As shown in Table 4, by far the highest proportion, compared with the population, is found in the Northern Territory (11 deaths per 100,000), followed by Western Australia (five) and Queensland (four). The Aboriginal deaths, however, show a different pattern with Western Australia having

the highest ratio at 87 Aboriginal deaths per 100,000 Aboriginal population, followed by South Australia (70) and Queensland (39).

Table 4 Deaths to population ratios by jurisdiction and race, 1980-88.

	Aboriginal deaths in custody	Aboriginal death ratios[1]	Non-Aboriginal deaths in custody	Non-Aboriginal death ratios[2]	Total deaths in custody	State death ratios[3]
NSW	13	22	121	2.3	134	2.5
VIC	3	24	97	2.4	100	2.5
QLD	24	39	71	2.8	95	3.7
WA	33	87	35	2.6	68	4.8
SA	10	70	27	2.0	37	2.7
TAS	1	15	9	2.1	10	2.3
NT	10	29	7	5.8	17	11.0
ACT	-	-	1	0.4	1	0.4
AUST	94	41	368	2.4	462	3.0

NOTES:
1. Aboriginal deaths in custody 1980-88 per 100,000 of the Aboriginal population at the 1986 census.
2. Non-Aboriginal deaths in custody 1980-88 per 100,000 of the non-Aboriginal population at the 1986 Census.
3. Total State/Territory deaths in custody 1980-88 per 100,000 population at the 1986 Census.

For deaths in custody occurring over the period 1980-88, many calculations use 1986 Census data. This is because, according to the ABS the 1986 Census data provide the best available indication of the size and composition of the Aboriginal population. As discussed in detail in the Royal Commission's Research Paper No 4 (Smyth 1989), the apparent increase in the Aboriginal population from 159,897 in the 1981 Census to 227,645 in the 1986 Census is probably explained, to a substantial degree, by both improved enumeration techniques and by an increasing proportion of Aboriginal people being prepared to identify themselves as such.

Demographic characteristics

Table 5 sets out the race and sex of the deceased. It will be noted that males accounted for most of the deaths (95 per cent), reflecting the sex compositions of the prison and police custody populations. Aboriginal people composed 20 per cent of the deaths, although they are less than 1.2 per cent of the Australian population aged 15 years and above. Six of the seven Aboriginal women who died in custody, died while in the custody of police.

Table 5 Deaths in custody by sex and race, 1980-88.

	Aboriginal		Non-Aboriginal		Total	
	N	%	N	%	N	%
Male	87	93	354	96	441	95
Female	7	7	14	4	21	5
Total	94	100	368	100	462	100

Again reflecting the population within which the deaths occurred, the age groups are skewed towards the younger end compared with the overall Australian

population, as shown in Table 6. As described in Research Paper No 4 (Smyth 1989), major differences in age structure exist between the Aboriginal and total Australian populations. The Aboriginal population is much younger than the total population (53 per cent aged under 20 years compared with 31 per cent) and has a much lower proportion over 59 years of age (four per cent compared with 15 per cent).

Table 6 Deaths in custody by age group and race, 1980-88.

	Aboriginal		Non-Aboriginal		Total		General population
Age group	N	%	N	%	N	%	%
<15	-	-	-	-	-	-	23
15-19	10	11	28	8	38	8	8
20-24	13	14	73	20	86	19	8
25-29	24	26	72	20	96	21	8
30-34	14	15	40	11	54	12	8
35-39	5	5	30	8	35	8	8
40-44	12	13	35	10	47	10	6
45-49	3	3	20	5	23	5	5
50-54	6	6	21	6	27	6	5
55-59	6	6	17	5	23	5	5
60-64	1	1	13	4	14	3	4
65+	-	-	19	5	19	4	11
Total	94	100	368	102	462	101	99

NOTE: The proportions for the 'general population' figures are based on 1986 Census data.

Interestingly, little difference exists, overall, between the ages of the Aboriginal and non-Aboriginal groups, when we consider only those who died in custody. The mean age was 34.9 years, with half the cases being aged less than 30 years. The corresponding figures for the Aboriginal deaths were 33.1 and 29.5 years, respectively; for non-Aboriginal deaths, 35.4 and 30.0 years respectively. The ages ranged from 17 to 62 among Aboriginal people and 17 to 88 among non-Aboriginal people.

A close examination of Table 6 reveals, however, that these summary data conceal differences in the age groups of the Aboriginal and non-Aboriginal people who died in custody. In particular, it will be noted that a substantially higher proportion of Aboriginal people, compared with non-Aboriginal people, were aged between 25 and 34 years. The reverse is seen in the 45 years and above age group.

In interpreting age data, it is also important to note the disparity that exists between the Aboriginal and non-Aboriginal populations, in the general community, regarding age-specific mortality rates and life expectancies. As summarised in a recent review: 'for almost all disease categories, rates for Aboriginal and Torres Strait Islanders are worse than for other Australians; death rates are up to four times higher, and life expectancy is up to twenty-two years less' (Australian Institute of Health 1988). Furthermore, as detailed below, suicide (reported as being a common cause of deaths in custody) is predominantly seen in young males who are, of course, substantially over-represented in the custody population compared with the general population. Finally, it is noted that the age distributions of people who died in police and prison custody are similar, with means of 36.0 and 34.2 years respectively.

The legal status (ie sentenced or on remand) of 268 of the 280 prison deaths and of one case of a person serving a short sentence in a police lock-up is known. As

shown in Table 7, some 28 per cent of these deaths occurred among remanded or unconvicted prisoners. Since remandees make up only 16 per cent of the Australian prison population,[5] it is clear that they are particularly at risk of dying in custody. Non-Aboriginal remandees had a substantially higher death rate than did Aboriginal remandees. Suicide was the most frequent reported cause of death among remandees, accounting for 72 per cent of their deaths, compared with only 35 per cent of the deaths of sentenced prisoners.

Table 7 Deaths in custody by legal status and race, 1980-88.

	Aboriginal		Non-Aboriginal		Total	
	N	%	N	%	N	%
Remanded	6	17	70	30	76	28
Sentenced	29	83	164	70	193	72
Total	35	100	234	100	269	100

Reported cause of death

Data are available on the reported cause of death in 445 (96 per cent) of the cases. The categories used in this study are suicide (or self-inflicted deaths), homicide, accident (including accidental drug overdoses), natural causes and a residual category 'other'. (These data are as reported by State authorities, frequently based on coroners' findings, and may not necessarily correspond with the determinations that are to be made by the Commission.) It should be noted that the term 'suicide' is used in this chapter with the meaning used by social scientists and in everyday speech (and also by the providers of the data), namely, covering all self-inflicted deaths, regardless of whether or not the person had formed a definite intention of dying by his or her own hand. This approach is different from the legal definition of 'suicide' in which the finding of intention to die by one's own hand, and the capacity to form such an intention, are crucial elements (Royal Commission into Aboriginal Deaths in Custody 1989, 53-57).

As shown in Table 8, self-inflicted death or suicide was by far the most common category, accounting for 204 or 46 per cent of the deaths for which the cause is known. Natural causes was the next most frequent category, accounting for 158 or 36 per cent of these deaths. A significant difference exists between the races: a higher proportion of Aboriginal people, compared with non-Aboriginal people, died of natural causes (46 per cent compared with 33 per cent) whereas the reverse holds for suicides with 49 per cent of non-Aboriginal people being reported to have died this way, compared with 34 per cent of Aborigines. This pattern is found in both the police and prison custody groups.

Table 8 Deaths in custody by reported cause of death and race, 1980-88.

Cause of death	Aboriginal		Non-Aboriginal		Total	
	N	%	N	%	N	%
Self-inflicted	31	34	173	49	204	46
Natural causes	42	46	116	33	158	36
Accident	10	11	41	12	51	11
Homicide	4	4	17	5	21	5
Other	5	5	6	2	11	2
Total	92	100	353	101	445	100

As one might expect, age is correlated with reported causes of death. Suicide was the most frequent reported cause of death among the younger age groups, accounting for 70 per cent of the cases under 30 years of age. It will be recalled that half (48 per cent) of the 13 deaths are found in this age group. Natural causes were dominant in the older age groups, accounting for 61 per cent of the cases aged over 40 years.

Information is available on the methods used in 200 of the 204 self-inflicted deaths. Hanging was by far the most frequent, representing 183 or 91 per cent of these deaths. The other methods used were drugs (six cases) and other forms of trauma (11 cases). This pattern is quite different from that of suicides in the general community, where hangings make up only 13 per cent of such deaths, 14 per cent among males and 9 per cent among females (Australian Bureau of Statistics 1983). In the broader community, suicide by poisoning and by the use of firearms are much more common than suicide by hanging. The differences between methods of self-inflicted death in custody as compared with non-custody circumstances are explained, in part, by the constrained options of the custodial environment, where there is virtually no access to poisons or firearms, where there is more continual surveillance, and where there is little choice of location. Therefore hanging is one of the few options available, and is a particularly lethal method which typically precludes intervention, change of mind, or resuscitation. Similar proportions of males and females were reported to have died by suicide in custody. This is in contrast to the general community where the male suicide death rate is almost four times that of females.

As mentioned at the beginning of this chapter, the patterns of deaths in custody for Aboriginal and non-Aboriginal people are very different, with 67 per cent of non-Aboriginal deaths having occurred in prison custody and 63 per cent of Aboriginal deaths having occurred in police custody. When these figures are broken down into the proportion of reported suicide deaths covered by these figures, it becomes clear that 114 of 173 non-Aboriginal suicides took place in prisons (66 per cent), whereas 20 of 31 Aboriginal suicides took place in police custody (65 per cent), as shown in Table 9. In other words, the differing pattern of self-inflicted deaths in custody closely parallels the more general pattern. This would suggest that police custody is more of an at-risk environment for Aboriginal people than it is for non-Aboriginal people. It is also noteworthy that suicides constitute 49 per cent of non-Aboriginal deaths in custody, as compared with a little less than 34 per cent of Aboriginal deaths in custody.

Table 9 Deaths in custody by reported cause of death, custodial authority and race, 1980-88.

