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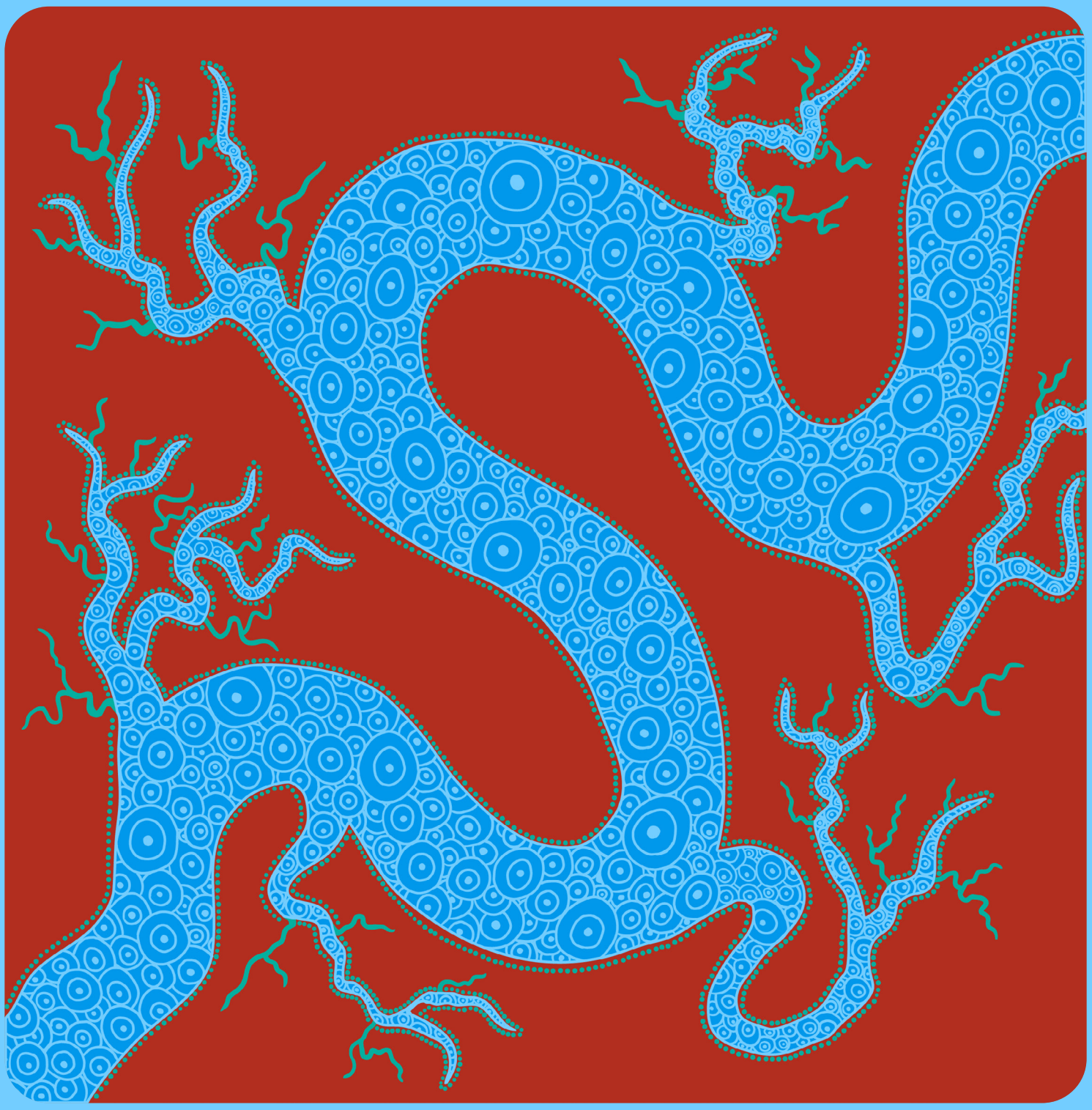

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'Temporal Currents' created by Jayde Hopkins of Nawula Almaren

'Temporal Currents' brings to life 'Knowledge meets Impact' – Menzies' tagline, and reflects Menzies' history and growth as an organisation.

The artwork also highlights the impact of knowledge sharing with our community — a two-way conversation where knowledge flows into Menzies and back out into the community.

The artwork depicts a great river system with branching tributaries, symbolising the journey of Menzies through time. Within the flowing water, knowledge is gathered and carried forward by the currents. Each tributary represents the diverse paths of research projects Menzies has undertaken over the last 40 years, shaping the landscape, influencing its surroundings, yet always remaining part of the greater system.

Beyond a river, the imagery is reminiscent of neural pathways in the brain, the intricate mycelial networks within soil, or the deep-reaching roots of a plant. These living connections show how knowledge flows, grows, and intertwines, shaping Menzies' story as it continues to unfold. As the river meanders across the canvas, it moves toward the future, carrying the past while embracing new discoveries.

Contents

Menzies collection introduction

It is an honour to introduce this collection of articles showcasing the remarkable work of Menzies School of Health Research (Menzies) over the past 40 years.

Founded in Darwin in 1985, Menzies has been at the forefront of Aboriginal and Torres Strait Islander health research in the Northern Territory and across Australia since its establishment. As the founding director, Prof John Mathews AM noted when Menzies first opened its doors, "(Menzies contributes) to the debate on Aboriginal Health by helping fill gaps in understanding, communication and implementation. It has attracted expert staff to the Northern Territory, driven research to identify areas of unmet need, tested innovative health interventions, and been an evaluator, critic and advocate of Aboriginal health policy."

This collection of 10 key publications demonstrates Menzies' deep connection to and respect for the communities with whom they work as well as a commitment to improve the health and wellbeing of the people in the region through genuine partnerships and excellence in community-centred research, translation and education. Each of these articles highlights how meaningful research and collaborations can create positive impact and change – to policy, to clinical guidelines and to health outcomes.

The strength and quality of Menzies' research is reflected in its continued success, including the consistent receipt of competitive grants and accolades. This collection, spanning the four decades of Menzies' work, features four articles that received the *MJA* Award for Best Original Clinical Research in their respective years of publication. The selection highlights Menzies' leadership in tackling the rising rates of diabetes in young First Nations Australians and its contributions to the prevention and management of acute rheumatic fever (ARF), rheumatic heart disease (RHD), and otitis media. It also sheds light on systemic disparities experienced by First Nations Australians in the health system and the serious consequences of miscommunication in their health care.

This collection illustrates the evolution of Menzies' work over the last 40 years. While its research initially focused on the Northern Territory, Menzies' impact now extends far beyond. I am proud that the NHMRC has supported many researchers involved in the research showcased in this collection, which has positively shaped and strengthened First Nations research in Australia. Through expanding research, strengthened partnerships, and a broader scope, Menzies now addresses critical health issues across Northern Australia and the Asia-Pacific region.

I would like to congratulate Menzies on its 40 years of outstanding research and impact. As we celebrate this achievement, we can be certain that this commitment to shaping a healthier, more equitable future will continue.

Professor Steve Wesselingh
CEO
National Health and Medical Research Council

Social and environmental factors in 10 Aboriginal communities in the Northern Territory: relationship to hospital admissions of children

Estrella Munoz, Jennifer R Powers, Terry G Nienhuys and John D Mathews

Objective: **To identify social and environmental differences associated with differences in admission rates of children from 10 rural Aboriginal communities in the Northern Territory.**

Design: **Between March 1986 and December 1987, records of hospital admissions of the cohort of children for 1976–1985 were examined retrospectively; cross-sectional measurements of 74 historical, social and environmental characteristics of each community were collected.**

Sample: **All 1961 children born between 1 January 1976 and 31 December 1985 and still living in the 10 communities.**

Method: **Scores on social and environmental factors for each community were generated by factor analysis. Generalised linear interactive modelling was used to investigate the association between these scores and admission rates.**

Results: **Mean admissions per child-year at risk were higher in Central Australian communities (range, 0.41–0.93) than Top End communities (0.26–0.38). Factor I accounted for 30% of the social and environmental differences between communities: communities with a high score on this factor had more houses, fewer shared toilets, more electrical appliances, better personal hygiene and a history of mission administration. High scores on this factor were predictive of lower admission rates and the factor explained most of the differences in admission rates between the Top End and Central Australian communities. Factor VI, correlated with dilapidated dwellings and fewer Aboriginal Health Workers, explained some differences in admission rates between six Top End communities.**

Conclusions: **Social and environmental factors correlated with the degree of community development are associated with the health of Aboriginal children. Improved development programs should be community-controlled and evaluated to identify the social, educational, behavioural and environmental changes that are most effective in improving health.**

(Med J Aust 1992; 156: 529-533)

The poor health of Aboriginal children has been well documented. Aboriginal infant mortality rates are falling, but are still higher than they are for other Australians;[1] patterns of mortality and morbidity for Aboriginal children are similar to those in developing countries,[2-4] with high rates of gastroenteritis, respiratory and other infections.[5,6]

The socioeconomic status of Aboriginal people in Australia is low, with high unemployment[7,8] and low educational achievement.[9,10] A large proportion of Aboriginal people have lived in substandard and overcrowded accommodation with poor water supplies and sanitary facilities;[11,12] these conditions persist in many communities. Inadequate housing and sanitary facilities have been recognised as determinants of poor health, as have unemployment and low educational achievement.[13-16] In international comparisons, better maternal education is strongly associated with better health outcomes after taking account of economic differences.[17]

Health improvements in developed and developing societies are due, in part, to historical improvements in sanitation and living standards.[18,19] Analytical studies have shown that broad criteria such as the degree of socioeconomic development are correlated with health improvements,[20,21] although more specific environmental measures are not always found to be associated with improved health. Such results may reflect methodological problems in the studies,[22] the time lags between social changes and health outcomes, and the complexity of the causal pathways involved; threshold–saturation models suggest that there is a threshold of change which must be reached before health improvements will follow, and a saturation point beyond which further improvements in social and environmental circumstances will not lead to further health improvements.[23]

In a separate paper (see page 524), we have shown that there are substantial differences in hospital admission rates for children from different Aboriginal communities in the Northern Territory.[24] In this paper we examine social and environmental differences between communities to identify the factors that are most strongly associated with high rates of hospital admission. Such information should increase the awareness of Aboriginal people, politicians, community leaders, administrators and health educators of the magnitude of the social, environmental and health differences between communities and of the strong rationale for interventions to improve the health of Aboriginal children.

Methods and results

As described previously,[24] the sample was ascertained in 10 Aboriginal communities in the Northern Territory; each community chosen had a population of more than 70 children under five years of age and required a nurse (E M) between March 1986 and December 1987. All children who were born between 1 January 1976 and 31 December 1985 and who were living in the communities were identified from records in the community health centre.

Hospital admissions for all 1961 children studied were ascertained retrospectively from health centre and hospital records for the period from birth until 31 December 1985 or five years of age, whichever was earlier. To protect privacy, communities have been identified by number; ethical procedures were as described previously.[24]

Hospital admissions

The GLIM (generalised linear interactive modelling) software package was used to calculate the number of admissions per child-year at risk (admission rate) for each community.[25,26] As we have reported for the larger data set that included outstations,[24] communities in Central Australia had higher admission rates than those in the Top End (north of latitude 15°S in the Northern Territory). There were marked differences in the admission rates between communities ($\chi^2_9 = 659.6$; $P < 0.001$).

Social and environmental data

With permission from community councils, information on living conditions and sanitary facilities was obtained from householders; tribal information was obtained from community councils; demographic, social and historical information was obtained from local organisations, government agencies, schools, community health centres, stores, councils, the Department of Aboriginal Affairs and the Australian National Archives.

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To overcome language difficulties, an Aboriginal Health Worker or a council employee assisted at all interviews; all people contacted agreed to participate. Attempts were made to visit all dwellings; when people were not at home, they were revisited whenever possible. Variables that could not be measured were graded by a single observer (E M). The few missing values were replaced by the mean value of observations from other communities.

Historical background

Traditionally, Aboriginal people did not live in fixed settlements. Before 1877, when community C9 settled around a mission, none of these communities had an established settlement. C3 was the last community to be established, in 1969. Six communities settled around missions. The number of tribal groups in each community ranged from one to 15. At the time of the study most of the communities were administered by Aboriginal community councils and all had freehold title to their land.

Location and communication

Two of the six communities in the Top End were accessible by road but only in the dry season (from about May to October); all had regular air flights. Communities in Central Australia were accessible by road and only one had regular commercial flights.

In 1986–1987 three communities in the Top End had telephones, while only one community in Central Australia was not within walking distance of a telephone. Four communities had television reception.

Type of dwelling

Between March 1986 and December 1987, 546 dwellings were visited, representing approximately 75% of the total number of dwellings in these communities. Although the majority of people lived in houses, 15% of the dwellings were humpies (Table 1). There were more humpies in Central Australian than in Top End communities. Table 2 summarises selected social and environmental conditions in the 10 communities.

