

Patterns, trends, and increasing disparities in mortality for Aboriginal and non-Aboriginal infants born in Western Australia, 1980–2001: population database study

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Summary

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Background Since there are known disparities between Aboriginal and non-Aboriginal populations in Australia, trends in infant mortality rates can be used to assess the effectiveness of programmes designed to improve the health of Aboriginal populations. We have examined mortality rates in these populations in Western Australia.

Method We used the most comprehensive and valid total population data available for an Australian state to determine all-cause and cause-specific mortality for Aboriginal and non-Aboriginal infants born in Western Australia from 1980 to 2001.

Findings Overall, infant mortality rates fell in both populations, but less so in Aboriginal (from 25.0 in 1980–84 to 16.1 in 1998–2001) than in non-Aboriginal infants (from 8.4 in 1980–84 to 3.7 in 1998–2001) such that disparities between the two groups increased for all major causes of infant death. The relative risk for Aboriginal compared with non-Aboriginal infants rose from 3.0 (95% CI 2.5–3.6) to 4.4 (3.5–5.5), and there were significantly more potentially preventable deaths, such as those caused by infections (5.9 per 1000 livebirths vs 0.7 per 1000 livebirths, RR 8.5, 95% CI 7.1–10.2). Additionally, for Aboriginal infants, postneonatal mortality rates were higher than neonatal mortality rates (11.2 per 1000 livebirths vs 9.7 per 1000 livebirths), trend analyses showed that previous reductions in deaths due to preterm birth (4.3 per 1000 livebirths—1.4 per 1000 livebirths from 1980–97) were not sustained in the most recent years studied (3.5 per 1000 livebirths), and rates of sudden infant death syndrome did not fall significantly (4.9 per 1000 livebirths vs 4.7 per 1000 livebirths).

Interpretation These increasing disparities between Aboriginal and non-Aboriginal infants, especially in remote areas, demand immediate action in partnership with Aboriginal communities, focusing on both access to primary health care and better living conditions. Implementation and assessment of policies to reduce the continuing social and economic disadvantage faced by Aboriginal families are vital.

Introduction

A child's risk of death is greatest around time of birth and in the first year of life. For the past 100 years or so, children who survived the first year had a good chance of living to adulthood. Since the most powerful determinants of infant mortality are social and economic, death in infancy is a good indicator of the social progress of a society, country, or group of people. Many of the causes of infant death, such as infections, are potentially preventable; others, such as low birthweight or preterm birth can be treated with good health care and services. Although we do not know the cause or causes of sudden infant death syndrome (SIDS), the frequency of this diagnosis fell substantially in non-Aboriginal infants after risk-intervention campaigns.¹ Thus infant mortality is also an important measure of the effectiveness and availability of health services for mothers and children. As a result, any disparities in infant mortality, as are seen between rich and poor countries or between Aboriginal and non-Aboriginal children in Australia, are indications of inequalities in social and economic status and in the availability of health care.²

Not only birthweight but also the pattern of infant mortality differs between rich and poor populations.

Those who are advantaged and have good services have higher neonatal than postneonatal mortality rates. The main causes of neonatal deaths are birth defects and preterm birth, which are not very amenable to either prevention or treatment. Postneonatal deaths are now mostly preventable—eg, infections and SIDS.

The excess burden of premature deaths in Aboriginal Australians and the disparity in the rates of infant and childhood deaths between Aboriginal and non-Aboriginal Australians are well known.^{3–5} A comprehensive and accurate profile of mortality is vital to enable effective prevention strategies, including appropriate health and other government policies to redress this disparity.

Despite a large amount of data describing the poor health status of Aboriginal people in Australia, few states have comprehensive death data for more than a few years, including those where most Aboriginal people live.⁶ Thus in 2003, when the National Health and Medical Research Council of Australia developed a plan for Aboriginal health research, a main recommendation was to obtain more data for deaths and for neglected groups such as urban Aboriginal and young people.⁷

Epidemiological data are essential to contribute to the prevention of infant and child deaths and should not only

include mortality trends over time but also measure potential risk factors; these include maternal and infant factors, specific causes of death, where families live, and where the infants die (as measures of access to health-care services). Complete death information is available in the unique total population linked data in Western Australia for all births since 1980. We have comprehensively reviewed all deaths to provide a complete and accurate epidemiological description of trends and potential risk factors.

Methods

Population data

The main source of data was the linked, total population Western Australian Maternal And Child Health Research Database (MCHRDB).⁸ The study population consisted of all livebirths in Western Australia from 1980 to 2001 inclusive that were linked to infant deaths. These data are more than 99% complete for both Aboriginal and non-Aboriginal infants.⁹ Descriptive data for causes of death from the office of the Registrar General of Western Australia and autopsy reports held at the forensic pathology department were individually scrutinised to improve and validate the information. We also used morbidity data for hospital patients to validate place of death and supplement data describing Aboriginal status. Supplementary mortality data were provided by the Western Australian Data Linkage Unit¹⁰ and the Western Australian Birth Defects Registry.¹¹ Birth defects were coded as the major cause of death only when they were identified by the coroner as the most likely underlying cause of death. Cases with a birth defect that was not the probable underlying cause of death were assigned a separate code. The detailed mortality data relating cause of death to possible risk factors are made possible by the availability in these databases of accurate numerator and denominator data for all birth cohorts over time, the accurate identification of Aboriginality, and the validated causes and place of death with appropriate epidemiological categorisation.