Cause of death	Aboriginal		Non-Aboriginal		Total		All
	Police	Prison	Police	Prison	Police	Prison	cases
Self-inflicted	20	11	59	114	79	125	204
Natural causes	23	19	39	77	62	96	158
Accident	8	2	14	27	22	29	51
Homicide	3	1	2	15	5	16	21
Other	3	2	1	5	4	7	11
Total	57	35	115	238	172	273	445

It may be useful, for preventive purposes, to explore further the deaths attributed to 'natural causes'. Some information on the diseases involved is available for 137 (87 per cent) of the 158 deaths by natural causes. These have been classified according to the broad categories used in the ICD-9 (World Health Organization 1977) as shown in Table 10. It can be seen that heart afflictions are the most common diseases causing deaths in custody, accounting for almost half (47 per cent) of these deaths. By comparison, 36 per cent of deaths in the community (that is, deaths from disease, rather than external causes such as suicide or motor vehicle crashes) are from heart disease (Australian Bureau of Statistics 1989). This high rate of deaths from heart disease is especially marked considering the younger ages of people who died in custody compared with the general community, as shown in Table 6. Cancer and stroke are the second and third most frequent causes of death by disease in the general community (cancer 26 per cent, cerebro-vascular disease including stroke 11 per cent); the deaths in custody rates are much lower, at three per cent and six per cent respectively, as one might expect with the younger custody population.

Table 10 Deaths in custody by natural causes by custodial authority and race, 1980-88.

	Police		Prison		Total		
Disease group	Aboriginal	Non-Aboriginal	Aboriginal	Non-Aboriginal	Aboriginal	Non Aboriginal	All cases
Heart disease	5	13	12	35	17	48	65
Respiratory tract diseases	4	4	1	6	5	10	15
Stroke	2	2	-	5	2	7	9
Infectious diseases	1	-	1	5	2	5	7
Cancers	-	-	-	4	-	4	4
Digestive system diseases	-	2	2	1	2	3	5
Other disease	6	6	1	5	7	11	1
Multiple diseases	2	4	1	7	3	11	14
Total	20	31	18	68	38	99	137

Finally, it is noted that little difference exists between the Aboriginal and non-Aboriginal patterns of deaths in custody by natural causes, but significant differences do exist between the custodial authorities, that is, police and prison authorities respectively. Of interest is the fact that deaths from heart diseases, infectious illnesses and cancers occur at higher rates in prison compared with police

custody, whereas the opposite is the case for deaths caused by respiratory system disease. The fact that people stay much longer in prison than in police custody is, perhaps, relevant here.

Fifty-one deaths in custody were reported to be caused by accidents. Drug overdoses were by far the most common (22 cases), followed by alcohol toxicity (eight cases), fire (eight), head injury (eight) and other accident types (seven). As shown in Table 11, non-Aboriginal people in prison accounted for 16 of the 22 drug overdose deaths. Homicide was reported to be the cause of death in 21 cases. As shown in Table 12, 16 of these occurred in prison, slightly more than one would expect from the size of the prison deaths category compared with the police deaths category. Aboriginal and non-Aboriginal homicide deaths were proportional to the sizes of their respective groups in the deaths in custody population. A high proportion (10 of the 21 reported homicides) occurred in New South Wales, which had only 29 per cent of the deaths in custody, and nine of those 10 occurred in prison custody.

Table 11 Accidental deaths in custody by custodial authority and race, 1980-88.

Accident type	Police		Prison		Total		All cases
	Aboriginal	Non-Aboriginal	Aboriginal	Non-Aboriginal	Aboriginal	Non-Aboriginal	
Drugs*	1	4	1	16	2	20	22
Alcohol*	1	7	-	-	1	7	8
Fire	-	-	-	8	-	8	8
Head injury	5	I	-	-	5	1	6
Other	1	2	1	3	2	5	7
Total	8	14	2	27	10	41	51

NOTE: The asterisk denotes deaths from overdose and/or sensitivity to alcohol or other drugs.

Table 12 Deaths in custody from homicide by custodial authority and race, 1980-88.

	Aboriginal	Non-Aboriginal	Total
Prison	1	15	16
Police	3	2	5
Total	4	17	21

Characteristics of custody

Data were sought on the most serious offence, committed by the deceased person, that led to him or her being placed in custody. Offence data are available on 365 of the cases (79 per cent, including 95 per cent of the prison cases and 54 per cent of the police cases). This discrepancy between the police and prison cases means that the data, especially the police data, must be used with caution.

The offence categories listed in Table 13, and used in other Research Papers of the Royal Commission, are based on the Australian National Classification of Offences developed by the ABS. Full details of the criminal behaviours included

within each category are found in ABS publications (Australian Bureau of Statistics 1986); for the purposes of this chapter, however, the following brief descriptions apply:

Homicide includes murder, attempted murder, conspiracy to murder, manslaughter and driving causing death.
Assault Offences includes occasioning grievous bodily harm, actual bodily harm etc.
Sexual Offences includes rape, carnal knowledge, incest, indecent assault, etc.
Robbery includes armed robbery, unarmed robbery, stealing with violence, etc.
Break and Enter includes burglary and unlawful entry.
Fraud includes misappropriation, forgery and false pretences.
Theft includes receiving and unlawful possession of stolen goods as well as motor vehicle theft and illegal use, stealing from the person, etc.
Property Damage includes arson and vandalism.
Justice Procedures includes breach of probation, parole, contempt of court, breach of family law court orders, escape from custody, fine default, etc.
Drunkenness includes both public drunkenness where is it is an offence and protective custody or detention where this form of behaviour has been decriminalised.
Other Good Order Offences includes offensive behaviour, weapons offences, etc.
Drug Offences includes possession or using, importing, dealing, or trafficking, manufacture and growing of any unlawful drugs.
Traffic Offences includes all driving offences (including driving under the influence of alcohol) and licence or registration offences.
Other Offences is a residual category for offences not elsewhere classified.

Table 13 shows the most serious offences that led to police custody or imprisonment for each deceased person. It will be noted that homicide is by far the most common offence, accounting for 20 per cent of the cases for which data are available. The four most serious categories (homicide, assault, sexual offences and robbery) together account for some 46 per cent of the cases. Custody owing to drunkenness, either as an offence or protective detentions where it has been decriminalised, comprised 56 cases (15 per cent).

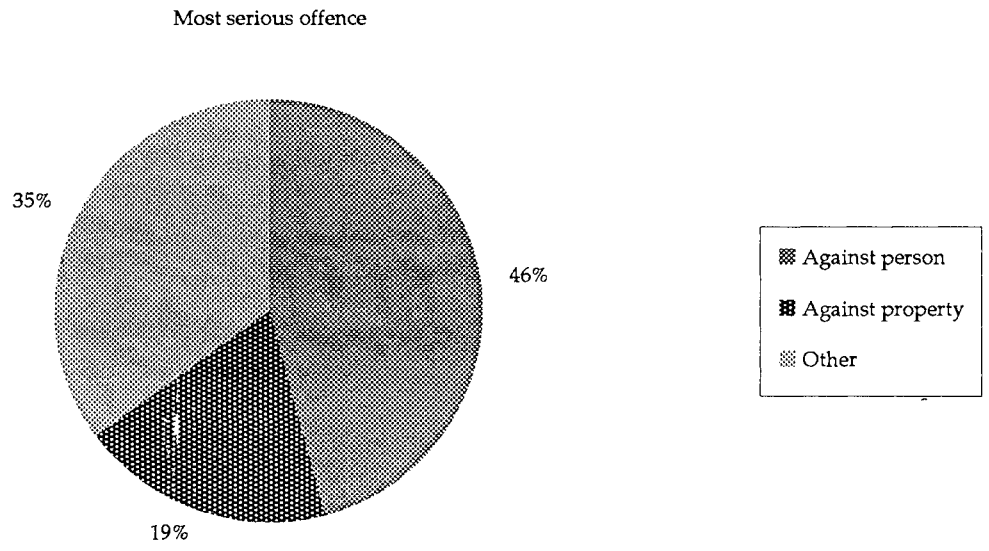
Table 13 also reveals substantial differences, regarding most serious offence, between the prison and police deaths. As one would expect, a higher proportion of the prison deaths were among people incarcerated because of serious offences, especially homicide, robbery, sexual offences and break and enter. By contrast, higher proportions of minor offences, especially drunkenness and protective custody detentions owing to intoxication where it is not an offence, were seen among the police custody deaths. Turning to patterns between the races, we find that little difference existed in offence patterns of those who died in police custody but that, in prison custody, non-Aboriginal prisoners were over-represented compared with Aboriginal prisoners in the offence of homicide (29 per cent compared with nine per cent). The reverse holds for sexual offences, which was the most serious offence category for 32 per cent of the

Table 13 Deaths in custody by most serious offence, custodial authority and race, 1980-88.

	Police		Prison		Total		All
Offence	Aboriginal	Non-Aboriginal	Aboriginal	Non-Aboriginal	Aboriginal	Non-Aboriginal	cases
Homicide	-	2	3	68	3	70	73
Assault	-	4	4	15	4	19	23
Sexual offences	-	1	11	21	11	22	33
Robbery	-	4	1	33	1	37	38
Break and enter	1	4	4	24	5	28	33
Fraud	-	-	-	3	-	3	3
Theft	-	5	2	21	2	26	28
Property damage	-	2	1	3	1	5	6
Justice procedures	4	5	2	12	6	17	23
Drunkenness	10	42	1	3	11	45	56
Other good order	4	3	1	8	5	11	16
Drug offences	-	1	-	15	-	16	16
Traffic offence	-	5	4	6	4	11	15
Other offences	-	-	-	-	-	2	2
Total	19	79	34	233	53	312	365

Aboriginal people who died in custody but for only nine per cent of the people of other races. Figure 2 shows the offence data grouped in another way, differentiating between offences against the person, offences against property, and other offences. Offences against the person, generally the more serious offences, dominate.

Figure 2 Most serious offence as a percentage of all deaths in custody, 1980-88.



Data were sought on the length of time that people were in custody prior to their dying. Unfortunately, Queensland was not able to provide data on this variable, even though 20 per cent of all deaths in custody, during the period under review, occurred there. Overall, data on length of incarceration prior to death are available for 123 of the police custody deaths (68 per cent) and 212 of the prison deaths (76 per cent).

As one would expect, the reported lengths of incarceration prior to death are quite different between the two custodial authorities, with averages in police custody of approximately 15 hours and in prison custody, approximately one year and 10 months.

In police custody, 50 per cent of people died within five hours, and 90 per cent died within 36 hours. Expressed differently, it is noted that, of 123 police custody deaths, 106 died on the first day, 12 on the second day and the remaining five in the period to the tenth day. No significant difference exists between Aborigines and non-Aborigines on this variable, but a temporal pattern is observed with regard to the cause of death. The first few hours of police custody appear to be especially problematical regarding suicide: 33 of the 57 reported suicides in police custody for which temporal data are available occurred in the first three hours of custody.