TABLE 1: Type of dwelling and people per type of dwelling

Type of dwelling	Dwellings	People
House	419 (77%)	3705 (84%)
Shed	44 (8%)	269 (6%)
Humpy	83 (15%)	426 (10%)
Total	546	4400

Dwelling occupancy

Over all 10 communities, the average number of people per dwelling ranged from 5.5 to 10.1 and the average number of adults per dwelling ranged from 3.2 to 6.0. The number of bedrooms per dwelling also differed between communities, but in many houses any floor space was used for sleeping; we recorded the number of bedrooms in houses (for humpies, all "rooms" were used for sleeping), and the number of adults (Table 2) and children per dwelling.

Sanitary facilities

Most houses had inside toilets, but some had access only to communal showers and toilets (Table 2). In some communities up to 32% of showers and toilets were not in working order. Pit toilets were common in Central Australian communities but only in those communities with humpies.

Household facilities

Differences in facilities for food storage, laundry, entertainment and communication were assessed in terms of the percentage of dwellings with refrigerators, washing machines, radio, television and video recorders (Table 2).

Environmental and personal hygiene

Methods of disposal of household waste, and monthly sales of cleaning materials and disposable nappies from the community store were measured; cleaning materials were standardised as kilograms or items sold per year per child in the cohort. Personal hygiene was measured (scale, 1–2) by rating the cleanliness of clothing (Table 2); tidiness of the community was assessed in terms of the amount of visible rubbish.

Health services

Staffing levels of health centres in each community were measured in terms of the number of children in the cohort per Aboriginal Health Worker, per nurse and per doctor-visit per year. Health care was measured by observing and grading (1–3) the follow-up and supervision of treatment for children with diarrhoea and chest infection. Health centre administration was measured (scale, 1–3) by observing the medical sundries stock and turnover of medications.

Education and literacy

School attendance, as a percentage of enrolment, ranged from 41% to 80%. Sales of reading material (newspapers, magazines and comics) from community stores were measured as items sold per child per year. Two communities sold no reading material; community C9 sold the most. At eight of the 10 communities all Aboriginal Health Workers could read and write.

Intoxicating substances

The availability of alcohol and kava and whether petrol sniffing was perceived as a problem was recorded for each community. Observed use and abuse of substances was graded 1–4. Two communities used kava. In five communities health personnel and council members believed petrol sniffing was a problem. Alcohol was sold in only three communities; despite this, inebriation was observed in all but one community.

Food availability

To assess the availability of fresh food, the store in each community was visited every day during the period of data collection; the frequency of availability of fresh fruit and vegetables was graded 1–3. In only four communities could fresh food be bought every day.

Economy

For the majority of families, the main source of income was from social security benefits. However, in some communities, mining royalties (recorded yes/no) and the sale of artefacts and paintings (scale, 1–3) provided extra income.

TABLE 2: Selected social and environmental variables in ten communities

Community	No. of dwellings surveyed	Mean number per dwelling		Percentage of dwellings								Personal hygiene
		Rooms	Adults	Houses	Humpies	Inside toilet	Communal toilet	Shower or bath	Electricity	Washing machine	Fridge	
Top End												
C1	74	2.4	5.9	88%	0	74%	0	73%	73%	27%	43%	good
C2	42	2.5	6.0	93%	2%	93%	0	95%	88%	64%	57%	good
C3	35	1.7	4.6	60%	9%	57%	14%	57%	66%	29%	26%	fair
C4	51	2.6	5.8	96%	0	80%	20%	80%	96%	27%	29%	good
C5	59	2.4	4.9	88%	0	68%	3%	68%	83%	24%	51%	good
C6	26	2.8	5.1	88%	0	88%	12%	88%	100%	65%	88%	good
Central Australian												
C7	62	1.7	3.2	53%	21%	50%	50%	50%	50%	18%	32%	fair
C8	90	2.1	4.4	69%	31%	60%	13%	61%	44%	21%	23%	fair
C9	30	2.2	4.0	97%	0	83%	0	80%	83%	53%	70%	good
C10	77	1.6	3.3	57%	42%	23%	60%	38%	1%	3%	0	fair

Statistical analysis

More variables were measured than there were communities and this would have contributed to statistical "overdetermination" in any attempt to predict hospital admission rates by means of all 74 social and environmental variables. Furthermore, there was a need to summarise and give meaning to the social and environmental measurements.

Accordingly, the data matrix (74 variables by 10 communities) was analysed by principal component factor analysis, a multivariate technique which reduces the large number of variables into a smaller number of factors comprising groups of variables.[27-29] This technique adjusts for the correlation between variables, and each factor is derived so as to be as independent as possible from each of the others.

Based on the community characteristics, the analysis generated a factor score for each community. These community scores were then incorporated into a linear model, as previously fitted under GLIM,[24] to assess whether differences in admissions between communities were correlated with differences in environmental factor scores. Having identified an important factor, those variables with the heaviest loadings on the factor were identified to examine their specific impact on admission rates.

Identification of socioenvironmental factors

Principal component analysis reduced the 74 variables to nine factors, each summarising a different set of characteristics. The variables with higher loadings on a factor ($P<0.05$) are closely related to that factor (see Box). Some of the variables measured were not significantly associated with any of the nine factors; this implies that community differences for these variables were not sufficiently correlated with community differences for other variables.

The meaning to be attached to factors can be illustrated by the loading of variables on Factor I that relate to community development, most particularly housing development. The four communities with the highest scores for this factor were established as missions over 30 years ago and at least 70% of the dwellings were houses (rather than humpies or sheds) with electricity and water connected, internal toilets and showers. Some houses also had washing machines and fridges. At these communities, levels of personal hygiene were good and all Aboriginal Health Workers could read and write. Less developed communities had more humpies, each able to house fewer adults, with communal toilets and without electricity, thus resulting in lower scores on Factor I (Table 2, Box, and Figure).

Factor I was important, firstly because it explained 30% of all the variance (differences) between communities in social and environmental variables, and secondly because it could be interpreted in terms of a single dimension that could be loosely identified with "community development". None of the other factors explained as much of the overall social and environmental variance (see Box), and none could be given such a simple interpretation.

Variables with maximum loadings on factors identified by principal component factor analysis with varimax rotation[29]

Factor I	**v = 29.7%**	
***	Humpies as % dwellings	−0.908
***	Houses as % dwellings	0.883
**	Rooms per dwelling	0.843
**	Adults per dwelling	0.753
*	No. dwellings surveyed	−0.651
	Percentage of dwellings with:	
***	Toilet inside	0.972
***	Shower and/or bath	0.968
***	Water tap inside	0.960
**	Communal toilet	−0.787
***	Electricity	0.944
***	Video	0.871
**	Washing machine	0.842
**	Fridge	0.799
***	Personal hygiene	0.877
*	Literacy of Aboriginal Health Workers	0.721
**	Past DAA administration	−0.750
**	Past mission administration	0.750
*	Year established	0.728
*	Anglican church	0.661
*	Arid zone	−0.630
*	Road open all year	−0.630
NS	Petrol sniffing	−0.542
Factor II	**v = 19.3%**	
***	Kava drinking observed	−0.943
***	Kava availability	−0.918
***	Uniting church	−0.918
*	Children per dwelling	−0.698
*	Detergent for dishes	−0.691
*	School attendance	0.687
*	Rubbish drum outside	0.671
*	Laundry water disposal	−0.654
*	% showers working	−0.628
NS	Laundry detergent	−0.579
Factor III	**v = 11.6%**	
***	Health care	0.924
***	Mops	0.918
**	Detergent for floors	0.831
*	Art and craft	0.733
NS	% children aged 0–5 years	0.585
NS	No toilet	0.501
Factor IV	**v = 11.2%**	
**	Magazines	0.832
**	Radio	0.819
**	Newspapers	0.796
**	Nappies sold (material)	0.770
*	Lutheran church	0.662
*	Public transport	0.637
NS	Tidiness of town	−0.503
Factor V	**v = 8.2%**	
**	No. of tribal groups	−0.775
**	% of taps working	0.743
*	Coastal	−0.721
*	Drunkenness observed	0.702
*	School enrolment	0.684
NS	Alcohol limitations	0.600
NS	Alcohol availability	−0.541
NS	Sniffing observed	0.481
Factor VI	**v = 7.4%**	
**	Dwellings dilapidated	0.787
**	Comics	0.734
*	Child/AHW ratio	0.679
*	Royalties	0.637
*	Fresh food availability	0.618
NS	Baptist church	0.597
Factor VII	**v = 5.1%**	
***	Telephone in community	−0.865
***	Nurses	0.851
*	CHC administration	0.691
*	Availability of doctor	−0.628
NS	Dogs per dwelling	0.544
Factor VIII	**v = 4.7%**	
***	Disposable nappies sold	0.943
**	Pit toilet	0.755
**	Sheds	0.745
Factor IX	**v = 2.8%**	
***	Sewerage	0.910
**	Television	0.787
**	Television reception	0.736
*	Dwellings not surveyed	0.682
NS	Island	0.579
NS	Bleach	0.544

v = percentage variance in social and environmental conditions explained by the factor. NS = not significant; * = $P<0.05$, ** = $P<0.01$, *** = $P<0.001$. These significance levels (derived from normal theory assumptions) may be biased, but they indicate the relative importance of each variable for each factor.

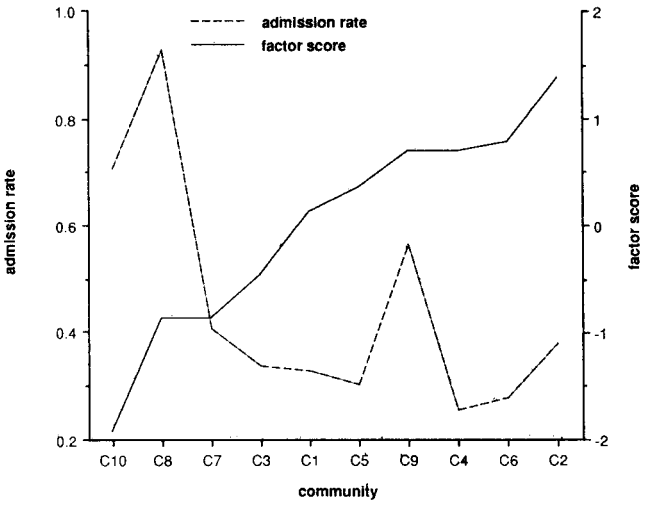

FIGURE: Mean admission rates, 1976–1985, and environmental score for Factor I (see Box) by community.

Socioenvironmental factors and hospital admission rates

Mean admission rates were higher in all four Central Australian communities (C7–C10) than

in any of the six Top End communities(C1–C6). There was greater variation in admission rates in Central Australian communities (range, 0.41–0.93 mean admissions per child-year at risk) than in Top End communities (range, 0.26–0.38) (Figure). Therefore the factor scores for each community were used to explore the relationships between social and environmental conditions and the admission rates of children.

Factors I and VI provided the best discrimination and explained up to 78% of the difference in admission rates between communities. Factor I, measuring aspects of community development, explained 43% of the deviance in admission rates due to communities (Table 3). While Factor I explained most of the differences between admission rates in the Top End and Central Australia as well as the differences between Central Australian communities, it did not explain the differences in admission rates between Top End communities (Figure), probably because there was little variation between Top End communities to be explained.

Factor VI, which included variables related to health centre staffing and empty dilapidated dwellings (see Box), explained a further 35% of the differences in admission rates between communities, and helped to explain community differences in admission rates in both the Top End and Central Australia.

The associations between admission rates and particular variables within Factors I and VI were explored further. Once the effects on admission rates of age, sex and year were removed, it could be shown that variables with the largest loading on Factor I correlated better with admission rates than variables with smaller loadings. Having removed the effects of age, sex, year and region, the proportion of dilapidated dwellings proved to have a stronger correlation with admission rate than any other variables from Factor VI.

Differences in admission rates between Top End communities were best explained by the average number of children for each Aboriginal Health Worker employed in the community (see Box).

The positive association of houses and mean number of adults per dwelling with Factor I and the negative association of this factor with admission rates suggest that Aboriginal children are less likely to be admitted to hospital if they live in an overcrowded standard house than if they live in a humpy. (The paradox in terms of the number of adults per dwelling would have been avoided if we had measured overcrowding in terms of the number of persons per unit area, rather than per dwelling.)

TABLE 3: Statistical summary relating factor scores to admission rates

Variables fitted*	Scaled deviance	Total df†	Deviance change‡	Percentage of community difference explained	Change in df	Regression coefficient§	Standard error of estimate¶
None	3578.6	878	—	—	—	—	—
Age + gender + year	1929.3	864	1649.3	—	14	—	—
+ Factor I score	1645.0	863	284.3	43.1%	1	−0.257	0.019
+ Factor VI score	1417.4	862	227.7	34.5%	1	0.231	0.014
+ Residual due to communities	1269.8	855	147.6	22.4%	7		

*The methods for model-fitting are described in the companion paper;[24] the background is described elsewhere.[26]
†The number of degrees of freedom (df) depends upon the number of informative cells in the data matrix: 5 age groups, 2 sexes, 10 years and 10 communities.
‡The deviance change in a Poisson model follows an asymptotic χ^2 distribution.
§The negative regression coefficient indicates that the admission rate decreases as the Factor I score increases; the positive coefficient for Factor VI indicates that admission rate tends to rise with an increasing factor score.
¶As these regression coefficients are so much larger than their standard errors, the coefficients are certainly of statistical significance.