We coded all deaths using a three-digit classification code developed specifically for child epidemiological research by a perinatal pathologist and epidemiologists⁵ and extended by Freemantle.⁴ All infant deaths were initially classified in nine categories (shown in tables 1–3) and then aggregated into four main causes of death for further analysis, which consisted of 86% (n=538) of Aboriginal and 85% (2608) of non-Aboriginal infant deaths. Deaths for which the cause was not known—ie, death was attributable to SIDS or cause of death was unascertainable—were combined and thence referred to as SIDS plus.¹² The remaining 14% (91) Aboriginal and 15% (476) non-Aboriginal infant deaths were those classified under maternal, intrapartum, cancer or leukaemia, and other specific conditions categories.

Infants whose mothers were identified in the MCHRDB as Aboriginal or Torres Strait Islander were recorded as Aboriginal (advice from our Kulunga Indigenous Network

	Aboriginal		Non-Aboriginal		RR (95% CI)†
	Number	IMR	Number	IMR	
Births	30 261 (6%)		504 661 (94%)		
Deaths					
Neonatal	293 (47%)	9.7	1971 (64%)	3.9	2.5 (2.2–2.8)
Postneonatal	336 (53%)	11.2‡	1113 (36%)	2.2‡	5.0 (4.4–5.7)
Infant	629 (100%)	20.8	3084 (100%)	6.1	3.4 (3.1–3.7)
Birth cohort					
1980–84	139 (22%)	25.0	882 (29%)	8.4	3.0 (2.5–3.6)
1985–89	166 (26%)	25.0	876 (28%)	7.6	3.3 (2.8–3.9)
1990–94	147 (23%)	20.1	664 (22%)	5.6	3.6 (3.0–4.3)
1995–97	75 (12%)	16.9	315 (10%)	4.4	3.8 (3.0–4.9)
1998–2001	102 (16%)	16.1	347 (11%)	3.7	4.4 (3.5–5.5)
Sex					
Male	346 (55%)	22.3	1792 (58%)	6.9	3.2 (2.9–3.6)
Female	283 (45%)	19.2	1292 (42%)	5.3	3.6 (3.2–4.1)
Area of birth					
Metropolitan	162 (26%)	17.9	2178 (71%)	5.9	3.0 (2.6–3.5)
Rural	132 (21%)	18.5	611 (20%)	6.5	2.9 (2.4–3.4)
Remote	335 (53%)	23.5	290 (9%)	6.8	3.5 (3.0–4.1)
Marital status					
Single	244 (39%)	21.3	471 (15%)	10.7	2.0 (1.7–2.3)
Married/defacto	377 (61%)	20.9	2596 (85%)	5.6	3.7 (3.3–4.1)
Maternal age (years)					
12–15	28 (4%)	29.1	11 (<1%)	18.0	1.6 (0.8–3.2)
16–19	143 (23%)	18.5	273 (9%)	11.0	1.7 (1.4–2.1)
20–24	225 (36%)	21.8	794 (26%)	7.3	2.9 (2.5–3.4)
25–29	143 (23%)	21.9	1003 (33%)	5.5	3.9 (2.3–4.6)
30–34	62 (10%)	19.8	691 (22%)	5.2	3.9 (3.0–5.0)
35–39	23 (4%)	23.5	251 (8%)	5.4	4.4 (2.9–6.7)
≥40	1 (<1%)		48 (2%)	6.6	0.9 (0.1–6.4)
Gestational age (weeks)					
20–27	126 (20%)	440.6	896 (29%)	448.9	1.0 (0.9–1.1)
28–32	39 (6%)	60.7	311 (10%)	57.7	1.1 (0.8–1.5)
33–36	99 (16%)	29.9	378 (12%)	12.8	2.3 (1.9–2.9)
37–42	309 (49%)	12.4	1428 (46%)	3.1	4.0 (3.6–4.5)
43–45	5 (1%)	18.1	28 (1%)	5.2	3.5 (1.3–8.9)
<20, >45	3 (<1%)		17 (1%)		
Plurality					
Singleton	597 (95%)	20.1	2661 (86%)	5.4	3.7 (3.4–4.1)
Multiple	32 (5%)	54.7	423 (14%)	31.5	1.8 (1.2–2.5)
Previous births (number)					
0	127 (20%)	16.9	889 (29%)	5.5	3.1 (2.6–3.7)
1–2	257 (40%)	21.1	1453 (47%)	5.6	3.8 (3.3–4.3)
3–4	106 (17%)	19.2	535 (17%)	7.9	2.4 (2.0–3.0)
≥5	129 (21%)	28.1	194 (6%)	11.5	2.4 (1.9–3.1)
Birthweight (g)					
<350	4 (<1%)	500.0	9 (1%)	163.7	
350–499	32 (5%)	864.9	214 (7%)	934.5	0.5 (0.1–1.5)
500–999	99 (16%)	440.0	656 (21%)	403.7	1.1 (0.9–1.3)
1000–1499	30 (5%)	92.9	244 (8%)	89.7	0.9 (0.6–1.3)
1500–2499	120 (19%)	39.0	491 (16%)	20.8	1.9 (1.6–2.3)
2500–4499	329 (53%)	12.6	1359 (46%)	3.0	4.2 (3.8–4.8)
>4499	9 (2%)	25.4	63 (2%)	8.4	3.1 (1.6–6.3)

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PEBW (categories)					
<75%	74 (15%)	57.2	550 (18%)	38.5	1.5 (1.2-1.8)
75%–<85%	102 (16%)	25.4	467 (15%)	10.0	2.6 (2.1-3.2)
85%–<115%	295 (47%)	15.0	1643 (53%)	4.3	3.5 (3.1-3.9)
115%–<125%	30 (5%)	17.3	164 (5%)	4.2	4.0 (2.7-5.9)
125%	20 (3%)	22.3	123 (4%)	9.1	2.5 (1.5-3.9)

PEBW=percentage of expected birthweight. Percentages might not add to 100% because of missing data. Relative risk (RR)=Aboriginal vs non-Aboriginal infants. IMR=infant mortality rate. *Per 1000 livebirths. †RR ‡Per 1000 neonatal survivors.