When reviewing the available data on the lengths of time that people spent in prison custody prior to death, it is noted that, unlike the corresponding police data, the first few hours do not stand out as being particularly dangerous. The distribution is quite skewed, with a mean of 682 days (one year and 10 months) and a median—the point above and below which half the cases fall—of 152 days (five months). Put another way, 135 of the prison deaths (64 per cent of the 212 prison cases for which temporal data are available) occurred in the first year and the balance are spread up to a maximum of 26 years. Only 14 deaths (seven per cent) occurred in the first 48 hours and 23 deaths (11 per cent) occurred after five years or longer in prison.

Differences are observed between the Aboriginal and non-Aboriginal deaths. (Data for this variable are available for 27 of the 35 Aboriginal deaths in prison custody and for 185 of the 245 non-Aboriginal prison deaths.) The prisoners who died in custody after serving more than three years were all non-Aboriginal people. One result of this difference is that the mean period to death of Aboriginal prisoners is 257 days (eight months) with half dying within the first three and a half months. Among the non-Aboriginal prisoners, however, the mean period was 744 days (24 months) with half dying within the first five months.

Data have been provided on the total lengths of sentence being served by 116 people who died in prison custody and who had been sentenced, rather than being held on remand. This is approximately 60 per cent of the relevant cases. The mean 'proportion of sentence served' was 41.8 per cent, with half the cases having served 37 per cent or less of their sentence. Aboriginal and non-Aboriginal people were similar, with means of 39.0 and 42.4 respectively. Nine of the 35 deaths reported to be by suicide occurred in the group which had served between 10 and 20 per cent of their sentences. No other cause of death showed any firm relationship to proportion of sentence served.

The police and prison authorities were asked to advise how long a period had elapsed between the time the person in custody was last inspected by police or prison personnel, and the time of death. This information was provided, in a usable form, for only 135 or 29 per cent of the cases (75 in prison custody and 60 in police custody). Although the high proportion of missing cases means that we must view the data with caution, the data reveal substantial differences between the police and prison

environments. As one might expect, the times between last inspection and death were much shorter in police custody compared with prison custody: means of 1.5 hours compared with 3.2 hours. No differences appear between Aboriginal and non-Aboriginal people on this variable.

The authorities were requested to advise whether the person was alone, or with others, at the time of death. When we exclude deaths in such places as hospital and concentrate on deaths in prison and police cells only, we find that data are available on 227 (61 per cent) of the 371 people who died in these locations (75 of 156 deaths in police cells and 152 of the 215 deaths in prison). As shown in Table 14, similar proportions, overall, of Aboriginal and non-Aboriginal people were alone at the time of death (75 per cent and 81 per cent respectively). In addition, no significant difference exists on this variable between the police and prison custody environments.

Table 14 Deaths in custody according to whether the deceased was alone or with others at the time of death by custodial authority and race, 1980-88.

	Police		Prison		Total		All
	Aboriginal	Non-Aboriginal	Aboriginal	Non-Aboriginal	Aboriginal	Non-Aboriginal	cases
Alone	8	56	13	105	21	161	182
With others	4	7	3	31	7	38	45
Total	12	63	16	136	28	199	227

Not surprisingly, the cause of death is related to whether the person was alone or not at the time of death. Most of the deaths reported to be by suicide (92 per cent, that is all of the Aboriginal suicides and 108 of the 119 non-Aboriginal suicides for which data are available) occurred while the person was alone. Similarly, most accidental deaths (76 per cent) and deaths from natural causes (70 per cent) occurred while the person was alone.

As noted previously, we have been using the shorthand terms 'police custody' and 'prison custody' to identify the agencies which had the legal authority for the custody of the people whose deaths are analysed. We pointed out, however, that not all the 'police' cases died in police cells (in the actual physical custody of police officers), nor did all the 'prison' deaths occur behind prison walls. Naturally, the actual place of death was, in many cases, a hospital or other location. For this reason, we differentiate here between the custodial authority (police or prison administration) on the one hand, and actual place of death, on the other. Significant differences exist between the Aboriginal and non-Aboriginal groups in deaths in police cells and in prisons, as shown in Table 15 below. This reflects, in the main, the distribution of the races between police and prison custody (two-thirds of the Aborigines died in police custody, two-thirds of the non-Aborigines died in prison custody), as outlined earlier.

Table 15 Deaths in custody by place of death and race, 1980-88.

	Aboriginal		Non-Aboriginal		Total	
Place of death	N	%	N	%	N	%
Police cells	42	45	114	31	156	34
Prison	23	24	192	52	215	47
General hospital	24	26	51	14	75	16
Psychiatric hospital	1	1	8	2	9	2
Other	4	4	3	1	7	2
Total	94	100	368	100	462	101

A higher proportion of Aborigines, compared with non-Aborigines, died in general hospitals; this pattern was seen in the deaths in each of the custodial environments. Some 24 per cent of Aboriginal deaths in police custody actually occurred in a general hospital, compared with only 6.5 per cent of the non-Aboriginal deaths in police custody. Similarly, 31 per cent of the Aboriginal prison deaths occurred in a general or psychiatric hospital compared with 21 per cent of the non-Aboriginal prison deaths. These differences, it is suspected, are due to the fact that the very existence of the Royal Commission has brought to notice more cases of Aboriginal deaths in custody than otherwise would have been the case. As one might expect, most (97 per cent) of the deaths from hanging occurred in police and prison cells while the deaths from the other main cause of death, natural causes, occurred roughly equally in police cells (27 per cent), prison (33 per cent) and general hospitals (36 per cent).

Overview of trends

The total number of deaths in custody for each year from 1980 to 1986 remained fairly consistent, but in 1987 there was a dramatic and unexpected increase in the number of deaths in both prison and police custody and among both Aborigines and non-Aborigines. The 1987 figure of 93 deaths is more than twice the annual mean for 1980-86 of 44 (see Figure 3). In 1988, the number of deaths remained higher than the 1980-86 average but was not as marked as the 1987 figure. Such a striking and unexpected increase as occurred in 1987 must be explored as carefully and comprehensively as possible.

As indicated by Table 16, for both police and prison custody in 1987 the number of deaths increased at approximately the same rate (by factors of 2.4 and 2.0 respectively) over the annual average for 1980-86. It is interesting to note that both Aboriginal and non-Aboriginal deaths increased at approximately the same rate (by factors of 2.4 and 2.0 respectively) in 1987 and that these increases were observed independently in the two types of custody. In police custody Aboriginal deaths increased at a higher rate than non-Aboriginal deaths (3.1 and 2.1 respectively); conversely in prison custody, non-Aboriginal deaths increased at a higher rate than Aboriginal deaths (2.0 and 1.5).

Figure 3 Deaths in custody by race, 1980-88.

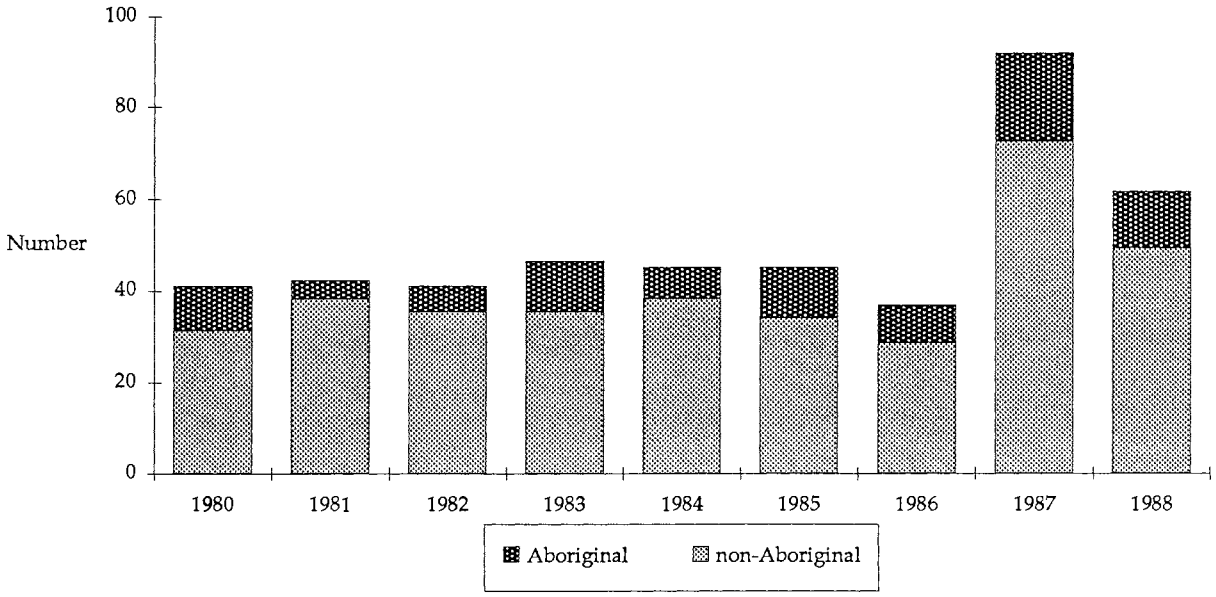


Table 16 Deaths in custody by year, custodial authority and race, 1980-88.

	Police			Prison			
	Aboriginal	Non-Aboriginal	Total	Aboriginal	Non-Aboriginal	Total	All cases
1980	5	7	12	5	25	30	42
1981	3	12	15	1	27	28	43
1982	4	15	19	3	21	24	43
1983	6	10	16	6	26	32	48
1984	3	12	15	4	27	31	46
1985	7	15	22	4	20	24	46
1986	8	13	21	1	16	17	38
1987	16	25	41	5	47	52	93
1988	7	14	21	6	36	42	63
Total	59	123	182	35	245	28	462

From Table 17 it can clearly be seen that the overwhelming majority of deaths over the period 1980-88 were male. The number of male deaths was fairly consistent between 1980 and 1986, with an annual mean of 41.6. In 1987 this rose to 90, representing an increase by a factor of 2.2. This increase was reflected in both Aboriginal and non-Aboriginal males, with the increase being slightly higher among Aboriginal males. The increases were by factors of 2.7 and 2.0 respectively over the annual average for 1980-86.