Discussion

This paper has documented the poor living conditions and social circumstances in 10 Aboriginal communities in 1986 and 1987 (Tables 1, 2). Our major finding is that hospital admission rates for children over the period 1976 to 1985 were highest for those communities with poorer living conditions and less community development (Table 3, Box, and Figure).

Although many community characteristics were strongly associated with differences in admission rates between communities, inferences about the causal significance of individual variables cannot be made easily, because at least some of the associations will be indirect and non-causal.

A second limitation of the study is that data on hospitalisation covered a preceding period of 10 years, while the social and environmental data reflected mainly circumstances at the time the communities were visited; much information on social and environmental conditions in the past was either unavailable or unreliable.

A third limitation is that hospital admission rates are an indirect measure of childhood morbidity; in our previous paper we suggested that community differences in admission rates are due more to differences in morbidity than to community differences in admission policy or practice.[24]

In spite of such potential shortcomings, our study has shown that hospital admission rates for Aboriginal children in the Northern Territory are higher for communities where housing, water supplies, sanitation and electric power are less well developed or maintained, where literacy and hygiene are less, where there are more empty, dilapidated houses and more children for each Aboriginal Health Worker employed. From what is already known about the social and environmental origins of childhood morbidity,[3,21,22] it is very likely that some of these variables contribute directly to higher childhood morbidity and hospital admission rates. Nevertheless, without an intervention study to show that reductions in the prevalence of putative risk factors are followed by reductions in hospital admissions, it is impossible to formally demonstrate the causal status of any of the associated variables.

Indeed, some of the associated variables may reflect the causal importance of other variables, such as those relating to beliefs and behaviour, that were not measured directly in this study. For example, dilapidated housing, a variable loading on Factor VI (see Box), is associated with hospital admissions. In Central Australia, houses become dilapidated when left vacant after a death in the house, whereas in the Top End, ceremonial cleansing allows reoccupation of the house within a short time of the death, so that there is less dilapidation of newer dwellings. Thus dilapidation will be correlated with all consistent differences between Central Australia and Top End communities, including any consistent differences in admission rates, and it is not clear whether the latter differences are partly caused by behavioural differences correlated with dilapidation of housing, or whether the causal pathways are even more indirect.

Nevertheless, our principal finding is irrefutable, namely that childhood morbidity, as measured by high hospital admission rates, was worse in those Aboriginal communities with poorer living conditions and less community development. In one sense, this result is not surprising, as it simply restates, in the context of a comparison between communities in the Northern Territory, the well-documented association of poor health with economic and social disadvantage.[3,17,21,22]

However, our studies are encouraging, firstly because they show that the health outcomes in some Aboriginal communities can be much better than in others,[24] and secondly because it is plausible, on the basis of the associations reported here, that improvements in social, behavioural and environmental conditions in Aboriginal communities will be followed by improvements in childhood health outcomes. There is already a strong rationale for Aboriginal community development on the grounds of social equity; longitudinal evaluations of broadly-based programs of community development are now needed to identify the most effective environmental, social, behavioural and medical strategies for health improvement.

Our cross-sectional findings strongly support the rationale for accelerated social action and community development because of the improved health outcome that will almost certainly follow. As has been observed with other disadvantaged populations,[17] outcomes in Aboriginal communities will improve more rapidly when there is a broadly based social and political commitment to better education and health for all. Programs of community development and social action are likely to be most

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effective when Aboriginal people have themselves acquired the knowledge[30] and are empowered to control the planning and management of changes in their own communities.[31] The social, educational and economic development of Aboriginal communities should continue until the present disparities in living standards and health outcomes between Aboriginal people and other Australians have been eliminated.

Acknowledgements

We thank the Aboriginal Health Workers, nurses, councils and residents in the communities for their support and assistance, the staff in the Department of Health and Community Services and colleagues at the School for their advice and assistance in various aspects of the work. Dr Anthony Smith provided valuable advice in the statistical analysis and Dr Tarun Weeramanthri, Dr Ivan Bastian, Judith Boswell and Sue Hutton provided helpful comments. This work was supported by the Northern Territory Government through its core grant to the Menzies School of Health Research.

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Birth size of Australian Aboriginal babies

Susan M Sayers and Jennifer R Powers

Objectives: **(i) To describe birth size of Aboriginal babies by sex, gestational age, and Aboriginality; (ii) to analyse the results with reference to standards of ponderal index and birthweight for gestational age.**

Subjects: **570 liveborn singletons routinely delivered at Royal Darwin Hospital between January 1987 and March 1991, and recorded in the Delivery Suite Register as being born to an Aboriginal mother.**

Main outcome measures: **Weight, length and head circumference at birth.**

Results: **The mean birthweight was 3098 g (standard deviation, 601 g), peak gestational age was 39 weeks, 13% were low birthweight and 7% were preterm. Preterm rates did not differ significantly for sex and Aboriginality. Babies without a non-Aboriginal ancestor had a lower mean birthweight and at term, were significantly smaller than babies with a non-Aboriginal ancestor as assessed by mean birthweight, length, head circumference and ponderal index. More than a quarter of babies (27%) without a non-Aboriginal ancestor were below the 10th percentile of birthweight for gestational age, compared with 14.2% of babies with a non-Aboriginal ancestor.**

Conclusions: **On the basis of postnatal clinical estimates of gestational age, Aboriginal babies have a preterm rate of 7% and Aboriginal babies without a non-Aboriginal ancestor are smaller in size at birth than babies with a non-Aboriginal ancestor.**

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Birth size and birthweight analyses are of clinical and epidemiological value. The clinician can identify babies at a higher risk of medical problems and organise appropriate intervention and follow-up strategies.[1] Epidemiologically, birth size analysis provides comparative indicators for the evaluation of health care.

Birth size has been studied by various methods, such as birthweight groupings,[2] percentiles of birthweight, length and head circumference for gestational age,[3,4] and the relationship of weight to length for gestational age.[5] There are few detailed studies of Aboriginal babies.

Accurate estimation of gestational age in Aboriginal babies is difficult.[6] Past studies have analysed birthweight without gestational age, concentrating on the low birthweight group;[7,8] other studies use imprecise gestational age estimates to define term and preterm babies.[9,10]

More recent studies have analysed gestational age and birthweight jointly but rely on mothers remembering the date of their last menstruation for gestational age estimation,[11] or sample only a community[12] or low birthweight group.[13]

Significant differences in mean birthweight between babies with and without a non-Aboriginal ancestor have been described,[14,15] but most studies report the Aboriginal population as a homogeneous group.[7-12]

We sought to describe the birth size of Aboriginal babies in terms of sex and Aboriginality using postnatal gestational age estimates and to analyse the results using reference standards of ponderal index[16] and birthweight for gestational age.[17]

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Methods

Subjects

The Darwin Health Region covers an area of approximately 120 000 km². The Aboriginal population is heterogeneous, including urban dwellers whose main language is English and traditional Aboriginal people living in remote communities.

The Royal Darwin Hospital serves a population of approximately 110 000 people, representing 65% of the population of the Northern Territory. The percentage of Aboriginal women having babies outside the hospital in the Darwin Health Region is low; in 1987, 89.2%, and in 1988, 90.7% of Northern Territory Aboriginal mothers delivered in hospital.[18]

Babies were eligible if they were liveborn singletons delivered at the Royal Darwin Hospital between January 1987 and March 1990 to a mother living in the Darwin Health Region and recorded as Aboriginal in the Delivery Suite Register. There were no exclusions. Of the 1053 eligible babies, 445 were not studied in detail because the paediatric investigator (S M S) was absent for these deliveries; 94% of the remaining 608 babies were enrolled in the prospective study. Those studied in detail were not randomly selected, but a binomial model and unpaired *t* tests showed no significant differences between the sex ratios or mean birthweights of subsets and the total population fitting the selection criteria (Table 1).

Procedures

Birthweights and crown–heel lengths were measured by midwives within two hours of delivery. Birthweights were recorded to the nearest gram with a balance scale. The crown-heel lengths were measured with a length-board by the standard anthropometric technique[19] and recorded to the nearest millimetre.

The paediatric investigator examined maternal case notes and, within four days of delivery, interviewed Aboriginal mothers and examined their babies. Information was

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obtained about home location and mothers' knowledge of a non-Aboriginal ancestor).

Gestational age was estimated according to neurological and physical criteria described by the Dubowitz scoring system.[20] Because this method of estimation of gestational age was central to the study, it was evaluated in detail on 344 Aboriginal babies born at the Royal Darwin Hospital.[6] Gestational age was estimated by the paediatric investigator using the Dubowitz scoring system and compared retrospectively with gestational age estimates from the first fetal ultrasound performed by one of seven District Medical Officers.

Two statistical methods for assessing agreement between methods of clinical measurement were used. The intraclass correlation coefficient (ICC), a measure of agreement between methods after adjusting for subject differences,[21] showed that there was good agreement between the Dubowitz and ultrasound estimates for all babies (Table 2), as ICC values of 0.40 to 0.75 represent fair to good agreement and values above 0.75 show excellent agreement.[22] However, the method of Bland and Altman[23] for calculating the mean difference between the two gestational age estimates (Dubowitz estimate minus ultrasound estimate, divided by the number of babies) showed no difference in babies with a non-Aboriginal ancestor, a difference of three days in babies without a non-Aboriginal ancestor and nine days for babies less than 2500 g.

The head circumference was measured by the paediatrician and recorded to the nearest millimetre.

Table 1: Number, sex and birthweight of babies fitting selection criteria

Babies	Boys	Girls	Total	Birthweight in grams Mean	(SD)
Total eligible	552	501	1053	3106	(580)
Paediatrician absent	243	202	445	3112	(562)
Paediatrician present	309	299	608	3102	(593)
Babies missed	16	22	38	3159	(460)
Babies in study	293	277	570	3098	(601)
Babies with Aboriginality and gestational age data	252	250	502	3090	(595)

SD = standard deviation.

Table 2: Agreement of Dubowitz score with ultrasound estimates of gestational age

Category	Number of babies	Mean difference (days) Point estimate	(95% CI)	Limits of agreement (weeks)	Intraclass correlation coefficient
All babies	344	2	1 to 4	-3.3 to 4.0	0.69
Group A	251	3	2 to 5	-3.2 to 4.2	0.66
Group B	93	0	-2 to 3	-3.4 to 3.5	0.77
Birthweight <2500g	54	9	5 to 13	-2.9 to 5.4	0.81

CI = confidence interval.
Group A = babies without a known non-Aboriginal ancestor.
Group B = babies with a known non-Aboriginal ancestor.

Table 3: Mean birthweight (g) by Aboriginality and sex

	Group A			Group B			Unpaired
	n	mean	(SD)	*n*	mean	(SD)	*t* test
Boys	181	3080	(587)	105	3405	(597)	4.412*
Girls	181	2969	(527)	86	3093	(617)	1.683
Total	362	3025	(560)	191	3265	(624)	4.604*

Group A = babies without a known non-Aboriginal ancestor.
Group B = babies with a known non-Aboriginal ancestor.
SD = standard deviation.
*$P < 0.001$.

Definitions

Gestational age: according to the WHO convention,[17] where 36 weeks' gestation means the period from exactly 36 weeks up to 36 weeks and six days.
Low birthweight: below 2500 g.
Preterm: below 37 weeks' gestation.
Term: From 37 to 42 weeks' gestation.
Ponderal Index (pi): a measure of weight in grams (bw) for length in centimetres (length), pi = (bw/length^3) × 100.
Group A: babies without a known non-Aboriginal ancestor.
Group B: babies with a known non-Aboriginal ancestor.

Reference standards

Kitchen's Melbourne-based study provided the reference standard for birthweight for gestational age;[17] the 10th percentile was selected to identify the babies within the cohort who were small for gestational age.

Miller's 10th percentile for ponderal index for gestational age was used to identify babies with reduced soft tissue mass within the cohort.[16]

Analysis

The analysis was performed by means of the statistical packages, GLIM[24] and SPIDA.[25]

Results

Birthweights were available for 570 babies; data on Aboriginality and gestational age were complete for 553 and 506 babies respectively; data on both were available for 502 babies (Table 1). The birthweights of all liveborn babies ranged from 850 to 5340 g. The mean birthweight was 3098 g (standard deviation, 601 g); the median was 3115 g; 13% were low birthweight.

Group B were significantly heavier than Group A (Table 3, Figure 1). Group B boys were significantly heavier than Group A boys, but Group B girls were not significantly heavier than those in Group A (Table 3). For babies for whom there were complete data, analysis of variance was used to examine the effects of Aboriginality and sex on birthweight after taking gestational age into account; Group B boys were still significantly heavier than the other babies.

Group A had a higher rate of low birthweight than Group B (Table 4), but this difference was not significant.