Table 1: Mortality data* and relative risk for Aboriginal and non-Aboriginal infants and RR (95% CI) for Aboriginal compared with non-Aboriginal infants born in Western Australia between 1980 and 2001 according to population variables

is that it is appropriate to use the term Aboriginal, to describe infants born to Aboriginal or Torres Strait Islander mothers, especially with the very small number of Torres Strait islander mothers in these data). This variable was based on self-identification and community affirmation (including mixed Aboriginal and non-Aboriginal ancestry) and is taken from the Midwives' Notification System.¹³ If the race of the mother was missing, information about the race of the infant on admission was sought from the morbidity inpatient data. Read¹⁴ reported that there were around 250 births per year (1% of all births in Western Australia) in which the father was Aboriginal and the mother non-Aboriginal. In these analyses, such infants are included with the non-Aboriginal population because they are not easily identified. Any infant for whom the race of mother or child could not be

identified (n=197, 0.04% of births in the study) was excluded from the analysis. Infant deaths are classified as infants born alive who died before their first birthday, neonatal deaths were those dying in the first 28 days of life and postneonatal deaths were those who died between 28 days and 12 months.

Place of birth was obtained from the MCHRDB and place of death was identified from the Registrar-General's database. The Hospital Morbidity Database System was used to validate information for place of death (in or out of hospital). Autopsy case reports were used to establish where the infant was living at time of death and, if the death occurred out of hospital, the geographical area of the death.

Socioeconomic status can be assessed by use of the Australian Bureau of Statistics socioeconomic index for areas since all mother-infant pairs are coded into their collection districts.¹⁵ However, there are some methodological drawbacks and inadequacies with use of these codes when considering Aboriginal socioeconomic status. For example, indicators that are used to construct measures of socioeconomic status include Aboriginality. Thus we did not use these codes in the analyses to adjust mortality rate differences between the Aboriginal and non-Aboriginal infant populations.

Statistical analysis

We calculated frequencies of births and deaths for Aboriginal and non-Aboriginal infants and stratified by several infant and maternal variables using SPSS version 10.1 statistical software. Mortality rates were calculated for infant, neonatal, and postneonatal deaths separately. Infant mortality rates (IMR) and neonatal mortality rates (NMR) were calculated per 1000 livebirths and postneonatal mortality rates (PMR) were calculated per 1000 neonatal survivors. Rate ratios of death for Aboriginal infants (compared with non-Aboriginal infants) were calculated with 95% CIs, estimated with Taylor series approximations in EpiInfo Statcalc version 3.2.2 in several subgroups obtained by stratification by variables of interest. These variables included mother's age, marital status, and parity at time of the infant's birth; gestation; plurality; percentage of expected birthweight; date of birth; date of death; cause of death; place of death (in or out of hospital); and geographical area of residence of the mother at time of birth and infant at time of death—according to metropolitan, rural, and remote geographical divisions. These divisions were amalgamated from 11 health zones that were defined in the mid 1990s¹⁶ and that took into account the isolation and distance from the major regional and metropolitan health-care facilities. The percentage of expected birthweight was calculated with mother's height and parity, and infant's birthweight, gestational age, plurality, and sex.¹⁷ The percentage of expected birthweight is a better measure of fetal growth than is birthweight, but we also analysed deaths in birthweight categories to compare with the work of others. All the demographic

	Aboriginal		Non-Aboriginal		Relative risk* RR (95%CI)
	IMR†	Number	IMR	Number	
Neonatal					
1980–2001	9.7	(293)	3.9	(1971)	2.5 (2.2-2.8)
1980–84	15.3	(85)	5.3	(552)	2.9 (2.3-3.7)
1985–89	10.2	(68)	5.1	(579)	2.0 (1.6-2.6)
1990–94	8.4	(61)	3.5	(411)	2.4 (1.9-3.2)
1995–97	7.2	(32)	2.9	(208)	2.5 (1.7-3.6)
1998–2001	7.4	(47)	2.3	(221)	3.2 (2.3-4.4)
p value‡	<0.0001		<0.0001		
Postneonatal					
1980–2001	11.2	(336)	2.2	(1113)	5.0 (4.4-5.7)
1980–84	9.9	(54)	3.2	(330)	3.1 (2.3-4.2)
1985–89	14.9	(98)	2.6	(297)	5.7 (4.6-7.2)
1990–94	11.9	(86)	2.1	(253)	5.7 (4.4-7.9)
1995–97	9.8	(43)	1.5	(107)	6.5 (4.6-9.3)
1998–2001	8.8	(55)	1.3	(126)	6.6 (4.8-9.0)
p value†	3.3	(0.7)	97.3	(<0.0001)	

*Aboriginal vs non-Aboriginal infants. †per 1000 livebirths. ‡Test for decreasing trend of rates.