The number of non-Aboriginal males who died in prison custody increased from an annual mean of 22 for 1980-86 to 45 in 1987. This increase did not occur among Aboriginal males in prison custody. In police custody, however, the rate of death for Aboriginal males increased substantially from an annual average for 1980-86 of 4.6 to 16. It is important to note that the number of deaths among non-Aboriginal males in police custody also increased but at a lower rate (2.1 compared with 3.5).

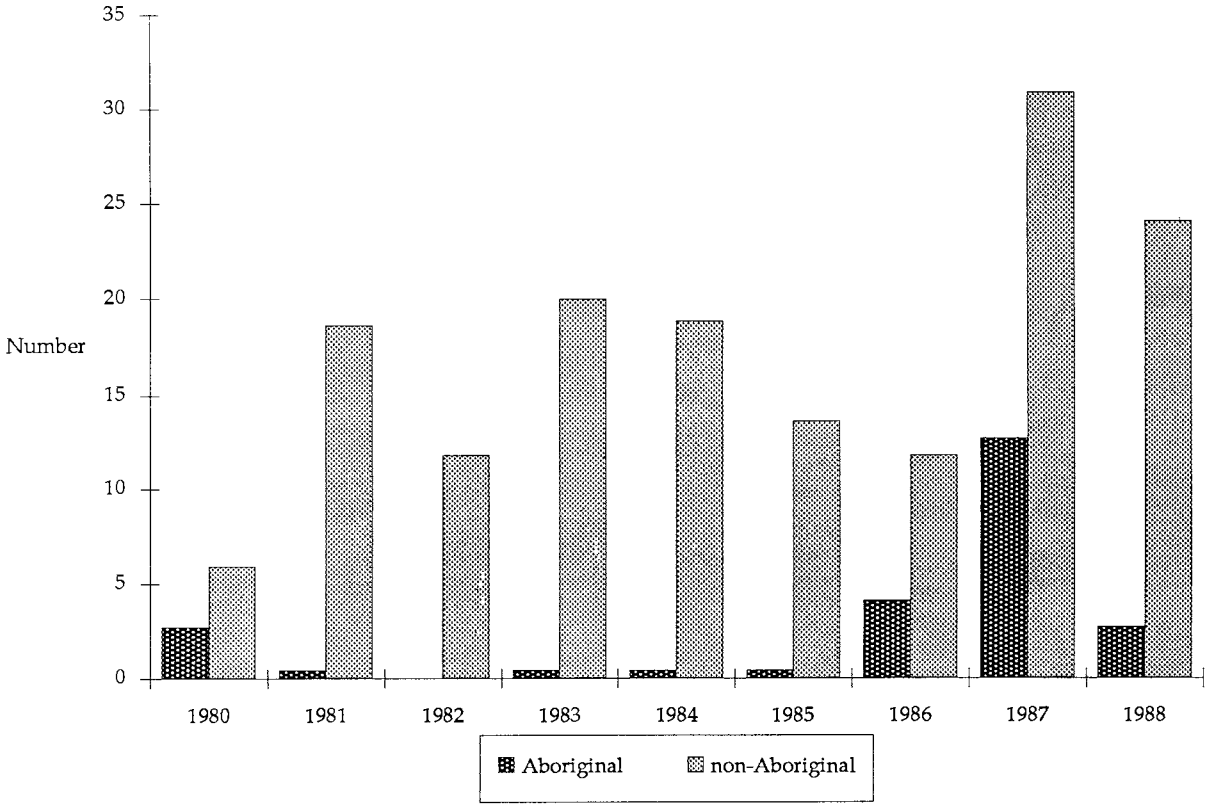
Table 17 Deaths in custody by year, sex and race, 1980-88.

	Aboriginal		Non-Aboriginal		Total	
	Males	Females	Males	Females	Males	Females
1980	9	1	32	-	41	1
1981	4	-	38	1	42	1
1982	5	2	32	4	37	6
1983	11	1	34	2	45	3
1984	7	-	39	-	46	-
1985	11	-	33	2	44	2
198	8	1	28	1	36	2
19	21	-	69	3	90	3
1988	11	2	49	1	60	3
Total	87	7	354	14	441	21

Table 18 Deaths in custody from hanging and all other causes, by year and race, 1980-88.

	Aboriginal			Non-Aboriginal			
	Hanging	All other	Total	Hanging	All other	Total	All cases
1980	3	7	10	6	24	30	40
1981	1	3	4	19	18	37	41
1982	-	7	7	12	21	33	40
1983	1	11	12	20	15	35	47
1984	1	6	7	19	20	39	46
1985	1	10	11	14	21	35	46
1986	4	5	9	12	15	27	36
1987	13	8	21	31	37	68	89
1988	3	8	11	24	22	46	57
Total	27	65	92	157	193	350	442

Figure 4 Deaths in custody from hanging, by year and race, 1980-88.



It is noteworthy that only 21 deaths in custody were female, seven Aboriginal and 14 non-Aboriginal. The highest number of female deaths occurred in 1982, when there was an annual total of six, and no female deaths were recorded in 1984. It is also worth noting that there was no significant increase in female deaths in 1987 compared with the annual average for 1980-86. The focus of attention must therefore be on the increase in deaths of males in 1987, both Aboriginal and non-Aboriginal, and in both police and prison custody.

The most dramatic aspect of the 1987 data is the increase in the number of deaths due to hanging among Aborigines in police custody. Aboriginal deaths by hanging increased from an annual average of 1.6 to 13, representing an eight-fold increase. Before 1987, very few Aboriginal deaths in custody were by hanging. In 1987, however, 13 of the 21 Aboriginal deaths (62 per cent) were attributable to hanging (see Table 18). The number of deaths by hanging among non-Aboriginal males also increased, albeit at a lower rate in 1987 from an annual average for 1980-86 of 15 to 31 (a factor of 2.1). However, unlike the Aboriginal deaths, there was no difference in the proportion of deaths by hanging in 1987 when compared with the average proportion for 1980-86. In other words, the increase in Aboriginal deaths in 1987 can be solely attributed to an increase in the number of hangings among Aboriginal males. This is not the case, however, for non-Aboriginal males where deaths from natural causes also increased at the same rate as hanging in 1987.

The number of deaths in custody increased among the younger age groups up to 45 years, as shown in Table 19. Beyond this age group there was no change in the number of deaths over the years. Of particular concern is the large increase in deaths among the 15 to 19 years age group in 1987 and 1988. The number of deaths among this age group increased in 1987 from a 1980-86 mean of 2.9 to 12, a four-fold increase. This increase was highest among Aborigines aged 15 to 19 who died at a rate of 8.3 times the annual average for 1980-86. Almost all of these deaths occurred in police custody (four out of five deaths) and all were reported suicides by hanging. Conversely, all of the seven non-Aboriginal deaths in this age group occurred in prison custody in 1987. This is a four-fold increase over the annual average for 1980 to 1986. Six of these deaths were also by hanging. It is important to point out that suicide accounts for almost all of the increase in deaths among 15 to 19 year old Aboriginal and non-Aboriginal males; of the 12 deaths in this age group in 1987, 11 were by hanging. This is also the case among the 20 to 24 years age group, where hangings accounted for 81 per cent of the deaths in 1987.

A substantial increase occurred in the 35 to 39 years age group. Deaths in this group increased from a 1980-86 annual average of 2.6 to 13 in 1987, representing an increase by a factor of five. This increase was similar for both Aboriginal and non-Aboriginal deaths in 1987 (by factors of 5.0 and 5.2 respectively). It is also worth noting that deaths in the 45 year and above age group showed little change over the nine year period. With the exception of a decline in 1986, the number of deaths in this age group has remained consistent even in 1987 and 1988.

Table 19 Deaths in custody by year and age group, 1980-88.

	Age in years										
	15-19	20-24	25-29	30-34	35-39	40-44	45-49	50-54	55-59	60-64	65+	Total
1980	2	10	5	5	4	5	1	5	1	3	1	42
1981	2	8	9	5	1	5	2	6	2	2	1	43
1982	3	8	10	4	1	3	3	6	3	1	1	43
1983	3	12	11	6	2	3	2	2	2	2	3	48
1984	4	7	11	8	2	3	23	3	3	2	1	46
1985	3	6	13	3	4	1	4	1	2	2	7	46
1986	3	3	9	4	4	11	1	-	2	1	-	38
1987	12	18	16	10	13	10	4	3	3	1	3	93
1988	6	14	12	9	4	6	4	1	5	-	2	63
Total	38	86	96	54	35	47	23	27	23	14	19	462

Table 20 Deaths in custody by jurisdiction, year and race, 1980-88.

	NSW	VIC	QLD	WA	SA	TAS	NT	ACT	AUST
Aboriginal									
1980	1	-	1	4	1	-	3	-	10
1981	1	-	2	1					4
1982	1	2	-	4					7
1983	2	-	-	5	5	-	-	-	12
1984	1	-	2	3					7
1985	1	-	3	5	-	-	2	-	11
1986	2	-	4	-	1	1	1	-	9
1987	3	1	10	4	2	-	1	-	21
1988	1	-	2	7	1	-	2	-	13
Total	13	3	24	33	10	1	10	-	94
Non-Aboriginal									
1980	8	4	10	4	6	-	-	-	32
1981	11	9	8	6	4	1	-	-	39
1982	9	7	9	4	5	2	-	-	36
1983	13	11	7	3	1	1	-	-	36
1984	14	13	7	5	-	-	-	-	39
1985	12	7	7	3	3	-	3	-	35
1986	12	4	8	1	2	-	2	-	29
1987	26	26	6	4	5	2	2	1	72
1988	16	16	9	5	1	3	-	-	50
Total	121	97	71	35	27	9	7	1	368
Persons									
1980	9	4	11	8	7	-	3	-	42
1981	12	9	10	7	4	1	-	-	43
1982	10	9	9	8	5	2	-	-	43
1983	15	11	7	8	6	1	-	-	48
1984	15	13	9	8	-	-	-	-	46
1985	13	7	10	8	3	-	5	-	46
1986	14	4	12	1	3	1	3	-	38
1987	29	27	16	8	7	2	3	1	93
1988	17	16	11	12	2	3	2	-	63
Total	134	100	95	68	37	10	17	1	462

As can be seen from Table 20, the most noticeable change over the 1980 to 1988 period within Australian jurisdictions occurred in 1987 in New South Wales, Victoria and Queensland. The number of deaths in all other States and Territories varied from year to year but remained within a small range. In contrast, in Western Australia the total number of deaths each year was reported as being either seven or eight, except in 1986 and 1988. In 1986 only one death was reported in that State for the year, while in 1988 the number increased to 12 from an annual average for 1980-87 of

seven. It is noteworthy that, in Western Australia, the number of deaths in 1987 was similar to that of the earlier years of the decade, even though it has more Aboriginal deaths to be investigated by the Royal Commission than any other State.