Gestational age for the 506 babies ranged from 26 to 41 weeks with a mean of 38.5 weeks and median of 39 weeks; 7% were preterm. The gestational age distribution was similar for Groups A and B, each showing a peak gestational age of 39 weeks (Figure 2). There were no significant differences in the preterm rate for Group A boys (6.6%) and girls (6.6%) or Group B boys (4.8%) and girls (7.0%).

Mean birthweights for Group B were greater than those for Group A at most gestational ages (Figure 3). Likewise, the mean birthweights for boys were greater than for girls at all gestational ages with sufficient numbers for comparison (data not shown). Mean birthweights could not be calculated for gestational ages below 33 weeks, as only seven babies were born before this gestational age. Only one Group B neonate was born after 40 weeks' gestation.

More than a quarter of Group A (90/333) were below the 10th percentile of birthweight for gestational age compared with 14.2% of Group B (24/169). Binominal models showed significantly more babies in Group A than in Group B were below the 10th percentile of birthweight for gestational age.

There were 467 babies born at term. Babies in Group A were significantly smaller than those in Group B as assessed by mean weight, length and head circumference at birth and mean ponderal index (Table 5).

Table 4: Number and percentage of low birthweight babies by Aboriginality and sex

	Low birthweight	Total	Percentage
Group A			
Boys	22	181	12.2%
Girls	31	181	17.1%
Total	53	362	14.6%
Group B			
Boys	7	105	6.7%
Girls	11	86	12.8%
Total	18	191	9.4%

Group A = babies without a known non-Aboriginal ancestor.
Group B = babies with a known non-Aboriginal ancestor.

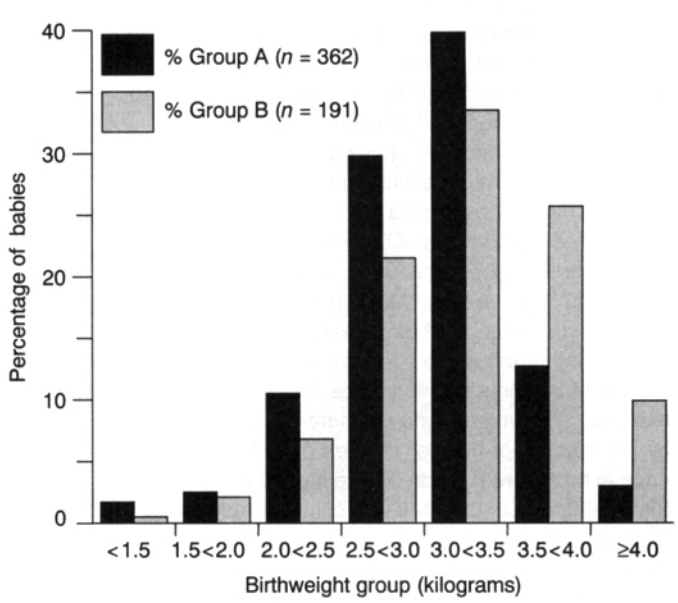

Figure 1: Birthweight distribution by Aboriginality.

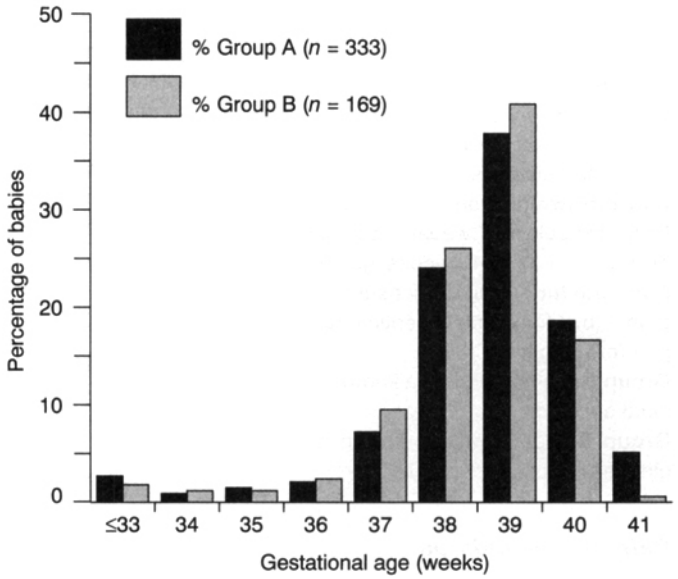

Figure 2: Gestational age distribution by Aboriginality.

Discussion

Previous reports of Aboriginal birth size have not identified babies with non-Aboriginal ancestors, nor adequately addressed the accuracy of gestational age estimations. By exploring these areas, we have made a more informative analysis of Aboriginal birth size.

Gestational age is usually estimated from the mother's recall of her last menstrual period, and verified with an early ultrasound measurement.[26] Nevertheless, a number of factors can invalidate the gestational age calculated by these methods.[27] Of the measurements used to classify babies at birth, the estimated gestational age is often the most unreliable. Aboriginal women rarely record the date of their last menstrual period,[28] and early ultrasound is impossible because they frequently present late for antenatal care.[6,9,29] Therefore other methods of estimating gestational age are needed.

At Royal Darwin Hospital, fetal ultrasound measurements are used to estimate fetal maturity for the obstetric management of Aboriginal pregnancies and the Dubowitz scoring system is used to estimate the gestational age of Aboriginal babies.

Although fetal ultrasound measures size rather than maturity, gestational age estimates based on fetal ultrasound measurements are reliable, particularly if done in the first trimester.[30] Ethnic differences are reported,[31] and Aboriginal fetal measurements have been shown to be less than those

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Table 5: Size at term by Aboriginality

	Group A			Group B			Unpaired
	n	mean	(SD)	*n*	mean	(SD)	*t* test
Birthweight	309	3099	(462)	158	3320	(549)	4.57*
Birth length	306	49.1	(2.2)	157	49.8	(2.4)	3.21*
Head circumference	307	33.9	(1.4)	158	34.3	(1.6)	2.47†
Ponderal index	306	2.60	(0.25)	157	2.67	(0.26)	2.62*

Group A = babies without a known non-Aboriginal ancestor.
Group B = babies with a known non-Aboriginal ancestor.
SD = standard deviation.
*$P < 0.01$.
†$P = 0.05$.

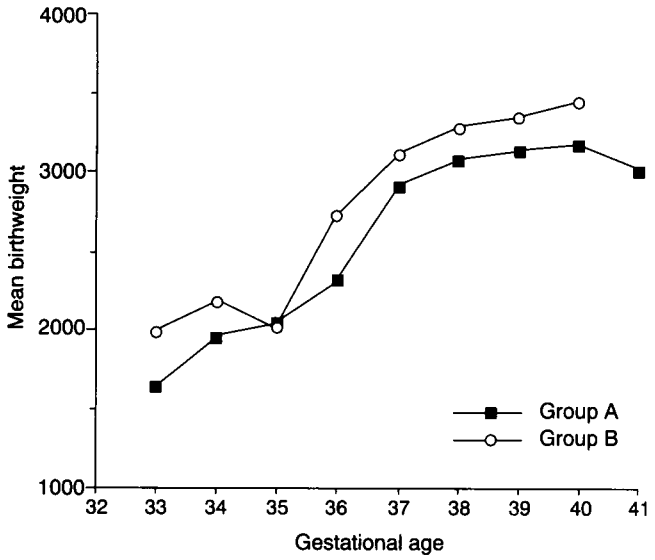

Figure 3: Mean birthweight by gestational age and Aboriginality.

of a United States population.[28] Gestational age estimates of Aboriginal babies based on fetal measurements with reference to Caucasian standards may underestimate maturity.

The postnatal Dubowitz method is widely used in clinical practice and scores criteria of physical and neurological maturation as an indirect measure of the duration of gestation.[20] Previous studies have established the accuracy of the Dubowitz scoring system in non-Aboriginal populations for which there are reliable last menstrual period data, for a mixture of Bantu, Indian and Malay infants in Capetown, Nigerian born infants, and Rhodesian born Africans.[20,32,33] There are suggestions that maternal and fetal factors influence maturation criteria[34,35] so that clinical assessment is unreliable over the entire gestational age range,[36] and conflicting reports, specifically about the Dubowitz score overestimating gestational age in low birthweight babies.[37-39]

There was good agreement between gestational age estimates based on the Dubowitz scoring system and estimates based on fetal ultrasound measurements taken throughout pregnancy. There was no difference between Dubowitz and ultrasound estimates for Group B babies, but Dubowitz estimates were greater than ultrasound estimates by two to five days for Group A babies (Table 2). While this represents a bias of three days, this is unlikely to be clinically significant. An intraclass correlation coefficient of 0.81 indicated excellent agreement between the Dubowitz and ultrasound estimates for low birthweight babies, but there was a mean difference of nine days between them with a 95% confidence interval of 5–13 days and a limit of agreement of –2.9 to 5.4 weeks. This represents a bias of clinical significance.

The Dubowitz method, which is based on the maturity of the baby, may overestimate the gestational age, or ultrasound, which uses the fetal size, may underestimate the gestational age, or both gestational age estimates may have some inaccuracy, as their true values are unknown. While there are inherent difficulties in both methods, it is likely that Dubowitz estimation by one paediatrician is more consistent than the fetal measurements performed by multiple medical officers.

In this study the preterm rate of 7% is lower than rates reported in other studies of Aboriginal births (11% in South Australia,[9] 11%–22% for seven communities in Queensland,[40] 16% in Western Australia[11] and 21.5% in the Northern Territory).[10] The methods of estimating gestational age are not reported in two studies.[9,40] The methods in the other retrospective studies were case note review of fundal heights[10] and mothers' recall of last menstrual date,[11] and are more likely to misclassify small babies as preterm.[41] None the less, because many pregnancies in this study could be considered stressed (because of the high proportion of babies below the 10th percentile of birthweight for gestational age), the lower preterm rate may be due to adverse maternal factors accelerating the maturation characteristics of the Dubowitz score. However, the 13% low birthweight rate resembles rates reported in developing countries,[42] where (according to a study of 11 developing nations which did address the issue of accuracy of gestational age) low birthweight rates above 10% have been attributed to increased numbers of small babies rather than increased numbers of preterm deliveries.[42]

Babies with and without a non-Aboriginal ancestor both had a peak gestational age of 39 weeks. Surprisingly, despite different methods of estimating gestational age, this peak of 39 weeks has been described previously for Aboriginal babies.[11,12] Most births are recorded world-wide at 40 weeks' gestation but as meticulous estimation of gestational age is not routine it is likely that some data are based on unreliable estimations of gestational age and that 40 weeks is arbitrarily chosen to record a term neonate. Nevertheless, there is little evidence that Afro-American women have a shorter gestational period than white American women.[43]

The similarity of the gestational age distributions for babies with and without a non-Aboriginal ancestor (Figure 2) suggests that the shorter gestational length is due to environmental rather than genetic factors. It is possible that the Dubowitz score underestimates gestational age over 40 weeks as this has been reported of a method of gestational

age estimation similar to the Dubowitz method;[44] this bias would be in the opposite direction to that postulated for the low birthweight group at the opposite end of the gestational age range.

Most studies report the Aboriginal population as homogeneous. Like other health institutions, the Royal Darwin Hospital records a child as Aboriginal if the mother identifies herself as an Aborigine (defined as a person of Aboriginal or Torres Strait Islander descent who identifies herself as an Aborigine or Torres Strait Islander and is accepted as such by the community in which she lives).[45] Unknown numbers of babies with an Aboriginal ancestor, born to white mothers and Aboriginal fathers, are classified as white. The proportion of babies classified as Aboriginal with a non-Aboriginal ancestor is also unknown, although there is evidence that this proportion is increasing. A preliminary figure from the 1991 census suggests an increase of 61% in the Aboriginal population in the previous 10 years.[46] This increase is significantly greater than the growth of the overall Australian population.[46] Part of this increase may be due to improved census taking in remote areas, but most is an increased willingness of people with an Aboriginal ancestor to identify themselves as Aboriginal.[46]

In our study direct questioning of the mother about knowledge of a non-Aboriginal ancestor showed that a third of Aboriginal babies had an identified non-Aboriginal ancestor.

Babies without a non-Aboriginal ancestor are lighter in weight than those with a non-Aboriginal ancestor (Figure 1) and these birthweight differences are not due to gestational age differences (Figures 2 and 3).

Internal cut-offs by means of percentiles are used to identify high risk babies in a known population, but percentiles of birthweight for gestational age could not be generated from this cohort as there were insufficient numbers in the lower gestational ages. Prolonged collection of more data would be inconsistent as there has been a change in low birthweight rates over time at the Royal Darwin Hospital.[7] Commonwealth Department of Health intrauterine growth charts[47] were considered, but parity and maternal height data were only recorded in the obstetric case notes of 361 babies. Consequently the Melbourne based reference was used to identify the babies who were small for gestational age. As it is likely that Aboriginal babies have a growth potential similar to other non-Aboriginal Australian babies,[48] it is disturbing to note that, against this reference, 27% of babies without a non-Aboriginal ancestor were below the 10th percentile.