Table 2: Neonatal and postneonatal mortality rates for Aboriginal and non-Aboriginal infants and RR (95% CI) for Aboriginal compared with non-Aboriginal infants born in Western Australia in birth-year groups, 1980–2001

variables that we examined are included in tables and no adjustment was made for multiple testing, according to Rothman's recommendations.¹⁸ Linear trends in rates were assessed with Mantel's extension trend test.¹⁹

Role of the funding source

Healthway Western Australia funded the doctoral fellowship and the Department for Community Development funded a research fellowship for C J Freemantle. The funders had no role in study design, data collection, data analysis, data interpretation, or writing of the report or in the decision to submit it for publication. The corresponding author had full access to all the data and had final responsibility for the decision to submit for publication.

Results

We assessed total-population mortality data for 534 922 births in Western Australia between 1980 and 2001, in this birth cohort there were 3713 deaths. Births to Aboriginal mothers accounted for 6% of all births in Western Australia, but for 17% of the deaths (table 1). The IMR for Aboriginal infants was over three times that for non-Aboriginal infants. By contrast with non-Aboriginal infants, the rate of postneonatal death in Aboriginal infants was higher than that of neonatal deaths, but in both age categories Aboriginal infants were significantly more likely to die than non-Aboriginal infants. This disparity was five-fold during the postneonatal period. Analyses of the IMR in the different birth-year groups showed that the rates fell significantly for the years studied, although not to the same magnitude in Aboriginal as in non-Aboriginal infants. Thus the increase in relative risk (RR) from 3.0 in 1980–84 to 4.4 in 1998–2001 was attributable to the drop in non-Aboriginal infant mortality, rather than a rise in Aboriginal infant mortality.

The rates were higher for both Aboriginal and non-Aboriginal boys than for girls in their respective populations. The IMR for both populations was highest in infants whose mothers were residing in remote areas at the time of the infants' births (table 1). The RR of death for Aboriginal infants (compared with non-Aboriginal) was also highest in remote areas (table 1). Death rate in Aboriginal infants did not differ by marital status. However, infants of non-Aboriginal single mothers were nearly twice as likely to die in infancy as those with married mothers: (RR 1.9, 95% CI 1.7–2.1).

27% of Aboriginal and 9% of non-Aboriginal infants who died in infancy were born to teenage mothers (table 1). The highest IMR was in the small group of mothers who were between ages 12 and 15 years at the time of giving birth and, although increased, the RR for Aboriginal compared with non-Aboriginal infants was not significant. When the teenage years were combined, the IMR was 19.6 per 1000 livebirths for Aboriginal infants and 11.4 per 1000 livebirths for non-Aboriginal infants. The rate for Aboriginal infants (21.85 per

	Aboriginal IMR*	Non-Aboriginal IMR	RR† (95% CI)
Metropolitan			
1980–2001	17.9	5.9	3.0 (2.6–3.5)
1980–84	18.5	8.1	2.3 (1.5–3.4)
1985–89	23.2	7.6	3.1 (3.3–4.2)
1990–94	19.3	5.4	3.6 (2.7–4.8)
1995–97	16.6	4.6	3.7 (2.4–5.5)
1998–2001	12.1	3.5	3.5 (2.3–5.2)
p value‡	0.03	<0.0001	
Rural			
1980–2001	18.5	6.5	2.9 (2.4–3.4)
1980–84	17.4	8.9	2.0 (1.3–2.9)
1985–89	24.8	7.3	3.4 (2.4–4.8)
1990–94	18.0	6.3	2.9 (1.9–4.3)
1995–97	10.5	4.7	2.2 (1.1–4.3)
1998–2001	18.3	3.9	4.7 (3.0–7.5)
p value	0.3	<0.0001	
Remote			
1980–2001	23.9	6.8	3.5 (3.0–6.8)
1980–84	32.6	10.0	3.3 (2.5–4.4)
1985–89	27.0	8.7	3.0 (2.2–4.0)
1990–94	22.0	5.7	3.9 (2.7–5.4)
1995–97	20.3	2.5	8.2 (4.5–15.1)
1998–2001	18.0	4.9	3.7 (2.4–5.6)
p value	0.0002	<0.0001	

*Per 1000 livebirths. †Aboriginal vs non-Aboriginal infants. ‡Test for decreasing trend of IMR.

Table 3: Mortality rates for Aboriginal and non-Aboriginal infants and RR (95% CI) for Aboriginal compared with non-Aboriginal infants born in Western Australia by geographical area and birth year groups, 1980–2001

1000 livebirths) whose mothers were aged between 20 and 29 years was significantly higher than for non-Aboriginal infants (6.4 per 1000 livebirths) (RR 3.5, 95% CI 3.1–3.9). However, the highest RR was in infants whose mothers were 30 years and older.

The only groups with large numbers of deaths and with no significant increase in RR of death for Aboriginal infants were the very preterm (<33 weeks) and very low birthweight babies (<1500 g). The highest RR were for Aboriginal infants who were born after more than 37 weeks' gestation, those who weighed more than 2500 g at birth, and those who weighed more than their expected birthweight.

The neonatal and postneonatal death rates for Aboriginal infants present a different pattern from that for non-Aboriginal infants (table 2). Unlike in the non-Aboriginal population, the postneonatal death rate in the Aboriginal population was higher than the neonatal mortality rate. In the neonatal period, and more so in the postneonatal period, the disparity between Aboriginal and non-Aboriginal infants increased during the years studied. Rates fell significantly during the years studied apart from postneonatal death in Aboriginal infants.