In New South Wales, the number of deaths per year between 1980 and 1986 fluctuated between nine and 15. In 1987, however, the number of deaths increased from an annual average for 1980-86 of 12.6 to 29 (a factor of 2.3). A similar pattern was seen in Victoria, where the number of deaths for 1987 increased from an annual average for 1980-86 of 8.1 to 27 (a factor of 3.3). These increases can be attributed almost entirely to non-Aboriginal deaths, which accounted for 26 out of the 29 deaths in New South Wales in 1987, and 26 out of the 27 in Victoria that year. Non-Aboriginal deaths increased in both police and prison custody in New South Wales by 2.6 and 2.2 respectively and in Victoria by 3.5 and 3.2.

There was a different pattern in Western Australia and Queensland with regard to Aboriginal deaths. They increased in Queensland from the 1980-86 annual average of 1.7 to 10 in 1987 (ie by a factor of 5.9). In Western Australia no increase in Aboriginal deaths occurred in 1987 compared with the 1980-86 average, but the 1988 figure of seven deaths was twice the annual average for 1980-87. The 10 Aboriginal deaths in Queensland in 1987 comprised seven deaths by hanging and three by natural causes. Of these, eight occurred in police custody. The deaths by hanging of young Aboriginal males in Queensland contributed substantially to the national increase in Aboriginal deaths in 1987.

NOTES

1. Caution must be exercised in the interpretation of statistics related to reported suicides, self-inflicted deaths or death by accident. The terms suicide or self-inflicted death are used interchangeably in this chapter as it is often impossible to know whether a death reported to be suicide was in fact intentional and therefore legally classifiable as suicide. Furthermore, the reported cause of death as suicide or accident may not necessarily correspond with the findings made by Commissioners in their reports on the inquiries into deaths of Aborigines in custody.
2. This chapter is an edited version of a paper produced by the same authors under the title 'Australian Deaths in Custody 1980-88: an analysis of Aboriginal and non-Aboriginal deaths in prison and police custody' and released in July 1989 by the Royal Commission into Aboriginal Deaths in Custody as Research Paper No 7. Any opinions expressed in this chapter are not necessarily endorsed by the Royal Commission.
3. It has become apparent during the course of this and other Royal Commission projects that the concept of death in custody is an amalgamation of two quite different phenomena: deaths in prisons and deaths in police custody. Different considerations apply to each and, to that end, it is proposed to produce supplementary papers to this which will focus in more detail on police and prison deaths. These papers may provide a basis for the development of different policies for the prevention of

deaths in different settings. The primary focus of this chapter, however, is on differences between Aboriginal and non-Aboriginal deaths rather than on differences between police and prison deaths.

4. It is hoped that the proposed research papers covering deaths in police and prison custody separately will include analyses of age-adjusted death rates in these environments. This will be possible in the case of prison deaths where the denominators (number of prisoners) are known from annual prison census data. Unfortunately, similar data are not available on the police custody population.

5. For remand rates, see Australian Prison Trends, published monthly since 1976 by the Australian Institute of Criminology, Canberra.

DISCUSSION

The issues which have been raised in discussion so far:

1. Antenatal care; its role in differentiating perinatal outcomes; lack of improvement in stillbirth rates; 'culturally acceptable' services; perpetuation of birth disadvantage; later effect of birthweight.
2. The pattern of high mortality in young and middle adulthood (25 to 50 years) across Australia; ischaemic heart disease and other circulatory system disease; diabetes; accidents/violence; what works to alter the pattern? (differences between communities; interactions between institutions; Aboriginal community control); risk factors. [Some of the ensuing discussion refers to the last issue, risk factors, as Point 6.]
3. Inadequate data collections; role of ABS (health surveys, mortality data; who pays?); bringing the available information to government and public notice; informing Aboriginal communities.
4. Need for skilled critical analysis of existing data; uniformity and soundness of analysis.
5. Do we need a prospective (cohort) study (15 to 20 years)?; interventions or events?

Mr G Briscoe I want to raise the point that I raised yesterday, which you have sneakily put back in your list, and that is the question of culture. I think that we can be a bit more specific about what it is we are talking about in that regard. Now instead of using the phrase 'culturally acceptable services', we can still use something we know about. Perhaps we could still use the word 'culture' but make it less prominent, so we say something like this—'social or cultural strategies for improving Aboriginal acceptability of health services'.

Now some people may have problems with restricting this to health services, but it is, in my view, a theoretical question of social equality, of distribution of scarce resources. I like flowery language just as much as anyone else, but the point is that once you start bringing in these vague terms, it gives the onus of decision to people whose lives are not affected one way or the other, like a policeman who arrests someone for a triviality and the poor bastard goes into the clinker and ends up dead. So it is a question of taking these people who are not accountable out of the system.

What I would like to do is perhaps change those words 'culturally acceptable services' which can take away the prospect of equality of services, of accessibility to services, by just tightening up that content in the way I suggested. I am open to suggestions on how it ought to be done, but I am suggesting that it ought to be done and that those words ought to be changed.

Dr L Smith There should be a mention of risk factors or predictors of mortality, but perhaps it is already implied in some of things you have listed in Item 2, and it seems to me that it is a concern that alludes to them. I wonder if we have a consensus actually about Item 6? I think probably what Aileen Plant is talking about is that we not only know the risk factors but we know what to do about them. In other words,

there is a sort of menu of interventions that are available at this time. The sense I have from some of the discussions I have been involved in, is that perhaps there is not a consensus, even in this group, about what those interventions might be.

Dr A Plant We do actually count where the action is and what time it is occurring, and we know quite a lot of detail about the excess mortality and for many of those causes we do know the dominant causally-related factors. Then it is a difficult problem of how you get people to hospital and adequately treated for pneumonia, for instance. Nonetheless, I think we do have a lot of information about points for intervention.

Dr A Gray We have visions of all 35 to 39 year old Aboriginal men having triple bypass surgery as preventive medicine. Surely we have to go back a lot further than the clinical intervention.

Dr A Plant No, I think that preventive medicine is more important than pure intervention. We have a lot of information and there are a lot of people who misuse it now. I think the truth of the matter is that we had much higher mortality rates from heart disease back say 20 years ago than we have now. We have managed to change things. We know some of the things that need to be done.

Dr L Smith How is that to be tackled? It seems to me that, if indeed that is true, we need some sort of consensus statement about what is to be done about Aboriginal mortality. Let me pose a few questions: for example, for what proportion of the population is the sort of dietary change that is being suggested a sensible option, in any real world situation? How many people in Australia are able to go onto a diet of kangaroo meat, oysters and what have you? In some senses you can say that we know what to do, because that has been demonstrated, but in a practical sense that has got to be translated into the sort of dietary change that can occur in suburban Sydney and outback New South Wales as well.

I do not think that there are any pointers there at all in a preventive sense for the whole Aboriginal population, even though in principle you can say it is all solved, that we know what to do about it. That is talking about primary prevention; when you start to talk about secondary and tertiary interventions, it seems to me that perhaps it is easier to talk in terms of what you would do with any patient with that sort of problem—but again, is talking about Aboriginal health in the same way we would talk about any patient a practical way of dealing with the public health problems that we face? I would have thought that if that was the case, there would be no problem if every Aboriginal person is just a patient like any other patient. So, in a sense what you say is true and unarguable, but on the other hand it seems to me that at a population level the answers are still really not there.

Dr A Plant I said it is not easy, but there is no question that there has been a turnaround in things like smoking rates, and I think it is iniquitous if we do not say we have heard here that up to 90 per cent of Aboriginal people are smoking. Now we must target that and point that out as a place where we can change people's behaviour. It is always difficult to change people's behaviour, it is difficult to change

our own behaviour, but it does not mean you should not try, and it does not mean that there has not been some history of success in other groups.

Dr R Streatfield I agree with what has just been said, and also with what Len Smith is saying, that we have to be careful that we do not infer that a 'blame the victim', clinical risk factor approach dominates the thinking. What I find exciting about the discussion is that we have identified where the clinical problem appears to be, or at least where the pathological change in the population is most likely, and we have identified that some population change in a direction that we know to be likely to prevent diabetes, obesity, heart disease—that trio—is likely to be in the long-term effective. What we have no ideas about at the moment is how to introduce that into the population environment in a way that is culturally acceptable and is likely to be practical.

Dr N Thomson It is hard to agree with what everyone has said, but I guess I did agree with Len Smith's concerns about how you put it into practice. I also agree with Aileen Plant that there are options that can be done straight away. I think that one has to look at how they are applied in a very intelligent way. There are a couple of anti-smoking campaigns, but at the same time, there are other things which we could be doing which we are not doing. They may not be reflected in mortality, but certainly in aspects of morbidity and potential mortality.

Look at rheumatic fever, for example: there are intervention strategies, as outlined by Ric Streatfield already, that have been taken in some parts of the country in intensively trying to do primary prevention of rheumatic fever. They are underway, but they are not underway throughout the country, so rheumatic fever is still occurring in other parts of Australia. I think that Hepatitis B is another example, and you could go on. There are a lot of things there that could be done. There is hookworm; it does not exist in North Queensland now. Why? Because it was eradicated about 30 to 40 years ago. If you go to the Kimberleys, there is a bloody lot of hookworm. I really do not think there is an excuse for us to go on, year in, year out, when we have known for ages that hookworm in this wealthy country can be eradicated by simple means. And roundworm is another one that we should just get rid of straight away. It is very easy and very safe.

Dr L Smith The thing about hookworm eradication is that it involves some fairly direct and authoritarian methods. That is one of the things that we have got to face up to, that public health is about doing things to people and making things happen. There is always a danger of being paralysed by the sort of non-interventionist liberal concerns about not doing things to people, waiting until they decide to do it themselves.

Dr A Gray I did not write that one down because I think it is fairly obvious that we would have a lot of difficulty getting a consensus on that. The emphasis on community control over health care is very strong in the protocol that the NH&MRC has been circulating recently about research in Aboriginal communities. Mind you, I think that this emphasis, although long overdue, overlooks the fact that there are

public health concerns which cannot possibly arise at a local community level, because they are not really noticeable at a local community level. There is another level and it does possibly involve some kind of acceptance of authority in public health, but I am certainly not proposing that we should write that down as something we ought to be doing at this stage.