The detailed analysis of the term babies was possible because of the larger number of babies (467). The term babies without a non-Aboriginal ancestor were significantly smaller and had a lower ponderal index than those with a non-Aboriginal ancestor (Table 5). These differences in birth size suggest that babies without a non-Aboriginal ancestor have different risk factors and long-term outcomes to those with a non-Aboriginal ancestor. We are now studying the outcomes of these subgroups and their relationships to birth antecedents such as maternal nutrition, living conditions, familial birth size patterns and smoking.

Conclusion

Currently, State and Territory health statistics regard the Aboriginal population as a homogeneous group, but babies with a non-Aboriginal ancestor are different in birth size to those without a non-Aboriginal ancestor. Failure to define these different Aboriginal neonatal populations may produce inaccurate information about their health status.

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INDIGENOUS HEALTH RESEARCH

Reducing premature death and renal failure in Australian Aboriginals

A community-based cardiovascular and renal protective program

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ABSTRACT

Objective: To describe results of a systematic treatment program to modify renal and cardiovascular disease in an Aboriginal community whose rates of renal failure and cardiovascular deaths are among the highest in Australia.

Design: Longitudinal survey of people during treatment, and comparison of rates of natural death and renal failure with those in a historical control group.

Setting: Tiwi Islands (population, about 1800), November 1995 to December 1998.

Participants: All adults with blood pressure ⩾ 140/90, with diabetes and urinary albumin/creatinine ratio (ACR) ⩾ 3.4 g/mol (microalbuminuria threshold), or with progressive overt albuminuria (ACR ⩾ 34 g/mol) were eligible for treatment. The historical control group comprised 229 people who satisfied these criteria in the pretreatment period 1992–1995.

Interventions: Perindopril, combined with calcium-channel blockers and diuretics if needed to achieve blood pressure goals; attempts to improve control of blood glucose and lipid levels; health education.

Main outcome measures: Blood pressure, ACR, serum creatinine level and glomerular filtration rate (GFR) over two years of treatment; rates of renal failure and natural death compared with control group (analysed on intention-to-treat basis).

Results: 258 people enrolled in the program, and 118 had complete data for two years of treatment. In these 118, blood pressures fell significantly, while ACR and GFR stabilised. Rates of the combined endpoints of renal failure and natural death per 100 person-years were 2.9 for the treatment group (95% CI, 1.7–4.6) and 4.8 for the control group (95% CI, 3.3–7.0). After adjustment for baseline ACR category, the relative risk of the treatment group versus the control group for these combined endpoints was 0.47 (95% CI, 0.25-0.86; $P = 0.013$). Treatment benefit was especially marked in people with overt albuminuria or hypertension and in non-diabetic people. The estimates of benefit were supported by a fall in community rates of death and renal failure.

Conclusions: Aboriginal people can participate enthusiastically in chronic disease management, with rapid, dramatic improvement in clinical profiles and mortality. Similar programs should be introduced urgently into other Aboriginal communities nationwide.

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ABORIGINAL PEOPLE in the Northern Territory are experiencing an epidemic of cardiovascular disease (CVD) and end-stage renal disease (ESRD). Age-standardised CVD death rates are three times those of non-Aboriginal people,[1] while the incidence of treated ESRD in Aboriginal people is approaching 1000 per million, and doubling every four years.[2] ESRD treatment costs, at $100 000 per person annually, are becoming a huge burden,[3] but premature death is the greater human catastrophe.

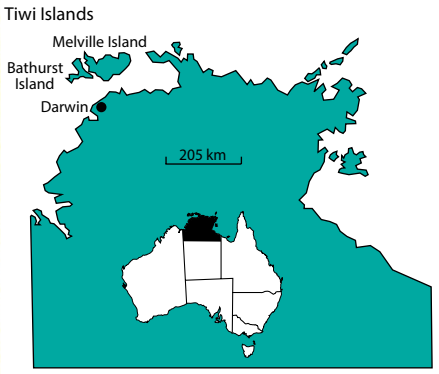
These problems are especially serious in the communities of the Tiwi Islands, north of Darwin (population, about 1800) (Box 1). The incidence of ESRD among Tiwi people recently reached 2700 per million, and they have one of the highest CVD mortality rates in Australia.[2,4] In a community-wide screening program starting in the early 1990s, we found a high prevalence of cardiovascular risk factors, including type 2 diabetes and hypertension, and albuminuria (measured by the albumin/creatinine ratio (ACR) of a random urine specimen).[5] Albuminuria correlated inversely with glomerular filtration rate (GFR), and its intensity predicted not only renal failure, but also cardiovascular deaths and all-cause natural deaths.[6-9]

In the early 1990s, use of antihypertensive drugs was increasing gradually in the Tiwi communities, but systematic management of the huge burden of morbidity identified by the screening program was beyond the capacity of the existing health services. In November 1995, we therefore introduced a systematic treatment program to reduce blood pressure and to modify the expression and progression of renal and cardiovascular disease. We describe the results of this program to the end of 1998.

METHODS

The study was a longitudinal survey of people in the Tiwi Islands communities

1: The "Top End" of the Northern Territory of Australia



during treatment, and comparison of endpoints with a historical control group. Treatment was offered to eligible people as part of improved standard care. All participants gave informed consent to have their course followed up for the projects *The epidemiology and prevention of Aboriginal renal disease, Parts 1 and 2*. These projects were approved by the Joint Institutional Ethics Committee of the Menzies School of Health Research and Territory Health Services, Darwin, and its Aboriginal subcommittee, and by the Tiwi Land Council (Part 1) and the Tiwi Health Board (Part 2).

Treatment program

The program relied considerably on screening and treatment algorithms. Interventions included education about diet, exercise, health behaviours and medical treatment. Medical treatment centred around use of a long-acting angiotensin-converting enzyme inhibitor (ACEi) (perindopril; Coversyl [Servier]), aggressive blood pressure control,[10,11] and, where appropriate, oral hypoglycaemic and lipid-lowering drugs. The choice of an ACEi was based on the well recognised antihypertensive and cardiovascular-protective effects of this class of drug[12] and several reports, subsequently substantiated, of an additional renal protective effect.[13-23]

If antihypertensive drugs had been prescribed before entry into the study, they were discontinued or tapered when perindopril was started. Objectives were to achieve a minimum daily dose of 4 mg perindopril and to lower blood pressure, initially to < 130/85, but more recently to < 120/75.[10] A stepped approach to achieve these blood pressures included increasing perindopril to 8 mg, with addition of long-acting calcium-channel blockers and/or diuretics if needed.

Participants were seen at least monthly while medications were introduced or changed, then at least every three months for the first year, and at least every six months thereafter. Each examination included a minimum of a brief history, medication review, and measurement of weight, blood pressure, urinary ACR and serum creatinine level and, in diabetics, evaluation of blood glucose control.

After a start-up period, the day-to-day program was largely conducted by local health workers and community project officers, who were supported by telephone contacts and regular visits by nurse coordinators from Darwin. Doctors, who reviewed eligibility assessments, supported or made treatment decisions and modified the protocols, were less intensively involved. The program has run in parallel with other clinic activities in Nguiu, Bathurst Island, but has been integrated into regular clinic activities at the Melville Island communities of Milikapiti and Pirlangimpi.

Participants

Treatment group: People eligible for ACEi therapy were those with:

- hypertension (blood pressure ≥ 140/90 mmHg);
- diabetes and ACR ≥ 3.4 g/mol (microalbuminuria threshold), regardless of blood pressure; or
- progressive overt albuminuria (ACR ≥ 34 g/mol on first testing and increasing over time), regardless of blood pressure or diabetes status.

All qualifying features needed to be confirmed on at least two occasions.

People with past adverse reactions and breastfeeding women were ineligible for ACEi therapy. Fertile women were advised about teratogenic risks and the options of contraception or discontinuation of ACEi medication early in unplanned pregnancy. People with serum creatinine levels over 250 µmol/L were considered ineligible for long-acting ACEi therapy in the first six months of the program, but were later enrolled when treatment proved safe and effective in people with mild and moderate renal insufficiency.

To some extent, enrolment was prioritised by disease severity. Thus, most people with overt albuminuria, uncontrolled blood pressure and renal insufficiency were enrolled in the first year of the program.

2: Baseline characteristics of participants in the treatment program and the historical control group

	All participants (*n* = 258)	Included in 2-year profiles		Historical control group (*n* = 229)	*P*†
		Yes (*n* = 118)	No* (*n* = 140)		
% Men	43%	47%	40%	51%	0.13
Mean age in years (SD)	43.4 (11.1)	43.5 (10.4)	43.4 (11.7)	40.8 (12.8)	0.02
Mean body mass index (kg/m²) (SD)	27.0 (5.7)	27.1 (5.7)	27.0 (5.8)	25.2 (5.4)	< 0.001
Blood pressure (mm Hg)					
Mean systolic (SD)	135 (20)	135 (20)	135 (21)	134 (20)	0.58
Mean diastolic (SD)	82 (14)	81 (13)	82 (15)	85 (15)	0.01
% With hypertension‡	65%	66%	63%	65%	0.91
% With diabetes	42%	46%	37%	26%	0.001
% With ACR ≥ 34 g/mol	65%	74%	58%	58%	0.02
% With raised serum creatinine level§	12%	13%	11%	12%	0.62
Previous ACEi	25%	33%	16%	NR	

ACR = urinary albumin/creatinine ratio. ACEi = angiotensin-converting enzyme inhibitor. NR = no result. *90 had been enrolled less than 2 years, 19 had been enrolled ≥ 2 years but did not have complete data for all visits, and 31 had dropped out. †For test of significance of difference between all participants (intention-to-treat group) and control group. ‡Blood pressure ≥ 140/90 or taking antihypertensive treatment. §Serum creatinine level > 106 µmol/L (women), > 120 µmol/L (men).

3: Clinical profiles over two years of treament in 118 Tiwi people

Variable	Baseline	6 months	12 months	24 months	P*
Blood pressure (mm Hg)					
Mean systolic (SD)	135 (20)	126 (21)	124 (20)	122 (22)	<0.001
Mean diastolic (SD)	81 (13)	75 (14)	77 (14)	74 (14)	<0.001
Mean† ACR (g/mol) (95% CI)	55 (43–70)	50 (39–64)	53 (41–69)	55 (43–69)	0.36
Mean† serum creatinine level (μmol/L) (95% CI)	89 (85–93)	88 (84–92)	88 (84–92)	84 (79–89)	0.44
Mean GFR (mL/min/1.73 m²) (SD)	89 (26)	91 (28)	89 (26)	93 (29)	0.54

ACR = urinary albumin/creatinine ratio. GFR = glomerular filtration rate.* Test for significance of difference in values among the four intervals by analysis of variance. † Geometric mean.

Control group: In the absence of a parallel control group, rates of renal failure and natural death in participants were compared with those of a historical control group from the pre-program period. This control group comprised adults whose results on a single screening examination between July 1992 and September 1995 met the eligibility criteria later used for the treatment program. Selection was blinded to their future course, which was followed to 30 October 1995.

Data analyses

Analyses were performed using STATA statistical software.[24] Clinical profiles in the treatment group were described at baseline, six, 12, and 24 months of treatment, regardless of compliance, and were compared by analysis of variance, using geometric means for ACR and serum creatinine level to normalise their distribution. All endpoint data in the treatment group were analysed on an intention-to-treat basis. Rates of natural death and renal failure were calculated by baseline ACR category for the intention-to-treat and control groups, and the risk ratios for the intention-to-treat group calculated in stratified analysis by ACR category by the Mantel–Haenszel method for cohort studies. Kaplan–Meier survival curves for both groups were derived, and survivals compared by the non-parametric Wilcoxon technique.

RESULTS

Enrolment

By 31 December 1998, 258 people had enrolled in the program (29% of all adults in the island communities) and 227 were still participating. Of these, 39 had completed over three years of treatment, 137 over two years, 168 over one year, and 192 over six months. Of 31 dropouts, nine had died, seven had begun dialysis (two of whom later died), seven had stopped taking the medication because of side effects (cough in four; angioedema, itching and dizziness in one each), four became normotensive without treatment, two chose to quit, one moved, and one entered palliative care with osteomyelitis of the skull.

Characteristics of people who enrolled are shown in Box 2: 42% had diabetes, almost two-thirds had hypertension, with a quarter already prescribed enalapril, and almost two-thirds had overt albuminuria.