	Aboriginal		Non-Aboriginal		RR (95% CI) †
	IMR*	number	IMR	number	
SIDS plus‡					
1980–2001	6.5	197	1.3	635	4.6 (4.4–6.1)
1980–84	4.9	27	1.8	185	2.8 (1.8–4.1)
1985–89	9.9	66	1.8	203	5.6 (4.3–7.4)
1990–94	6.7	49	1.2	140	5.7 (4.1–7.9)
1995–97	5.6	25	0.7	49	8.2 (5.1–13.3)
1998–2001	4.7	30	0.6	57	7.9 (5.1–12.2)
p value§	0.1		<0.0001		
Infection					
1980–2001	5.9	178	0.7	349	8.5 (7.1–10.2)
1980–84	6.3	35	0.9	98	6.8 (4.6–9.9)
1985–89	6.5	43	0.6	66	11.2 (7.7–16.5)
1990–94	6.9	50	0.8	93	8.8 (6.2–12.4)
1995–97	5.4	24	0.7	49	7.9 (4.8–12.8)
1998–2001	4.1	26	0.5	43	9.0 (5.6–14.7)
p value	0.08		0.002		
Prematurity					
1980–2001	3.3	(99)	1.7	(840)	2.0 (1.6–2.4)
1980–84	4.3	(24)	2.3	(241)	1.9 (1.2–2.9)
1985–89	3.8	(25)	2.3	(258)	1.7 (1.1–2.5)
1990–94	3.0	(22)	1.3	(160)	2.2 (1.4–3.5)
1995–97	1.4	(6)	1.3	(89)	1.1 (0.5–2.5)
1998–2001	3.5	(22)	1.0	(92)	3.6 (2.3–5.7)
p value	1.1		<0.0001		
Birth defects					
1980–2001	3.1	(93)	1.6	(826)	19 (15–23)
1980–84	4.5	(25)	2.2	(230)	21 (14–31)
1985–89	3.2	(21)	2.2	(250)	15 (09–23)
1990–94	2.5	(18)	1.5	(180)	16 (10–27)
1995–97	2.9	(13)	1.1	(80)	26 (15–47)
1998–2001	2.5	(16)	0.9	(86)	28 (16–47)
p value	0.08		<0.0001		
*Per 1000 livebirths. †Aboriginal vs non-Aboriginal infants. ‡SIDS plus unascertainable deaths.11 §Test for decreasing trend of IMR.					
Table 4: Mortality rates for main causes of death in Aboriginal and non-Aboriginal infants and RR (95% CI) for Aboriginal compared with non-Aboriginal infants born in Western Australia, 1980–2001					

Although there was a significant fall in the IMR for the total Western Australian population between birth years 1980 and 1997 in all geographical areas, for 1998–2001 the rate has risen in remote areas, remained the same in rural areas, and significantly decreased in metropolitan areas (RR 0.8, 95% CI 0.7–0.9) (data not shown). Over the past 22 years, the IMR was highest in remote areas for Aboriginal and non-Aboriginal infants (table 3). The rates fell significantly for Aboriginal infants in metropolitan and remote areas, and for non-Aboriginal infants in all areas. The mortality risk for Aboriginal infants was significantly higher than for non-Aboriginal infants in all geographical areas and at all times.

The increase in the IMR in non-Aboriginal infants living in remote areas in the years 1998–2001 was the reason for

the increase in the total population IMR from 7.1 per 1000 livebirths in 1995 to 12.3 per 1000 livebirths in 1998–2001. For Aboriginal infants born between 1998 and 2001 in the same years, the all-cause IMR was more-or-less similar in rural and remote areas and lowest in metropolitan regions. However, these differences in risk were not significant, apart from non-Aboriginal infants in remote compared with metropolitan areas (RR 1.4, 95% CI 1.0–2.0).

The rates for all causes of deaths were significantly higher in Aboriginal than non-Aboriginal infants apart from deaths due to cancer or leukaemia and postneonatal death (data not shown) attributable to intrapartum disorders, for which the numbers were very small. The most obvious excesses were for infection and SIDS plus (table 4). Maternal conditions and accidents had increased rate ratios, but the numbers were very small. The most common causes of neonatal death in both populations were prematurity and birth defects (table 4). However, in the postneonatal period, the main causes of Aboriginal death were SIDS plus and infection, and in the postneonatal period for non-Aboriginal infants the main causes were SIDS plus and birth defects (data not shown).

The cause-specific IMR significantly decreased for all main causes of non-Aboriginal infant death (table 4). However, for Aboriginal infants, although the overall IMR fell significantly ($\chi^2=19.1$, $p=0.0001$), when the main causes of death were analysed separately, the falls were not significant. The RR for Aboriginal compared with non-Aboriginal infants was significantly higher for all main causes of death and for all year groups apart from prematurity in 1995–97 and birth defects in 1985–89. Deaths due to sequelae of prematurity in Aboriginal infants, which had fallen in earlier years (4.3 per 1000 in 1980–84 to 1.4 per 1000 livebirths in 1995–97, $p=0.008$) to rates similar to those for non-Aboriginal infants, significantly increased (RR 2.6, 95% CI 1.0–6.3) in 1998–2001.