Dr R Streatfield Historically, I think, we had it easy. It had nothing to do with me, but the way things were done in Queensland in the old days was rounding people up and doing it. That is how we find it so easy to screen children. We ask the community and they are so used to having it done that we have no problems. If we went into the Northern Territory and did the same thing, I imagine we would have no end of problem in getting cooperation when a habit is not built up. The same thing happened with the worms, the hookworm and roundworm control. It was done routinely every three months. But we cannot do that any more in Queensland and neither should we, and with our trichuriasis eradication program—which I don't fully agree with, because I think it is impossible with the existing sewerage deficiencies up in some of our communities—it was taken to the community and put to them in a way that they agreed to have it done because they could see the problems.

I am an optimist but I think we can go the community-involved way. All it takes is us putting it to the community in such a way that they can see that it is to their obvious advantage. I think we should do that all the time. That is the only way that we have got our rheumatic fever program going. Rheumatic fever must be one of the hardest diseases to describe to someone, because there are so few people affected and it is such a difficult thing to explain, how your sore throat damages your heart. But we managed to do that and I think that if we managed to do that and get the community on side, then we can convince them about losing weight, jogging and all that sort of thing.

Dr N Thomson There is one thing I wanted to say relating to Item 6 and I guess to Item 1. We do know a lot about Aboriginal mortality and what to do about it, but my warning would be that in fact if we tied Item 6 and Item 1 together and looked at, say, infant mortality as a specific example of Aboriginal mortality, I do not think we really know what to do to reduce it to the level we want.

The experience of reducing low birthweight is an example. It has not changed. I did not say it yesterday, but if you fiddle around with Australian data about low birthweight babies and look at the excess mortality just on an Australia-wide basis, the mortality of those low birthweight babies is 40 times that of non-low birthweight babies. So if you just play a statistical game you could come up with an estimate that Aboriginal infant mortality will remain about 18 or 19 per 1,000 based on that low birthweight percentage.

I do not agree with the implication of Item 1 because we do not know if antenatal care is going to do that *per se*. It is probably going to come up in those more general things tomorrow, that we do not know precisely how to handle it. The issue is that we have a relatively high infant mortality tied up with a low birthweight, and I do not think that it is such a simple linkage.

Dr A Gray Bob Douglas asked a question this morning about possible connection between diabetes and early onset of heart disease or circulatory system disease. It is certainly not clear from the mortality data because diabetes as a cause of death tends to be in the older age groups, much older than those where circulatory system disease gets mentioned. So that connection is probably just overlooked by doctors writing down causes of death.

The last slide [by Dr R Streatfield] was also very important, emphasising the causes in lifetime nutrition patterns. That is one of the things about which there has only been speculation in the past, as well. This is the type of material which I would actually like to see a lot more research done on because it clarifies that issue of causation of circulatory system disease, of at least one type, quite nicely.

Dr K Streatfield The pattern is very clear. Aileen Plant emphasised that circulatory diseases are a major problem, and she made the point that now that we know this, in some ways we do not need to continue to document it. The more interesting areas are the intervention problems. That raises the issue that Ric Streatfield mentioned, where health staff at Lockhardt River alert people to the serious nature of the consequences of circulatory diseases, in heart attacks in young men. But the Lockhardt River community refused to participate in what you saw as a possible intervention or what would lead to an intervention.

I would like to put in a plug for not purely a medical focus in this, but investigation into the reasons why some communities will accept a particular intervention and others will not, and put much more emphasis on documenting the kinds of interventions that have been tried. Why are they not acceptable in some environments and acceptable in others, and what are the factors that underlie acceptability of these kind of interventions in the programs that people are trying? You [Dr R Streatfield] have mentioned one that you are trying but maybe we should be documenting what other people are doing as well, not just the mortality patterns but the interventions.

Dr N Thomson I am very delighted to see estimates that we can have across the country. My concern is with comparisons between Northern Territory and Australia-wide figures. I took the opportunity of looking at Aileen Plant's thesis, which was based upon recorded age-specific mortality in Northern Territory for that period of time.

When you compare the Northern Territory estimates for 1964-68 with those for 1979-83, that difference between female rates that you [Dr A Gray] introduce by the model certainly does not appear to be reflected in a real sense in reported deaths. One of the worries that I have is, if one draws conclusions from models like that when we actually have good data, in certain cases like the Northern Territory, we may be drawing the wrong conclusions by comparing model data with what has been reported in the past.

The interesting thing that I noted was your estimated age-specific mortality for females based on your model. For the young group, they are certainly well under what actually was documented by Aileen Plant for the 1979 to 1983 period. I think that a diagram of Aileen's estimates would show the decline in female mortality at infant

and young childhood ages, but I do not think it would show the decline that your model shows for the young adult years, certainly for the age-specific data that is recorded here.

Your age-specific mortality estimates for females in the Northern Territory are very similar to all the other places, whereas the actual reported deaths, which I looked at very carefully over a slightly earlier period, showed a substantially higher age-specific mortality, particularly in the young female years. By age 50 to 54, they were really very similar. I think the difference you are highlighting between 1964-68 and the more recent period in the Northern Territory is a bit of an artefact of the model.

Dr A Gray Some of the fluctuations in actual data can be just due to random influence.

Dr K Streatfield Alan [Gray], you implied that in 1981 there was something like a 30 per cent under-enumeration in New South Wales only. What about the other States, say the Northern Territory? If there was anything like that kind of under-enumeration in the Northern Territory then you would get overestimation. You would get overestimation of the rates if you calculated rates from reported deaths and an under-enumerated population.

Dr A Plant I used the 1981 Census. The other thing of course is that in the 1985 health indicators publication [Northern Territory Department of Health], at least in the initial version, they did not specify what their source population was nor what they did with deaths that occurred, say, in Alice Springs from South Australian people or people from the Northern Territory who died in other places, whereas I went over that quite carefully. Now, in 1985 they might have changed the way they determined which deaths to include. Of course, differences in the determination of deaths would also give you different rates, so the numerator could be different as well as the denominator.

Mr N Khalidi I have a problem in understanding Aileen Plant's methodology. In the part where you were talking about the potential years of life lost, the estimates were calculated by using the life expectancy that would have been expected at mid-point of each age group. From the paper or from your presentation I failed to understand this point, as my understanding of expectation of life is that it is the expectation of life at exact ages zero, one, five, 10, 15 and so on. Could you please explain how you did the calculation for the mid-points of age intervals?

Dr A Plant I took the mid-point of the age group, then took the expectation of life at that point. You can read it straight off the life table, for example at the age of 62 life expectancy is say 20 years, something like that.

Dr K Streatfield If I could ask David [McDonald] about the duration that people have been in custody? About two-thirds of the Aboriginal deaths in custody were in police custody, not in prison. You said of those about two-thirds died within the first three hours of custody. Now, I wonder whether you are prepared to say much more about

how you think this happened. To me this is pretty amazing, that overall a third of these deaths in custody occurred within three hours. These are hardly likely to be natural causes. Would you like to say a little bit more about how you think this happens and whether there is any kind of mass hysteria in this, or whatever term you might like to use, and about the fact that it peaked in 1987, as it did in other countries, and then dropped back again? Would you like to interpret that?

Mr D McDonald We are talking about deaths in police custody, not in prison. There was no temporal factor of any interest in prison deaths. In police custody the high rate of deaths is in the first few hours of custody and they are almost all deaths by hanging. That is the first point, that they are deaths by hanging.

Do we have any information or speculation about the reasons for or the patterns of those deaths by hanging? We know that a very large proportion of those people are in custody because of intoxication, and we know that there is a relationship between withdrawal and psychiatric syndromes of depression and anxiety. Hunter is speculating quite a lot from his preliminary data about that. So there is an issue there.

There are other issues though that are very much culturally oriented, about young Aboriginal people who have been treated, who have come from a very violent situation, say a fight, a brawl, drunkenness, picked up by police, dealt with in a very harsh physical manner, processed rapidly into police cells and who then have a response, a psychological reaction of immense anxiety, which is then brought forward and seen in terms of internally directed anxiety or suicide attempts. So that is a line of argument.

We also know that for quite a lot of Aboriginal people, being in custody, being in a police cell, is a particularly frightening experience because being alone is an unusual thing and being alone at a time of stress is a very dangerous thing. I would suspect the Aboriginal people in this room would confirm that it is frightening to a greater degree than for non-Aboriginal people. By the nature of the family and kinship systems of Aboriginal people, there are always helpers who are around at a time of stress and so during that period in the cell when somebody is very frequently intoxicated, is under immense stress, it is a situation of violence. The anxiety level is immense.

So they are the kind of patterns that we think exist. We hope that through the actual enquiries being conducted by the Commissioners into individual deaths, more of those will become clear. Insofar as that is true, it leads to suggestions for prevention, for monitoring and surveillance and care of people to reduce and minimise the stress. I have said that almost everybody who gets into a cell has gone through a period of behaviour which is pretty violent. The vast majority of the Aboriginal people who are in police cells have come in as a result of altercations on the street or as a result of drunkenness. The police treat all such cases basically in the same way. About 60 per cent of the Aboriginal people who are in the cells go in because of drunkenness.

Mr G Briscoe On whose data?

Mr D McDonald On police data. Well, that is a problem but it is the only source that we have and it certainly fits with my experience in the Northern Territory.

Mr G Briscoe What research has been done on the kind of people who run these institutions?

Mr D McDonald That is really why we have the Royal Commission, isn't it? It is certainly alleged that a lot of those people have been badly maltreated by police officers. I think every enquiry that is taking place is studying in great detail what has happened to that individual, in fact the person's whole life, particularly the antecedents of the death. This is coming through in the enquiries, where we are finding that somebody is found intoxicated, unconscious, on a street, is left there till a patrol car comes around next time, is picked up, put in the car, left there for half an hour, taken to the cell, hosed down because the person is dirty, temperatures are 12 or 15 degrees, left in the cell. So, those kind of patterns are coming forward in evidence and will be collated later on to try to fill in some of those gaps about the nature of the external aspects of it, as well as the human responses to the custodial environment.

Dr K Streatfield In situations like this, you just cannot help feeling slightly sceptical about the police version of the story.