Medications and participation

Doses of perindopril prescribed for the 227 people participating at the end of 1998 were 2 mg (5 people; 2%), 4 mg (72; 32%), and 8 mg (150; 66%). Calcium-channel blockers were being taken

4: Clinical profiles over two years of treatment in 118 Tiwi people, by clinical category at baseline

		Blood pressure		Previous ACEi	Diabetes		Albuminuria		Serum creatinine level	
		<140/90 (n=69)	≥140/90 (n=49)	(n=39)	No (n=64)	Yes (n=54)	Micro-* (n=28)	Overt† (n=86)	Normal (n=98§)	Raised‡ (n=15§)
BP (mm Hg)										
Mean systolic (SD)	Baseline	123 (11)	152 (17)	136 (19)	135 (21)	136 (19)	134 (24)	135 (18)	135 (20)	139 (19)
	24 months	117 (20)	130 (22)	128 (23)	122 (15)	122 (23)	123 (23)	120 (21)	123 (22)	118 (20)
Mean diastolic (SD)	Baseline	75 (9)	90 (12)	83 (11)	81 (15)	81 (10)	82 (15)	81 (12)	82 (13)	78 (13)
	24 months	72 (14)	77 (13)	78 (14)	76 (15)	72 (11)	73 (15)	74 (14)	75 (14)	71 (13)
Mean¶ ACR (g/mol) (95% CI)	Baseline	66 (50–87)	43 (27–61)	62 (40–97)	48 (34–68)	62 (45–91)	16 (13–19)	104 (90–121)	49 (38–65)	125 (83–187)
	24 months	75 (57–98)	35 (23–54)	66 (45–98)	50 (36–70)	60 (41–87)	19 (15–25)	90 (72–112)	50 (38–66)	88 (45–174)
Mean¶ serum creatinine level (μmol/L) (95% CI)	Baseline	88 (82–94)	90 (85–95)	94 (85–103)	90 (85–95)	87 (81–93)	85 (79–92)	90 (85–95)	83 (82–88)	137 (123–153)
	24 months	84 (77–92)	84 (77–91)	93 (82–105)	83 (77–89)	86 (78–94)	75 (70–81)	88 (82–95)	78 (75–81)	119 (98–144)
Mean GFR (SD) (mL/min/1.73 m²)	Baseline	91 (29)	86 (21)	91 (31)	90 (26)	88 (26)	88 (23)	89 (28)	95 (23)	55 (18)
	24 months	95 (32)	91 (25)	92 (32)	96 (28)	90 (24)	102 (24)	91 (28)	99 (24)	57 (30)

ACEi = angiotensin-converting enzyme inhibitor. BP = blood pressure. ACR = urinary albumin/creatinine ratio. GFR = glomerular filtration rate.
*ACR, 3.4–33 g/mol. †ACR ≥ 34 g/mol. ‡Serum creatinine level > 106 μmol/L (women), > 120 μmol/L (men). §Data not available for all participants. ¶Geometric mean.

5: Rates of endpoints in historical control and intention-to-treat groups

Endpoint	Baseline ACR (g/mol)	Historical control group (n=229) Cases	Person-years	Rate per 100 person-years (95% CI)	Intention-to-treat group (n=258) Cases	Person-years	Rate per 100 person-years (95% CI)	Relative risk (RR) (95% CI) Crude*	Adjusted†	P (for adjusted RR)
Dialysis	All	9	564	1.6 (0.8–3.1)	7	548	1.3 (0.6–2.6)	0.77 (0.24–2.33)	0.43 (0.17–1.12)	0.08
	⩾100	9	120	7.5 (3.9–14.4)	7	197	3.6 (1.7–7.0)			
Natural death	All	18	555	3.2 (1.0–5.1)	11	560	2.0 (1.1–3.4)	0.59 (0.25–1.31)	0.55 (0.26–1.16)	0.11
	<34	2	226	0.9 (0.2–3.5)	3	178	1.7 (0.5–5.2)			
	34–99	9	205	4.4 (2.2–8.4)	3	194	1.5 (0.4–4.8)			
	⩾100	7	124	5.6 (2.7–11.2)	5	188	2.7 (1.1–6.4)			
Combined (dialysis or natural death)	All	26‡	543	4.8 (3.3–7.0)	16	549	2.9 (1.7–4.6)	0.59 (0.30–1.14)	0.47 (0.25–0.86)	0.01
	<34	2	227	0.9 (0.2–3.5)	3	178	1.7 (0.5–5.2)			
	34–99	9	205	4.4 (2.3–8.4)	3	193	1.6 (0.5–4.8)			
	100–199	6	74	8.1 (3.6–18.0)	3	104	2.9 (0.9–8.9)			
	⩾200	9	37	24.2 (12.6–46.5)	7	73	9.6 (4.6–20.2)			

ACR = urinary albumin/creatinine ratio. * Overall estimate for treatment group versus control group. † Relative risk adjusted for baseline ACR category. ‡ People who underwent dialysis and later died were counted only once for the combined endpoint.

6: Estimated survival advantage for intention-to-treat versus control group, adjusted for ACR category*

Clinical category at baseline	Relative risk (95% CI)†	P
All	0.47 (0.25–0.86)	0.01
Overt albuminuria	0.36 (0.18–0.72)	0.004
Diabetes		
No	0.28 (0.09–0.93)	0.02
Yes	0.65 (0.28–1.51)	0.31
Hypertension		
No	0.59 (0.25–1.44)	0.24
Yes	0.38 (0.15–0.98)	0.04

*ACR = urinary albumin/creatinine ratio. Categories: <3.4, 3.4–33, 34–99, 100–199, ⩾200 g/mol. †For combined endpoints of natural death and renal failure, intention-to-treat versus control group.

by 37 people (16%), diuretics by 15 (7%), and both by 13 (6%). Participation was enthusiastic, and compliance increased over time; 65% were taking >70% of their prescribed dose (assessed by pill counts and interview), 27% were taking medicine occasionally, and 7% were taking little or no medication at the end of 1998.

Two-year clinical profiles

Of the 137 people who had been treated for at least two years, 118 had largely complete follow-up data and were included in the two-year profile. These 118 were well matched with participants not included in this profile for age, BMI, and blood pressure, but were more likely to have diabetes, overt albuminuria, and to have been taking prior ACEi therapy (Box 2). These differences reflected prioritisation of sicker people for early entry into the program.

Two-year clinical profiles for the 118 people are shown in Box 3. Treatment was associated with a swift and sustained fall in blood pressure, as well as stabilisation of ACR and GFR. Results are presented according to participants' clinical categories at baseline in Box 4. The fall in blood pressure was marked in people with hypertension at baseline and less marked but still apparent in those who had been normotensive, as well as in those previously prescribed an ACEi. Good blood pressure responses were seen in people both with and without diabetes, those with micro- and overt albuminuria and those with "normal" and raised levels of serum creatinine. Stabilisation of ACR and GFR was seen in all clinical categories. Indeed, serum creatinine level tended to fall and GFR to rise in all categories.

Baseline weight did not change (mean, 74 kg; SD, 16 kg), while mean serum potassium level rose non-significantly from 4.04 mmol/L (SD, 0.46 mmol/L) to 4.14 mmol/L (SD, 0.49 mmol/L). No one developed significant hyperkalaemia. There was no evidence that ACEi therapy accelerated progression to renal insufficiency.

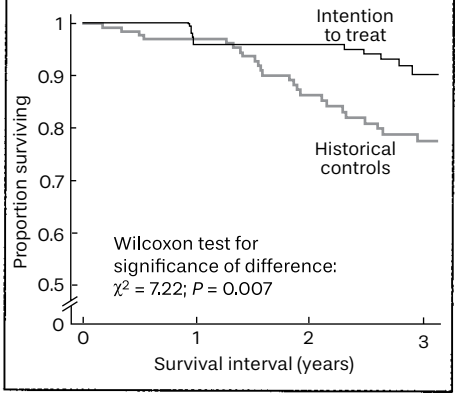
Comparisons with control group

Two hundred and twenty-nine people qualified as controls from the pre-program period, comprising 123 people who subsequently went onto the treatment program and 106 people who did not. Reasons for not going onto the program included death, dialysis, failure to qualify on subsequent examinations, presence of exclusion criteria (eg, pregnancy, breastfeeding), declining treatment, or moving.

Baseline characteristics of the control and intention-to-treat groups are compared in Box 2. The control group was younger at enrolment, had lower BMI, and included fewer people with diabetes or overt albuminuria. The control group was followed up for a total of 564 years (individual mean, 2.5 years; range, 1 month to 3.3 years) and the intention-to-treat group for 560 years (individual mean, 2.2 years; range, 2 weeks to 3.1 years).

Endpoints of the two groups are compared in Box 5. The treatment

7: Kaplan–Meier survival estimates for Tiwi people with overt albuminuria at baseline



group as a whole had lower rates of dialysis, natural death and the combined endpoint (dialysis or death) than the control group, although the differences were not significant. However, rates of endpoints were strongly correlated with baseline ACR category. Indeed, renal failure necessitating dialysis was confined to people with ACR ⩾ 100 g/mol at baseline, and in these people the treatment group had an estimated 57% lower dialysis rate than the control group. In contrast, rates of natural death and of the combined endpoint were lower in the treatment group than in the control group for all categories of baseline overt albuminuria. After adjustment for ACR category, the treatment group had an estimated 45% lower rate of natural death and an estimated 53% lower rate of the combined endpoint.

Box 6 shows estimates of the survival advantage in people with various baseline clinical profiles after adjustment for ACR category. Treatment benefit was strong in people with overt albuminuria, non-diabetic people and people with hypertension. It was less marked in diabetic or normotensive people.

Survival estimates for people with overt albuminuria at baseline are shown in Box 7. Although the intention-to-treat group showed attrition during the first year (representing ESRD and deaths of seriously ill people prioritised for early entry), a survival advantage over the control group was clear by two years of the treatment program.

DISCUSSION

This study found that the introduction of a systematic treatment program to the Tiwi Island communities was associated with marked improvements in blood pressure and stabilisation of renal function in people receiving treatment. These changes contrasted sharply with the increase in blood pressure and ACR and fall in GFR noted previously in people matched for ACR category in the pretreatment status quo.[10]

The treatment program was also associated with a swift and dramatic decrease in rates of renal failure and natural death in the treated group compared with a historical control group, suggesting that the program prevented or at least delayed these outcomes. Further evidence for the existence of this estimated survival benefit was the decrease in community-wide rates of ESRD and natural death — previously increasing — after introduction of the program (Box 8). In contrast, ESRD continued to increase among non-Tiwi Aboriginal people in the Top End (Box 9), arguing against a chance background effect. Preliminary estimates of cost effectiveness of the program, based solely on avoidance or delay of dialysis, are already startling.[3,25]

These results show that Aboriginal people are interested in health issues and receptive to health messages, and will take medications over the long term to protect against future health risk, with excellent response.

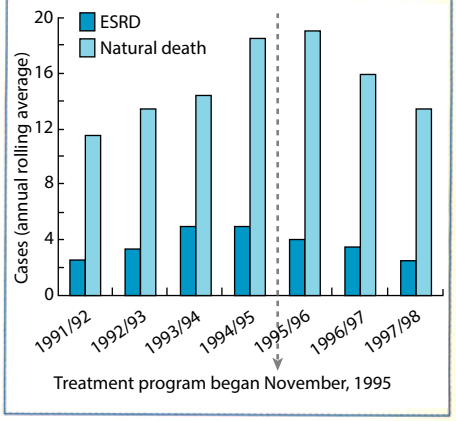
They also show that a systematic approach, with testing and treatment algorithms and clear goals, is superior to the previous approach of gradually improving medical management. While we cannot apportion relative benefit to individual elements of the treatment program, the observed fall in blood pressures alone would be expected to markedly reduce cardiovascular deaths and progression of renal disease,[10,11] compatible with the effects we found.

Our intention-to-treat analyses probably underestimate the therapeutic efficacy of treatment, as a third of the intention-to-treat group took the prescribed medications only occasionally or not at all. Use of the historical control group was also a potential source of bias. On the one hand, it may have also led to underestimates of treatment benefit because of the group's potentially better survival prospects, based on its younger mean age, milder disease and the probable inclusion of people with borderline blood pressure or ACR readings, as eligibility for the group was not confirmed by a second examination. On the other hand, the 123 controls who subsequently entered the treatment program might have had superior survival characteristics to the controls who did not enter the program, potentially inflating the apparent benefit of the program.

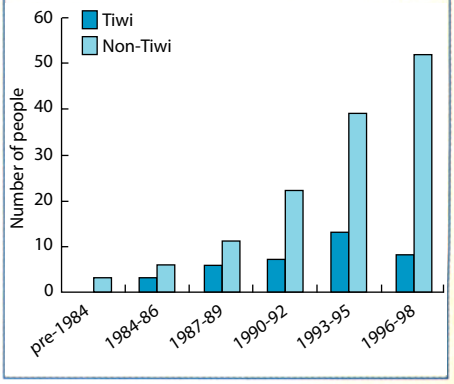
Another source of bias was the prioritisation of the sickest people for early enrolment in the treatment program, many of whom were failing previous management regimens. This predisposes to poor short term outcomes of the program and underestimates of its benefits. Analyses of program results at four and five years, when more people have passed through one to two years of treatment, will dilute the impact of these early events. Longer-term analyses will also be needed to evaluate any survival effect of treatment in people without overt albuminuria, and the extent to which treatment has delayed rather than prevented ESRD and death in people with overt albuminuria.