Although infection was a significantly more important cause of death for Aboriginal than for non-Aboriginal infants, with the exception of a small number of Aboriginal postneonatal deaths due to gastroenteritis, the distribution of site of infection did not differ between Aboriginal and non-Aboriginal deaths. The main causes of deaths from infection in both Aboriginal and non-Aboriginal infants were respiratory infection, followed by chorioamnionitis (infection in the placental membranes). The percentage of deaths attributable to chorioamnionitis and septicaemia contributed greater proportions of deaths in non-Aboriginal than in Aboriginal infants. For both populations, deaths from chorioamnionitis and septicaemia were higher in the neonatal period than in the other groups and respiratory infections were the main cause of death from infection in the postneonatal period (data not shown).

The IMR attributable to infection was higher for Aboriginal than non-Aboriginal infants wherever they lived (figure 1). However, the risk of death from infection

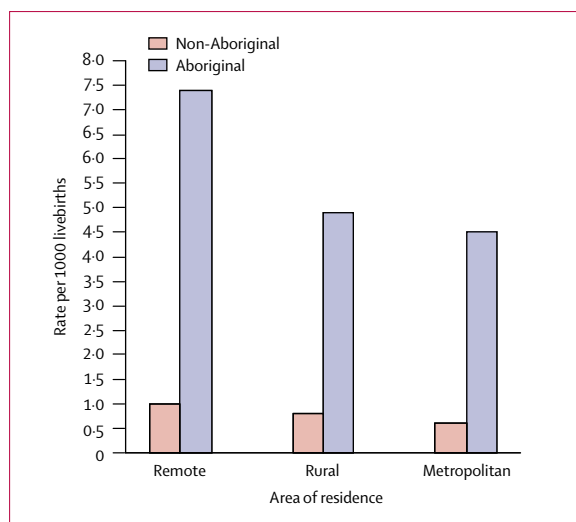


Figure 1: Death rates from infection for Aboriginal and non-Aboriginal infants born in Western Australia according to geographical area of mother's residence at time of infant's birth, 1980–2001.

for both Aboriginal and non-Aboriginal infants living in remote areas was significantly higher than those living in rural or metropolitan areas. Comparisons between Aboriginal infants showed that the risk of dying from infection for those living in remote areas was significantly higher than for those in metropolitan (RR 1.6, 95% CI 1.1–2.3) or rural areas (1.5, 1.0–2.2). A similar pattern was seen for non-Aboriginal infants (remote compared with metropolitan 1.7, 1.2–2.3; and rural compared with metropolitan 1.3, 1.0–1.7). The IMR from respiratory infection was highest for infants living in remote areas (Aboriginal 3.1 per 1000 livebirths; non-Aboriginal 0.5 per 1000 livebirths, data not shown). The IMR due to chorioamnionitis was also highest in remote areas for Aboriginal infants (1.2 per 1000 livebirths), but was similar in all areas for non-Aboriginal infants (0.2 per 1000 livebirths) (data not shown).

In the 1990s generally, there were falls in Aboriginal infant deaths due to infection in remote and metropolitan areas. However, in the birth year groups 1998–2001, Aboriginal infant deaths from infection increased in rural areas. Non-Aboriginal infant deaths from infection

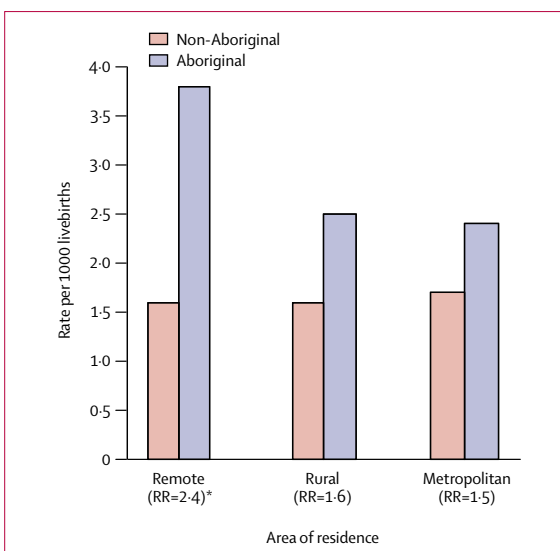


Figure 2: Death rates from birth defects for Aboriginal and non-Aboriginal infants born in Western Australia by geographical area of mother's residence at the time of infant's birth, 1980–2001

Relative risk for Aboriginal compared with non-Aboriginal. *Significant difference.

fell in metropolitan and rural areas, and there was a slight rise in the birth-year group 1998–2001 in remote areas (data not shown).

55% (n=346) of Aboriginal and 71% (n=2190) of non-Aboriginal infants died in hospital. Most deaths due to sequelae of prematurity and birth defects occurred in hospital, whereas most attributable to SIDS plus took place out of hospital for both populations (data not shown). The main difference between the two populations was rate of infections, with about 40% of Aboriginal deaths occurring out of hospital compared with 22% of non-Aboriginal deaths (table 5). This disparity arose in the remote and rural areas. The RR of deaths due to infection that took place out of hospital for Aboriginal infants was significantly higher than non-Aboriginal infants in all areas, but was especially high in metropolitan areas. The high RR in the metropolitan locations resulted from the low rate of non-Aboriginal deaths from infection occurring out of hospital.