Mr D McDonald One of the major reasons that we have a Royal Commission is because the Aboriginal community rightly said there are far too many of these deaths. Sure as hell, many of them are being caused by either actual neglect or worse on the part of custodial officers. That is why we have a Royal Commission, to try to find that out. The Royal Commission is the most powerful technique that our legal system has to find out such things.

Mr G Briscoe While you are getting data that is being collected by the police, the whole thing seems to backfire. First in the sense that the real victim is said to have a suicidal nature and second that the victims are the ones that are said to be the real cause.

Mr D McDonald The data we have presented in the paper that I am discussing have come from police and prison sources, I grant you that. When the Commission's final report comes out, using different sources not what police and prison officers say, but rather what is found through the enquiries, particularly the work of Aboriginal field officers out with the families and with the people who are actually in the field at the same time, many of the actual causes of death will be changed from the information that we have got to date. So what we have presented now is the best information available at present.

Mr G Briscoe How is this other information being handled? Is it simply going to be a narrative explanation, or is there going to be some sort of scientific analysis?

Mr D McDonald The Commissioners are producing what you described as a narrative explanation, literally a book that describes each death. What we are doing then is going through and pulling out some facts and figures that are similar to the kind of facts and figures that I have presented in this paper, but we will correct them and update them to give more accurate sources of information than police and prison officers' own reports.

Dr R Streatfield Natural causes could mean subdural haemorrhage just being accepted. That to me is not natural causes.

Mr D McDonald 'Natural causes' is a legal term that is used by Coroners, and a very large number of them are in fact subdural haemorrhage as a result of fights, or worse, bashings. Yes, absolutely. That is one of the benefits that will come from exactly the point that we have been discussing here. If we find in fact that the external cause was the police bashing a bloke it will come out as 'natural causes' but go into some other classification, depending on the outcome of the Commission. [Natural causes] includes heart attack, respiratory illness, pneumonia, those sorts of things. We have defined it in the paper. Remember we are working to a different audience from epidemiologists. We are talking about diseases and things.

Mr G Briscoe Instead of using the abstract term 'natural', it should be broken down to much more concrete form.

Mr D McDonald As I said in my initial presentation, we are using the term 'natural causes' to mean all causes other than ICD-9 codes for external causes. But that again is not a useful way of communicating to the public.

Ms C Brown I have an observation about [Alan Gray's] paper, about early childhood mortality in South Australia being high. From checking out the accuracy of birth registration records we meet matters of definition of an Aboriginal baby, which can be the child of either an Aboriginal father or a mother. In 1988, the official position of the pregnancy outcome unit of the Health Commission was that there were about 330 Aboriginal births because they were children to an Aboriginal mother. We are calculating mortality based on that denominator. When we checked out births to an Aboriginal father as well the figure was nearer to 500. Then my point is: what is an Aboriginal child or an Aboriginal baby?

Dr A Gray Generally speaking, we have tended to pass over these problems of definition of the Aboriginal population in this workshop so far. It is always good to bring it up, to remind us that the data that we are dealing with is always going to be fuzzy around the edges in some way, because of definitional problems, and there are always going to be hazards in comparing data on recorded vital events, whether births, deaths or marriages, with census data used as a population base—even when we have, as happened much to the credit of ABS, no doubt due to its far-sighted planning of the 1986 Census, what appears to be a very good census enumeration. I hope that the 1986 standard that was set continues on in future censuses so that we can more readily do

these intercensal comparisons. Even with the best of information from the census, with what we think is good vital information, we are going to have those problems of fuzziness between the numerators and denominators of the rates because they come from different sources, and we are never going to be quite sure what they mean anyway.

Dr G Durling Certainly in the Northern Territory perinatal register our criterion is the mother's ethnicity, not the father's ethnicity. So if the baby is black but the mother was white it's classed as a Caucasian because the only data we have on the form is maternal ethnicity. I do believe that is the practice with all perinatal registers.

Dr A Gray It is sensible to identify the Aboriginality of the mother in a perinatal data collection because it is the risk factors related to the mother that are important. In that case it is the mother's Aboriginality that we are interested in.

Mr G Briscoe Throwing measures around, the way you are throwing your measures and rules around, you will probably find yourself outside. The definition of Aborigines has changed, that is true. It was first defined by Charles Rowley; then subsequently, because he got it wrong, it was changed again at some point, in the 1970s. This just shows the inflexibility not only of people in the State, but people in institutions as well. You can have somebody in Darwin making changes and adjustments convenient to his own feelings. He decides what race that baby is, now after he has decided that, five years up the track the child itself decides that the race has changed again. We have to take our benchmarks or our rulers seriously. I mean it is not just a matter of apologising for the ABS.

Dr A Gray I was not apologising for the ABS, I was saying that what we are interested in, in a perinatal data collection, is the characteristics of the mother, not the baby. We are interested in whether the baby lives or not and we are interested in what it weighs, but it is the characteristics of the mother [that are important] because the risk factors are associated with the mother. So that is why we identify the mother as Aboriginal or not. We are not particularly interested whether the baby is classified as Aboriginal or not Aboriginal. I am not disagreeing with any definition of Aboriginality.

Mr G Briscoe You are prepared to argue historically about whether a child is wasted or whatever, all that stuff that you have been talking about there in a demographic history form. So that person is born and, okay, we might miss that point and be interested in the Aboriginal mother and not whether the child is Aboriginal, but you are also looking at children from nought to 12, 15 and up the track and whether they are female or not female. Now, that person at four months is not an Aborigine and all of a sudden at six years it is still not an Aborigine, at 10 years when that child decides it is an Aborigine, it becomes an Aborigine.

Prof R Douglas Surely the point that Gordon is making, is that, there ought to be standardisation and uniformity across the country about an issue that apparently may not be clearly stated across the country. Is that true or not?

Dr A Gray It is not true that there ought to be a uniform definition of Aboriginality because we would be taking away the very purpose of the definition which gives people the opportunity to identify themselves as Aboriginal or not.

Mr G Briscoe Surely there ought to be some uniformity about the question whether a father should be acknowledged as Aboriginal, and allow that child to be included in the Aboriginal people.

Mr D Black There is a broader definition where the father's and mother's Aboriginality both impact, but in that collection of birth certificates, it is the mother's health status that is the basis of that information. Now, for your other statistics on births and the health of the child as it grows, you have your other data collection.

Prof R Douglas You mean you have a different definition for a birth certificate? You do not reassure me one little bit.

Ms V Drury Well, I can see foresee problems in Western Australia. There are Aboriginal people who do not identify as being Aboriginal, so how do you overcome that?

Mr D Black You cannot. This is a problem, because in the census and all of these collections we rely on self-identification. You cannot say the ABS can come up with a benchmark figure, because ABS can only ask people whether or not they are Aborigines.

Mr G Briscoe What you are doing is not citing fact. What you are talking about is what is accepted as the mother's origin. Now you are not talking about scientific methods. You at the ABS are talking about customary measures.

Dr R Streatfield No, no, Aboriginal people have selected it, we have not. We get around it by using our health workers to identify the Aboriginal babies, whether mother or father. Why don't you use the Aboriginal people in the community to identify them?

Dr A Gray I am going to wind up this discussion because it is not really going to lead us in a productive direction. It is fairly obvious that there are problems about measurement of Aboriginality, which is what I started off saying, and that there is going to be disagreement about how we should do it for particular purposes.

CONCLUDING DISCUSSION

Dr A Gray I would like to get some opinions about the issues that we have had listed, from participants from all parts of Australia, if possible. To effect that I would like to start off by asking Tim Threlfall and Violet Drury to give us some views from Western Australia. Then we might move to South Australia, the Northern Territory, and so on.

Dr T Threlfall One thing that comes to mind immediately is the question we have been arguing about, the cultural appropriateness or otherwise of health care services. The clinical services of the [Aboriginal] Medical Services have something in common with a hospital setting, especially with antenatal patients, so that we are at a point where we are trying to bring the two systems together. The problem is that people work on Aboriginal time where appointments are not particularly important—you do things when there is a need to do them. This is contrasted very starkly with the hospital system, where if people do not turn up they have wasted a time slot in the doctor's busy schedule, and there is a little bit of irritation involved when we get three or four letters from the hospital clinics, one after the other, saying such and such a person has not fronted up, and we won't be issuing any more appointments. It is a very stressful job for all the health workers in this sort of system to try and get these two systems working together. As far as antenatal care goes, the concept of culturally acceptable antenatal care can be served largely by having female doctors available without appointments, so people can get followed up when they care to come into the clinic, or else the emphasis has got to be increased on home visiting of antenatal patients. We do a lot of that in our community nurse service. There is a limit to what we can do with any sort of central clinic.

Dr D Hicks When you look at these figures the thing that strikes me most is the high mortality in young and middle-aged adults, both male and female, from cardiovascular disease and from accidents, poisonings and violence. You can look at all the health factors that contribute to those, but I also wonder about the role that poverty has in the situation. The income which Aboriginal families have is lower than the income of other families. The employment status is much worse. I no longer work in Aboriginal health, but I think that if I wanted to change the system I would be looking at trying to change the financial status of Aborigines, rather than trying to change the health status. I think the health status will probably change itself if you change the financial status.

Dr A Gray In the draft communiqué we do address that in a much more general way. We recognise that the health standards of Aborigines are determined by the position of Aborigines in Australian society. We do not need really to expand on that very much because it is not just poverty, it is discrimination and a whole range of other things.

Ms V Drury What we will be looking at is why Aboriginal people are not accessing the facilities that are available. We are looking at whether it is front-desk staff who are making people feel as if they are not wanted, whether it is discrimination by other

service providers, or whether it is in fact the building, the sterile sort of atmosphere that people have to go into at clinics and whether that is off-putting to people. Apart from the fact that Aboriginal women feel a little bit uncomfortable, it is cultural shame for some to have male doctors look after them. So we are looking at why they are not accessing the facilities that are available.

Dr A Gray Do you feel that any of that is due to lack of knowledge of the types of health problems that we have been talking about?

Ms V Drury I think that what we are trying to do is pass on the information, that they need to be aware of. I think it is the advanced technology too, the unknown. If they have to go to something and they do not really know what it is, it is sort of frightening. To get the information to Aboriginal people, we are hoping to do that through the health workers in our training programs.

Dr A Gray What about this question about high mortality in adulthood?

Ms V Drury Obviously that would have to be a team effort from all people involved, including doctors giving check-ups to see they are okay. I do not really know how.