The program could still be improved. Blood pressure control should be better; at two-year follow-up, 31% of people had blood pressures ⩾ 140/90, and 50% had blood pressures > 120/75.[10] Hypertension, and therefore eligibility for treatment even in the absence of albuminuria, should probably be redefined as blood pressures > 130/80 in this high-risk population.[10] Control of blood glucose and lipid levels needs to

8: New cases of end-stage renal disease and natural deaths in Tiwi adults aged 20 years and over



9: Aboriginal people starting dialysis in the "Top End" of the Northern Territory



improve. Finally, we might reassess the notions of the maximally renal-protective dose of ACEi and/or add other renal-protective drugs, such as angiotensin II receptor blocking agents,[26,27] for poor responders.

Much of the success of this particular program derives from a strong sense of community ownership and control, a non-judgemental, non-authoritarian style, and respect for competing personal and community perspectives and priorities. Individuals appreciate personalisation of their health goals, and many are slowly adopting lifestyle changes.

This program is now being integrated into normal clinic activities at Nguiu. Its protocols have also been incorporated into standard care guidelines for Aboriginal adults in the Top End of the NT.[28] Extension of its principles to other Aboriginal communities with high burdens of disease nationwide is a matter of urgency.[29] Allocation of adequate resources is a challenge, but the clinical benefit and cost-effectiveness mandate the short- and intermediate-term investment.

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RESEARCH

Diagnostic and therapeutic procedures among Australian hospital patients identified as Indigenous

Joan Cunningham

ABSTRACT

Objectives: To determine whether hospital patients identified as Indigenous are less likely than other inpatients to have a principal procedure recorded, and the extent to which any disparity in procedure use can be explained by differences in patient, episode and hospital characteristics.

Design: Retrospective analysis of routinely collected administrative data from the National Hospital Morbidity Database (NHMD).

Setting: Australian public and private hospitals.

Patients: All patients included in the NHMD whose episode type was recorded as acute and whose separation occurred between 1 July 1997 and 30 June 1998. Patients admitted for routine dialysis treatment were excluded.

Main outcome measure: Whether a principal procedure was recorded.

Results: In public hospitals, patients identified as Indigenous were significantly less likely than other patients to have a principal procedure recorded, even after adjusting for patient, episode and hospital characteristics (adjusted odds ratio [OR], 0.67; 95% CI, 0.66–0.68). This disparity was apparent for most diseases and conditions. In private hospitals, no significant difference was observed (adjusted OR, 0.94; 95% CI, 0.83–1.06).

Conclusions: The disparity in procedure use after adjustment for relevant factors indicates that in Australian public hospitals there may be systematic differences in the treatment of patients identified as Indigenous.

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SEVERAL STUDIES in other countries have shown that some groups of hospital patients, such as African-Americans and women, are less likely than white male patients to receive a variety of diagnostic and therapeutic procedures.[1-12] Disparities have been found for procedures for treating heart disease,[1-5] for organ transplantation,[6-7] and for orthopaedic[8-11] and gastrointestinal procedures.[8,12]

Little research in this area has been done in Australia, and the extent to which such disparities exist for Indigenous patients compared with non-Indigenous patients is unknown, in part because of incomplete identification of Indigenous patients in hospitals in most jurisdictions.[13] However, a recently published national report on hospital separations for the financial year 1997–98 (for which I was a co-author) noted that patients identified as Indigenous were less likely than other admitted patients to have a principal procedure recorded (45% v 75%, after excluding admissions for routine dialysis treatment).[14] This finding was not the focus of the report, and we did not consider more than one patient characteristic at a time or look at more specific illnesses and conditions. Moreover, no information was available about hospital type and size.

Here, I report a more detailed analysis of hospital separations for 1997–98, which examines and adjusts for a larger number of factors. The aim was to assess the extent to which observed disparities in the probability of having a recorded hospital procedure could be explained by differences in patient, episode and hospital characteristics.

METHODS

Description of the dataset

Data were obtained from the National Hospital Morbidity Database (NHMD), which is managed by the Australian Institute of Health and Welfare (AIHW) and includes information on characteristics, diagnoses and care of admitted patients in almost all public and private hospitals in Australia. NHMD records are based on separations (episodes of care) rather than individual patients; a given patient may have multiple separations within the same year.[15]

Data for this analysis relate to hospital separations between 1 July 1997 and 30 June 1998. All data on diagnoses and procedures for that year were coded using the coding scheme of the ninth revision of the *International classification of diseases*, clinical modification (ICD-9-CM).[16] Data for 1998–99 were not used, as two different versions, ICD-9 and ICD-10, were in use in Australia in that year.[17] ICD-9-CM coding was used rather than Australian national diagnosis-related groups (AN-DRGs), because AN-DRGs are determined in part by whether a procedure has been performed.[18]

Permission to access, analyse and publish data was sought and received from all States and the Northern Territory. The Australian Capital Territory (ACT) denied permission, but this jurisdiction accounted for only 0.4% of separations of patients identified as Indigenous (and 1.3% of all separations) in 1997–98.[15]

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Only separations of patients whose episode type was recorded as acute (as opposed to rehabilitative, palliative or other care) were included. Separations for dialysis visits (ICD-9-CM code V56) were excluded, as this code is based on a procedure rather than a diagnosis. Most are same-day admissions of a few hours' duration, and most dialysis-visit separations are accounted for by many repeat visits by a relatively small number of patients.

Public and private hospitals were considered separately because of large differences in apparent use of private hospitals by Indigenous and non-Indigenous people, as well as differences in the levels of recorded procedures for all patients. Analysis of private hospitals was limited to New South Wales, Queensland, South Australia and Western Australia, as only these jurisdictions had recorded any acute, non-dialysis separations of patients identified as Indigenous. No information on Indigenous status of patients was available for 1997–98 for private hospitals in Victoria, and no data were available for the single private hospital in the Northern Territory.

Variables of interest

The outcome of interest was any recorded principal procedure. According to the *National health data dictionary*, the principal procedure is the most significant procedure performed for treatment of the principal diagnosis (Box 1).[19] If no procedure is performed for treatment of the principal diagnosis, then a principal procedure should be selected according to a hierarchy based on type of procedure (therapeutic or diagnostic/exploratory) and whether the procedure is related to the principal diagnosis.[19] Thus, if any procedures are recorded for a given episode of care, a principal procedure should be included in the NHMD, although it may or may not be related to the principal diagnosis.

Explanatory variables of interest related to characteristics of the patient, the episode of care, and the hospital. Patient characteristics included age group, sex, area of residence[20] and Indigenous status as recorded by the hospital (Box 2). Studies in individual hospitals have shown that the proportion of Indigenous patients correctly identified varies widely, from below 50% to almost

1: Principal diagnosis and principal procedure — an example

A 27-year-old man is admitted as a public patient to a major referral hospital with abdominal pain, nausea and vomiting. After examination, an abdominal x-ray is performed, and he is provisionally diagnosed as having acute appendicitis. He has an appendicectomy and the diagnosis is confirmed. Three days after surgery, while still in hospital, he develops a wound abscess, which requires incision and drainage. For this patient, the principal diagnosis is acute appendicitis (ICD-9 code 540) and the principal procedure is appendicectomy (ICD-9 code 47.0).

2: Proportion of separations with a principal procedure recorded — Australian public and private hospitals, 1997–98

	Public hospital patients		Private hospital patients*	
	Identified as Indigenous (*n* = 107 793)	Other (*n* = 3 121 305)	Identified as Indigenous (*n* = 3199)	Other (*n* = 1 172 555)
Overall	44.7%	68.9%	89.1%	88.6%
Sex				
Male	43.0%	68.5%	88.3%	89.3%
Female	45.9%	69.2%	89.6%	88.0%
Age group (years)				
Under 1	27.7%	43.3%	48.9%	47.2%
1–14	39.5%	58.8%	83.1%	82.6%
15–34	47.9%	65.6%	90.4%	89.4%
35–54	48.4%	74.1%	92.8%	91.2%
55–64	48.4%	77.1%	93.8%	92.5%
65 and over	44.8%	71.1%	84.5%	86.4%
Place of residence†				
Urban	60.0%	71.8%	93.2%	89.3%
Rural	41.6%	64.2%	83.1%	86.9%
Remote	39.9%	54.4%	95.6%	92.6%
Unknown	58.4%	70.0%	83.5%	83.0%
Same-day admission				
Yes	53.8%	78.2%	97.7%	95.9%
No	42.3%	63.2%	74.2%	80.6%
Patient accommodation‡				
Private	74.6%	78.0%	—	—
Public	44.4%	67.8%	—	—
Other/unknown	47.5%	71.4%	—	—
Hospital category‡				
Principal referral	66.7%	75.8%	—	—
Major	53.3%	67.1%	—	—
Medium	42.1%	64.3%	—	—
Small	25.8%	35.6%	—	—
Sub- and non-acute	12.2%	21.3%	—	—
Psychiatric	39.0%	34.6%	—	—
Other/unknown	32.6%	67.6%	—	—

* New South Wales, Queensland, South Australia and Western Australia only. † Based on Rural, Remote and Metropolitan Area classification. ‡ Public hospitals only. Hospital categories are based on 1998–99 data supplied by the Australian Insitute of Health and Welfare.

100%.[14] The focus of this analysis is on patients *identified* as Indigenous rather than all Indigenous patients.

Hospital characteristics included type of hospital and hospital category (public hospitals only) (Box 2).[17]

Characteristics of the episode included principal diagnosis (ICD-9-CM codes), whether or not it was a same-day admission, and, for public hospital patients, patient accommodation status (Box 2).

Statistical analysis

Statistical analysis was performed using Stata.[21] Logistic regression was used to assess the relationship between explanatory variables of interest and the probability of having a principal procedure recorded. Odds ratios (OR) and 95% CI are reported. Public and private hospitals were analysed separately. Public hospital data were further stratified by principal diagnosis at the level of ICD-9-CM chapters (eg, circulatory diseases, injury) and for 23 more specific groups of conditions (eg, asthma, epilepsy) for which there were at least 500 separations of patients identified as Indigenous.

Ethical approval

The study was approved by the Joint Institutional Ethics Committee of the Royal Darwin Hospital and the Menzies School of Health Research.

RESULTS

A total of 4 867 368 acute, non-dialysis separations were recorded in Australia (excluding the ACT) in 1997–98. About 66% of these were for public hospitals and 34% were for private hospitals. Overall, 75% of separations had a principal procedure recorded. In about 2% of separations the patients were identified as Indigenous, with 97% of these being recorded for public hospitals.

Public hospitals

In public hospitals, a principal procedure was recorded in 68% of separations. The proportion was considerably lower for patients identified as Indigenous (45% of separations) than for other patients (69% of separations). A difference was apparent regardless of sex, age, place of residence, type of admission, patient accommodation status, or hospital category (Box 2). For all patients, procedures were more likely to be recorded in principal referral and other major hospitals, for same-day admissions, for private patients and for patients from urban areas.

After adjusting for the factors shown in Box 2, patients identified as Indigenous were significantly less likely than other patients to have a principal procedure recorded, both overall and for every ICD-9-CM chapter, except infectious/parasitic diseases and injury (Box 3). The difference was especially marked for diseases of the circulatory, digestive and genitourinary systems and for congenital anomalies, with adjusted odds ratios of about 0.5 for each of these disease categories. In general, adjustment for hospital category resulted in a greater attenuation of the odds ratios for Indigenous status than did adjustment for other factors.

There are important heterogeneities within ICD-9-CM chapters with respect to the appropriateness of and need for procedures. Although it is critical to look at more specific diseases and conditions, it is difficult to do so because of the relatively small numbers of separations of patients identified as Indigenous for most principal diagnoses. Box 4 presents the relative odds of having a recorded principal procedure for conditions with at least 500 separations of patients identified as Indigenous. For each disease/condition, patients identified as Indigenous were less likely than other patients to have a principal procedure recorded. After adjusting for other factors, the dispari-

3: Unadjusted and adjusted relative odds of having a principal procedure recorded for patients (separations) identified as Indigenous in public hospitals, 1997–98

Principal diagnosis (ICD-9-CM codes)	Separations identified as Indigenous	Odds ratio (OR) of having a principal procedure recorded for Indigenous compared with other patients: Unadjusted OR	Adjusted* OR (95% CI)
All diagnoses (001–999, V1–V82†)‡	107 793	0.37	0.67 (0.66–0.68)
Infectious/parasitic (001–139)	4 604	0.49	1.06 (0.97–1.16)
Neoplasms (140–239)	2 044	0.48	0.60 (0.52–0.69)
Endocrine/nutritional (240–279)	3 198	0.41	0.81 (0.73–0.89)
Blood, blood-forming organs (280–289)	721	0.24	0.79 (0.65–0.96)
Mental disorders (290–319)	5 976	0.55	0.86 (0.80–0.92)
Nervous (320–389)	5 194	0.22	0.64 (0.60–0.69)
Circulatory (390–459)	5 839	0.43	0.53 (0.50–0.57)
Respiratory (460–519)	15 411	0.39	0.80 (0.77–0.84)
Digestive (520–579)	8 260	0.31	0.52 (0.49–0.54)
Genitourinary (580–629)	5 812	0.29	0.50 (0.47–0.53)
Pregnancy and childbirth (630–676)	14 117	0.56	0.68 (0.66–0.71)
Skin, subcutaneous tissue (680–709)	4 805	0.46	0.89 (0.83–0.96)
Musculoskeletal (710–739)	2 802	0.34	0.59 (0.54– 0.65)
Congenital anomalies (740–759)	605	0.32	0.50 (0.40–0.63)
Certain perinatal conditions (760–779)	1 871	0.72	0.86 (0.77–0.96)
Ill-defined (780–799)	6 985	0.41	0.67 (0.63–0.71)
Injury and poisoning (800–999)	14 811	0.61	0.96 (0.93–1.00)
Other reasons for contact (V1–V82†)	4 704	0.18	0.58 (0.54–0.63)

* Adjusted for sex, age group, same-day admission, patient accommodation, hospital category, area of residence. † Excluding V56, visits for dialysis. ‡ Includes 34 separations without a principal diagnosis specified.

ties were reduced or eliminated (and changed direction in some cases), but, for 12 of the diseases/conditions, patients identified as Indigenous remained significantly less likely than other patients to have a principal procedure recorded.