When analysed by area, the disparity between the percentage of Aboriginal and non-Aboriginal infants dying out of hospital was greatest in remote areas (table 5) and almost double in rural areas. There was no disparity in metropolitan areas. Non-Aboriginal infants living in remote (RR 3.3, 95% CI 1.8–6.3) and rural (1.8, 1.0–3.3) areas were at significantly greater risk of death from infection out of hospital than were non-Aboriginal infants living in metropolitan areas (data not shown).

The IMR attributable to birth defects was highest in remote areas for Aboriginal infants and in metropolitan areas for non-Aboriginal infants (figure 2). The risk of Aboriginal death was significantly higher compared with non-Aboriginal infants in remote areas. There were no

	Aboriginal		Non-Aboriginal		RR (95% CI)†
	Number	IMR*	Number	IMR	
Remote	35 (20%)	2.5	13 (4%)	0.3	8.2 (4.3–15.4)
Rural	12 (8%)	1.7	16 (5%)	0.2	9.9 (4.7–20.9)
Metropolitan	22 (12%)	2.4	44 (13%)	0.1	26.0 (15.4–44.8)
All areas	69 (40%)	2.3	73 (22%)	0.2	15.8 (11.4–21.9)

*Per 1000 livebirths. †Aboriginal vs non-Aboriginal infants.

Table 5: Number (%), mortality rate, and RR of out of hospital deaths from infection of Aboriginal compared with non-Aboriginal infants born in Western Australia, 1980–2001

significant differences in risk of death from birth defects in different areas in Aboriginal or in non-Aboriginal infants, although there was a nearly two-fold difference between remote and metropolitan Aboriginal infants (RR 1.6, 95% CI 1.0–2.6).

Discussion

This research has extended the previous reports of infant mortality in Western Australia and described all-cause and cause-specific mortality to the end of 2002. We have shown that the main causes of infant death in Western Australia are SIDS and infection in Aboriginal infants and birth defects and prematurity in non-Aboriginal infants.⁵ Notably, the only significantly reduced rates in Aboriginal infants were overall neonatal deaths and those due to sequelae of prematurity up to birth years 1995–97. However, these rates then rose in the most recent years studied. Conversely, significantly decreasing rates were reported in non-Aboriginal infants for deaths attributable to prematurity, infection, SIDS, and birth defects. Although the significant reduction in death from prematurity in all infants was encouraging, that this decrease was not sustained for Aboriginal infants further emphasises the inequity in health outcomes for this population.

Despite the reported prevalence of birth defects being higher in non-Aboriginal than Aboriginal infants born in Western Australia,²⁰ the risk of death from birth defects was substantially higher in Aboriginal infants, significantly so if they lived in remote communities compared with Aboriginal infants living in metropolitan areas. These differences could be due to several factors, such as the reduced antenatal screening for birth defects in Aboriginal women living in remote areas, parental decisions not to terminate a pregnancy, poor identification of birth defects, and the inadequacy of clinical services in remote areas for Aboriginal infants.

Infant mortality for non-Aboriginal and Aboriginal infants according to where they were living at the time of death was similar to that in a study in British Columbia between 1981 and 2000,²¹ in which the disparity in mortality was reported to be 3.6-fold higher in First Nations compared with non-First Nations infants in both urban and rural areas. Similar to our findings, reductions in the disparities in infant mortality by race were less substantial in urban than in rural areas. In our study, the disparities were higher in rural and remote than in metropolitan areas in the most recent years studied, and were highest in remote areas.

The relative disadvantages in health and social outcomes are well documented for Aboriginal Australians.^{22,23} Since most people living in remote areas are Aboriginal and because Aboriginal Australians carry an excess burden of mortality at all ages compared with non-Aboriginal Australians,²⁴ the infant death rate in remote areas would be expected to be higher than in metropolitan areas, as we recorded. However, the data

also show that Aboriginality was a dominant factor independent of place of birth. As such, Aboriginal infants living in the metropolitan location were almost as disadvantaged as those living more remotely. Thus the social and other disadvantages of being Aboriginal outweighed any others.

The much higher risk of death from infection for Aboriginal infants living in remote areas could be explained by the exposure to adverse environmental factors when they return to remote communities after birth. Moreover, poor access to optimum and appropriate maternal-health and infant-health services in remote areas might mean that pregnant Aboriginal women and their infants are less likely to be diagnosed or treated in a timely fashion. We were not able to investigate the adequacy of immunisation in this study. Effective maternal and infant antenatal and postnatal health services promotion and education programmes need to be developed in partnership with Aboriginal families as has been done in Kalgoorlie, a rural town in Western Australia.²⁵

A greater percentage of Aboriginal (45%) than non-Aboriginal (29%) infant deaths took place out of hospital. The different proportions of Aboriginal compared with non-Aboriginal deaths from infection arising out of hospital in remote and rural areas suggest delayed presentation, diagnosis, or treatment. These delays would also affect the infant's chances of survival once treatment is given, and would account in part for the increased risk of death from infection in hospital for remote-living Aboriginal infants.² Further investigation of where these delays occur is needed, especially since most out of hospital deaths from infection were a result of respiratory infection for both populations (73% of all deaths due to infection). At present, these data are being used to guide the new statewide-health clinical network, which has a specific task of improving services for children across the whole state of Western Australia.

Not only did Aboriginal infants have many more potentially preventable deaths, especially infections, but also there were increasing disparities between Aboriginal and non-Aboriginal infants for all major causes of deaths (SIDS, infections, and birth defects) and, more recently, for deaths associated with preterm births. Infant mortality rates have improved generally, and these increasing disparities are caused by the non-Aboriginal rate improving faster than that for Aboriginal children. Furthermore, although the death rates were higher in remote areas for both populations, they were highest for Aboriginal than non-Aboriginal infants.