Dr A Gray Any other thoughts on that question before we go on to South Australia? The question is how we get the message about high mortality and its causes to Aboriginal communities.

Mr G Briscoe You talk to people, don't you. You talk to people. You plan to organise resources. You organise the resources, you get money from the State government and Federal government. You develop a strategy for dealing with the problem. You put your strategy into some organised framework, some kind of submission to the government explaining what the policy is. You then petition the government and the politicians to support that program. Then you employ people to go out there, sit on their behinds, where people live, out bush. In the cities, obviously, the implementation of that theory will be slightly different, but for bush people you can organise strategies to do that. You can employ people to go out there. How do you grow forests? You employ foresters. Health workers are just human resources, some walking around with little white hats on and starched whatsanames, and other people with brooms and whatever. You can organise to do it.

Dr R Hall There is another problem which is perhaps more clearly illustrated with antenatal care and the perinatal outcome, which is that of people's values. Certainly in some communities that I have been associated with, they are quite aware of the mortality, both at the perinatal age and also in the middle-age groups. It is not that they are unaware of the problem, it is partly that they do not know what to do about it, and partly the whole value system. I have a suspicion that some people value the cultural appropriateness of delivery care higher than they value the outcome.

Dr R Streatfield I am the eternal optimist as usual. I have learned over the 10 years working with Aboriginal people that if you can tap into their social structure, you can quite often get messages through a lot easier than you can, I imagine, in a white community where we are all disjointed and living in separate little houses, where we rely on getting our messages through to people through television and the media.

We learned that several times in our northern communities in Queensland, especially with AIDS education. I would be willing to stand up anywhere and say that in Yarrabah community, North Queensland, every individual knows more about AIDS than in any other community, white or black, in Australia. We have been working there for two years solidly in that community, on a one-to-one basis through Aboriginal people to Aboriginal people, and we have discussed not only the facts but what this disease means to their community; what it means to them to have a son or daughter with AIDS; what they are going to do, mother or husband. That is the way to do it.

As Gordon says, it is painstaking, it takes a long time, but we have got to look at the advantages of Aboriginal society, how we can use the positive structures in their society to get the message through. As I said yesterday, we got the message through about rheumatic fever, and that is a fairly complicated disease. We have got the message through now about AIDS and that is also a very complex issue. So I do not think we should be too pessimistic about getting this message through. I think people really know, they see the end-product, but as someone said they do not know what the causes are. It is our job to inform Aboriginal people, who then are the informants of their communities, as Gordon says.

Mr G Briscoe Hang on, when I am talking about organisation I do not want to descend into some anthropological jargon. People in most societies are not aware of many of the problems that are taking place. Look how long it took 'you mob' to see how the industrial structure has been poisoning your social system. Now, we don't descend into some anthropological jargon about how you are going to overcome that problem.

The question of the phenomenon of the death of Aboriginal women in Aboriginal society has to be told to Aboriginal people, to Aboriginal society. I do not want to talk about social structures, or anything like that. What we have got to talk about is strategy. We have got to develop our theoretical framework for the whole of Australia, because this is where it is happening. It is not happening in Sydney only, women aren't getting bashed to death, poisoned, dying from childbirth and all these things only in Sydney, it is all over Australia. We need to pinpoint further resources to hone in on those areas that are most affected, but we also need to put our theoretical strategies together as well. So perhaps it does need a little bit more thinking about. Firstly, where those pressure points are ; the form of words that we need to specify what the problem is in its rhetorical sense; the resources needed to train people up; employment of people; and to proselytise the theoretical framework.

Dr A Gray I have a quite different view about what publicity health promotion efforts in Aboriginal communities achieve. I think it is possible, as Ric Streatfield says, that you can get everyone in a community knowing a lot about AIDS if you put a lot of

work into that. However, most of the health promotion information that I see going into Aboriginal communities goes to the clinic, it goes to the Aboriginal Medical Service, it goes to the health workers, and it goes no further. It certainly goes nowhere near the people that you most want to get, who never come anywhere near the clinic or the health service. Now the question is, how do you get the information about the levels of Aboriginal adult mortality to the entire Aboriginal community in an effective way, so that there develops a community awareness about the problem?

Ms V Drury In Western Australia, if you are looking at using the mass media, GWN is the television channel that all Aboriginal people throughout the State watch, and can give you the information on anything that you like to know about, because they all watch that. That is just one suggestion if you are thinking about going that way. That gets to the community people without going directly through the health worker.

Dr T Threlfall I am not sure how far we want to go along this line of discussion here, because it is not a communications or health promotions strategies workshop. We could go a long way and not be very productive, but I think that we need to put on the record the fact that dissemination of information by definition is not health promotion. We all know that. When we are talking about health promotion, we are talking about a whole bunch of things in addition to the pieces of paper which make up dissemination of information, or the information channels like television, radio or anything else. We all know that but I am not sure that it is an appropriate use of our time this morning to go to the next steps of talking about strategies of communication and health promotion, but rather continue as we started to draw out more central issues about mortality. The communication of those things is really a separate issue.

Dr A Gray I disagree very strongly with that. For years we have been criticised for doing research and not feeding it back to the communities, and it has to be one of our primary aims from a workshop like this to get information back to the Aboriginal community.

Dr T Threlfall I disagree, but that being the case let us agree around the group that that is what we do, and spend our remaining time or an appropriate proportion of it, to do it properly, not in a superficial way, because it is too important to do in a superficial way. I do not think that is an appropriate use of the remaining hour and a half for what we have to do. I believe that the approach that you have indicated this morning is a more fruitful one, to draw out statements from the group about the state-of-the-art as it were, bring together what we have done over the last day and a half and have that as the basis of the remaining period of time we have available. We did not have at the beginning of the workshop, of course, a statement of aims and objectives of the workshop but I think we all implicitly agreed that it was drawing together information. Your guide says that an outcome would be a monograph, produced in one place as the state-of-the-art. We never came to this workshop with a view to developing detailed implementation strategies relating to improving the mortality rates.

Prof R Douglas I agree with what has just been said, that it is important that we do not re-invent all the wheels that have been invented in other forums. This forum is unique in that it brings together the people who have some insights into what is happening to mortality, and that is where we should put our primary effort. That is not to disagree with your assertion that the communication of our findings is absolutely imperative. I do not think that what you and the previous speaker are saying is in conflict, but I do think we ought to quickly move to reach the consensus, which I think exists.

Ms C Brown One of the points that we often miss, working in statistics or as health professionals, and seeing staggering information like the rates of mortality, is that we make the assumption that this message is going to be easily understood by people in an Aboriginal community. We should also be looking at the perception of the health problems, and this is the other dimension on which there is little focus. There was one study done in a community in South Australia where the researcher asked the community very simple questions like: 'How many times did kids not turn up at school because they had diarrhoea?'; and 'How many times did someone in your family experience episodes of diarrhoea?' What he found was that what was reported through the interviews and what actually showed up in the clinic statistics was quite different. So while it is bad that people are dying early and dying from various causes, we somehow have to translate that concern and somehow match it, or at least firstly find out what people's perceptions are of what is killing them, or if mortality is an issue. We are assuming it is an issue—but is it really at a community level?

I also want to make the point that we have to admit that health structures and health professionals are not the only solution. We talk about health education, and we say nutrition and good diet, but there are other restricting factors outside the control of the health structure, and that is namely what is stocked up in stores in communities. The buzz-word is intersectoral approach and that sounds great on paper, but it is hard to work on the ground. Really we have a lot of restrictions, I mean the health industry is restricted by the infrastructure of government.

Dr G Durling First let me put in a disclaimer. I am a statistician who is relatively new to the health area, I am not an epidemiologist and I do not work in the Aboriginal section of the Northern Territory Health and Community Services Department. I am in statistical services. All the data I have worked with have a large number of Aborigines and the data are always separated out into Aboriginal and non-Aboriginal. As has been apparent in a fair number of papers here, the Northern Territory data have been treated. I see what has come out, and I have not been surprised by what I have seen. There is a bit more detail than I have seen in my somewhat more casual observation of the data, but at the same time I am also looking at somewhat later data and I see the same patterns occurring in 1988 and 1989 that I see in this earlier data. So, I recognise that these problems do exist and strongly support the approach that is being taken. At the same time I cannot commit the Northern Territory Government nor the Health and Community Services Department to any explicit action as such.

Dr A Gray Before we close, I would like to express my very deep gratitude for the way in which everybody here has participated in this workshop. It has been marvellous to get the concentrated attention that you have all given to it, and we have had, I think, an extremely valuable exchange of ideas. I am confident that we will be able to get something very useful coming out of it. Also, thanks to Noor Khalidi for the effort he has put into the organisation of the workshop.

Dr L Smith All we really should do is to thank Alan Gray and Noor Khalidi and all the others involved in organising this. It has been a landmark and an excellent initiative.

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APPENDIX: CONVENTIONAL LIFE TABLE MEASURES

The columns of a life table are labelled in a conventional form, and usually include at least six columns, headed ${}_nq_x$, 1_x, ${}_nd_x$, ${}_nL_x$, T_x and $e_x{}^0$. The suffix x in these labels refers to an exact age (for example 10 years exactly) and the prefix n refers to the length of an age group, often five years. For example ${}_5q_{10}$ would signify the probability of death between exact age 10 years and exact age fifteen years. The prefix 1 is often omitted in a single-year life table. The interpretation of the columns of a life table is as follows:

${}_nq_x$ – the proportion of people who reach exact age x who are not still living at exact age x+n (the estimated probability of dying between exact ages x and x+n);

1_x – the number of survivors from the cohort at exact age x (conventionally, 1_0 is taken to be 100,000, sometimes some other large number, but occasionally it is just 1 – the value chosen for 1_0 is known as the *radix* of the life table);

${}_nd_x$ – the number of members of the cohort who die between exact age x and exact age x+n;

${}_nL_x$ – the number of person-years lived by members of the cohort between exact ages x and x+n;

T_x – the number of person-years lived by the cohort above exact age x;

e_x0 – the expected number of years that a person who reaches exact age x will live.

Some other columns may also be included in a life table:

${}_nm_x$ – the death rate between exact age x and exact age x+n;

μ_x – the instantaneous force of mortality at exact age x;

${}_np_x$ – the proportion of people who reach exact age x that are still alive at exact age x+n;

${}_ns_x$ – survival ratio – the proportion of people who survive to age group x to x+n from the immediately preceding age group, or from birth to the first age group.