The significant decrease in neonatal death rate for both Aboriginal and non-Aboriginal infants in Western Australia between 1980 and 1997 that we have shown is in accord with Alessandri and colleagues' findings.⁵ The pattern of higher postneonatal than neonatal death rates in Aboriginal infants, often reported in less developed countries and in Native Americans,²⁶ shows the high rate

of preventable death associated with poverty, disadvantage, marginalisation, and poor health care. In non-Aboriginal infants, the death rate was significantly higher in the neonatal than the postneonatal period, which is the pattern generally seen in more advantaged populations.

Several factors might have determined this pattern of mortality. First, there have been substantial improvements in neonatal care and neonatal transport, resulting in a greater proportion of Aboriginal mothers having their babies in hospital.¹³ These factors are also apparent in the fairly low death rate attributable to sequelae of prematurity in Aboriginal infants between 1995 and 1997. Second, the higher rate of death from postneonatal infection in Aboriginal than in non-Aboriginal infants could be a result of poor environmental conditions, such as inadequate and overcrowded housing, poor nutrition, poor hygiene, and exposure to cigarette smoke.²⁷ Immunisation rates for Aboriginal infants in Western Australia are known to be inconsistent, which is an indication of service models,²⁸ and breastfeeding rates are known to be low.²⁹ The high rates of infection could also be due to the absence of timely care, especially in remote areas, and to failure of clinical staff, family, or both to recognise the seriousness of the illness.

Infants born to teenage mothers (less than 20 years old), especially those aged less than 16 years, are generally at a much higher risk of death than those born to older women.³⁰ The association of poor outcome with teenage motherhood might operate via socioeconomic factors such as poverty, inadequate education, poor antenatal attendance, high rates of smoking and alcohol use, and poor maternal nutrition,^{31,32} as well as physical immaturity. In this study, although the risks were higher for non-Aboriginal infants born to teenagers, the increase in risk for those born to Aboriginal teenagers was only apparent in those less than 16 years old. Although no adequate measures of social and economic status are available for the Aboriginal population, the high mortality rates in the infants of non-Aboriginal teenage mothers and single mothers strongly suggest that a background of social and economic disadvantage constitutes the main difficulty, rather than genetic or biological factors based on racial differences. Furthermore, conventional measures of socioeconomic status and single motherhood within Aboriginal groups are poor predictors of outcome, by contrast with the strong association in the non-Aboriginal population. Being Aboriginal is overwhelmingly disadvantaging because of the history of marginalisation that such people have been subjected to for generations. The history of marginalisation (rather than their Aboriginality) is closely related to the high risk of infant death. Although social and economic differences do occur within the Aboriginal population, they seem to be weakly associated with outcomes.³³

Our analysis will enable a contextual assessment of trends. The high death rates in Aboriginal infants are

nearly identical to those seen in non-Aboriginal infants in Australia in 1900. This modern pattern in Aboriginal infants indicates their adverse environments, as was done for Australian children in 1900.³⁴ This research has also shown that a reduction in infant mortality due to prematurity is achievable, although in the most recent years, unsustainable in the Aboriginal population. The reduction is probably attributable to initiatives introduced to reduce perinatal mortality. These measures include the improvement of neonatal intensive-care facilities and perinatal transport and the rise in hospital births in Aboriginal mothers. Such positive achievements show that with well planned, focused, and determined efforts, substantial improvements in health outcomes can be achieved. However, programmes aimed at reducing SIDS, decreasing serious infections, and improving antenatal care to reduce low birthweight and preterm birth are likely to be more cost-effective in decreasing both neonatal and postneonatal deaths in Aboriginal infants.

The higher rates of death in remote areas, particularly deaths from infection and birth defects and the increasing disparities in the IMR in these areas demand immediate scrutiny, with a focus on existing service provision, including immunisation, antenatal care, clinical services, and environmental factors. The pathways associated with these inequalities should be clarified and outcomes improved for these vulnerable infants.

Our findings draw attention to the increasing disparities in death rates between Aboriginal infants and their non-Aboriginal peers, which provide an important indicator of the overall health and wellbeing of Aboriginal communities and the long-term effect that racism, discrimination, and dispossession have had on Aboriginal people.²² Moreover, high IMRs in marginalised, disadvantaged minority populations indicate the stresses and challenges these communities face, not only at the moment of birth and in the first year of life, but throughout the entire lifecycle and from one generation to another.²² These disparities demand immediate attention and the bipartisan (Aboriginal and non-Aboriginal) formulation of policies and strategies to redress the excess deaths in Aboriginal infants.

Contributors

C J Freemantle developed the mortality database, participated in data collection, data analysis, data validation, and writing of the report. A W Read participated in the development of the mortality database, data collection, data analysis, data validation, and writing of the report. N H DeKlerk participated in the development of the mortality database, data analysis, and writing of the report. I P Anderson participated in the study design, the supervision of research activities, data interpretation, and the writing of the paper. D McAullay participated in the development of the research proposal, provided advice throughout the research activities, and participated in the analysis of the data, and writing of the report. F J Stanley participated in the development of the study, supervised the research activities, and participated in the writing of the report. All authors have seen and approved the final version.

Conflict of interest statement

We declare that we have no conflict of interest.

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