Private hospitals

Most separations (89%) in private hospitals in New South Wales, Queensland, South Australia and Western Australia had a principal procedure recorded, and the proportion was similar regardless of recorded Indigenous status across a range of other variables (Box 2).

Patients identified as Indigenous were not significantly less likely than other patients to have a principal procedure recorded, either before or after adjusting for sex, age group, same-day admission, and place of residence (unadjusted odds ratio [OR], 1.05; 95% CI, 0.94–1.18; adjusted OR, 0.94; 95% CI, 0.83–1.06). There were too few separations of patients identified as Indigenous to allow for separate analysis by ICD-9-CM chapter.

DISCUSSION

My analysis confirms a preliminary report[14] that patients identified as Indigenous are less likely than other patients to have a principal procedure recorded, at least in public hospitals. This disparity is partly explained by characteristics of the patient, the episode and, to a larger extent, the hospital, but a considerable difference remains. Within some disease categories, patients identified as Indigenous had only half the odds of other patients in public hospitals of having a procedure recorded, even after adjusting for other factors.

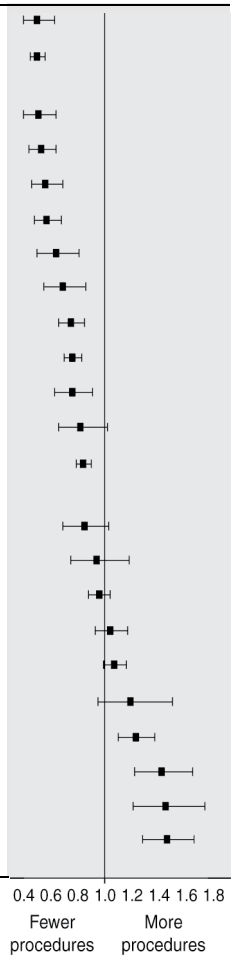
In private hospitals, the probability of having a recorded procedure was similar for all patients. This may reflect the influence of private health insurance. Patients in private hospitals were more likely than those in public hospitals to have a procedure recorded, regardless of whether they were identified as Indigenous. Within public hospitals, private rather than public patients were more likely to have a principal procedure recorded (especially those identified as Indigenous). However, most patients identified as Indigenous were public patients in public hospitals, the group least likely to have a procedure recorded. This is consistent with the relatively low rate of private health insurance coverage of Indigenous people in Australia.[13]

These results from Australian public hospitals are largely consistent with previous studies in the United States showing a significantly reduced probability of having a range of procedures among African-American hospital patients.[8,11,12] Another study found that whites had higher utilisation rates for more discretionary procedures, while blacks had higher rates for medical rather than surgical admissions.[9] Finally, a study found that, among patients considered eligible, based on discharge diagnosis, for high-technology procedures with scope for clinical discretion, blacks were significantly less likely to receive five of the nine procedures, and were not significantly more likely to receive any.[6]

The disparity between patients identified as Indigenous and other patients is real, but the appropriate response depends on the reason or reasons for the disparity. Although several relevant factors (including age, sex, area of residence, same-day admission, patient

4: Relative odds (adjusted and unadjusted) of having a principal procedure recorded for patients (separations) identified as Indigenous in public hospitals, by principal diagnosis,* 1997–98

Principal diagnosis (ICD-9-CM codes)	Odds ratio (OR) of having a principal procedure recorded for Indigenous compared with other patients: Unadjusted OR	Adjusted† OR (95% CI)
Pelvic inflammatory disease (614–616)	0.19	
Alcohol/drug-related psychoses, dependence, abuse (291–292, 303–305)	0.41	
Fracture of radius and ulna (813)	0.52	
Gastritis and duodenitis (535)	0.14	
Disorders of the back (720–724)	0.26	
Epilepsy (345)	0.29	
Bronchitis, emphysema (490–492)	0.23	
Schizophrenic disorders (295)	0.50	
Completely normal childbirth (650)	0.55	
Fractures, dislocations, sprains, strains (800–848)	0.56	
Cholelithiasis (574)	0.52	
Nephritis, nephrotic syndrome, nephrosis (580–589)	0.51	
Open wounds, intracranial, internal, blood vessel injuries (850–904)	0.61	
Acute myocardial infarction (410)	0.67	
Suppurative otitis media (382)	0.31	
Infections of skin and subcutaneous tissue (680–686)	0.74	
Asthma (493)	0.64	
Pneumonia and influenza (480–487)	0.47	
Cerebrovascular disease (430–438)	0.54	
Acute respiratory infection (460–466)	0.82	
Intestinal infectious diseases (001–009)	0.96	
Concussion (850)	0.84	
Diabetes (250)	0.83	



*Includes relatively specific diseases/conditions with more than 500 separations of patients identified as Indigenous. † Adjusted for sex, age group, same-day admission, patient accommodation status, hospital category, and area of residence.

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accommodation status, type of hospital and, to some extent, principal diagnosis) have been accounted for in the analysis, there remain other important factors which could not be adequately measured using routinely collected data. Most importantly, it was not possible to control for whether a procedure was clinically indicated. Even within individual ICD-9-CM codes, there is considerable heterogeneity of disease severity, appropriate care, etc.

The recorded principal procedure may have been for a condition other than the principal diagnosis, but, given the high burden of morbidity among Indigenous Australians,[13] this would more easily explain a higher rather than a lower probability of patients identified as Indigenous having a procedure recorded. It is also possible that some procedures were performed but not recorded.

Decisions about procedures should generally be made in consultation with the patient. It was not possible in this analysis to determine the role played by patient choice, but informed decision-making by patients requires adequate understanding of available options. For some Indigenous patients, this may be limited by communication difficulties due to patient–doctor differences in language, culture, priorities, and so on. One possible indication of failed communication processes and/or lack of shared understanding is that patients identified as Indigenous are much more likely than other patients to leave hospital against medical advice.[14] Inadequate communication can lead to potentially useful procedures not being performed, as well as to procedures being performed on patients who did not fully consent.

Having a procedure is not always better than not having one. Concerns about overservicing and unnecessary surgery have been raised,[22] and the AIHW monitors variation in rates of sentinel procedures.[17] However, given the relatively high mortality rate of Indigenous Australians,[13,23] it seems unlikely that the lower probability of having a (recorded) procedure has resulted in overall health benefits for this group.

In my analysis, it was only possible to distinguish between patients identified as Indigenous and other patients. It is not known to what extent the results apply to Indigenous patients who were not correctly identified and therefore included in the "other" group. It could be argued that the experiences of such people are less relevant if discriminatory treatment is responsible for any of the disparity. However, not all discrimination is interpersonal (ie, the result of individual behaviours).[24] Institutional factors ("the system") may also result in unfair treatment, often unintentional, for members of some groups. For example, any underservicing in remote areas disproportionately affects Indigenous people simply because they are more likely than other Australians to live there.[13] The potential for discrimination exists at multiple points within the healthcare system, including access to services, diagnosis, referral, treatment and outcome. Work is urgently needed to characterise more fully the nature, level, sources and consequences of institutional and interpersonal discrimination so that we can reduce unfair treatment, ensure equitable care and improve outcomes for the most disadvantaged Australians.

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COMPETING INTERESTS

None declared.

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INDIGENOUS HEALTH

Sharing the true stories: improving communication between Aboriginal patients and healthcare workers

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ABSTRACT

Objectives: To identify factors limiting the effectiveness of communication between Aboriginal patients with end-stage renal disease and healthcare workers, and to identify strategies for improving communication.

Design: Qualitative study, gathering data through (a) videotaped interactions between patients and staff, and (b) in-depth interviews with all participants, in their first language, about their perceptions of the interaction, their interpretation of the video record and their broader experience with intercultural communication.

Setting: A satellite dialysis unit in suburban Darwin, Northern Territory. The interactions occurred between March and July 2001.

Participants: Aboriginal patients from the Yolngu language group of north-east Arnhem Land and their medical, nursing and allied professional carers.

Main outcome measures: Factors influencing the quality of communication.

Results: A shared understanding of key concepts was rarely achieved. Miscommunication often went unrecognised. Sources of miscommunication included lack of patient control over the language, timing, content and circumstances of interactions; differing modes of discourse; dominance of biomedical knowledge and marginalisation of Yolngu knowledge; absence of opportunities and resources to construct a body of shared understanding; cultural and linguistic distance; lack of staff training in intercultural communication; and lack of involvement of trained interpreters.

Conclusions: Miscommunication is pervasive. Trained interpreters provide only a partial solution. Fundamental change is required for Aboriginal patients to have significant input into the management of their illness. Educational resources are needed to facilitate a shared understanding, not only of renal physiology, disease and treatment, but also of the cultural, social and economic dimensions of the illness experience of Aboriginal people.

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DOCTOR–PATIENT COMMUNICATION, by creating good interpersonal relationships, allowing the exchange of information and facilitating treatment-related decisions, is fundamental to optimal medical care.[1] Effective communication correlates with improved outcomes, including physiological criteria such as levels of blood pressure and blood sugar.[2] Conversely, professional, language and cultural barriers can impede communication.[3,4]

Few investigators have studied the extent and consequences of miscommunication in Australian Aboriginal healthcare,[5] an area in which effective communication is extremely important.[6] Previous studies involving interviews with service providers and Aboriginal patients have identified significant concerns about communication.[7-9] Some researchers have identified an acceptance, as the *norm*, of a grossly deficient standard of cross-cultural communication.[8] We believe that previous studies, based as they have been on indirect reporting or simulated interactions[10] (rather than direct observation and analysis of the interaction itself), probably understate the degree of miscommunication. The communication gap may be so wide, and so ingrained in healthcare, that it is not even perceived by staff.[11] Similar misunderstandings in Australian court cases often go unrecognised by the participants.[12]

In our study of staff–patient interactions in a dialysis unit in Darwin, NT, we attempted to develop a more informed understanding of intercultural communication between Aboriginal patients and non-Aboriginal staff and to devise strategies for improvement.

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METHODS

Participants and setting

The participants were patients and staff of a satellite dialysis unit in suburban Darwin. The interactions on which our study is based occurred between March and July 2001. The patients came from the Yolngu language group in north-east Arnhem Land. Five interactions were videotaped, each involving a single patient (although family members were present on two of these occasions). Four interactions involved a single staff member and one involved a doctor and a nurse. The interviews occurred at the dialysis unit and at a remote Aboriginal community several hundred kilometres from Darwin.

Design

We used qualitative research methods to reflect the perspectives of all participants. The research design drew on "grounded theory", which describes the inductive process of identifying analytical categories to describe and explain key issues as they emerge from the data.[13] Hypotheses were developed from the ground up,

rather than being defined a priori, as is usually done in *quantitative* research.

Recognising that the effectiveness of communication is inextricably connected with structural issues of poverty, dispossession, marginalisation, low educational achievement and racial discrimination,[7] we chose a "participatory action" approach. This is a style of research in which the demarcation between "researcher" and "subject" is blurred, research design is negotiated, and the participants perceive the need to change and are willing to participate actively in the change process.[14] The research process is illustrated in Box 1.

Sampling

Five clinical interactions, identified beforehand in consultation with both patients and staff, were selected. These concerned diagnosis, treatment and chronic disease management. Staff were asked to follow their usual practice regarding the use of interpreters. The interactions included two medical reviews (one with a patient on regular haemodialysis and one with a patient with chronic renal disease close to needing maintenance dialysis), two education sessions (a nurse providing feedback on blood-test results and a consultation between an allied health professional and a new patient), and an interaction between a nurse and a patient during dialysis.

We selected participants using a "maximum variation sampling approach", wherein a small sample is selected to reflect maximum diversity across specified attributes.[16] The participants covered as wide a range as possible in terms of age, sex, duration of renal experience (receiving or providing treatment), degree of familiarity with the culture and language of the other group, and experience in cross-cultural communication.

Collection of data

The five interactions were videotaped and analysed by all participants, the research team and professional interpreters. Multilayered descriptions of the interactions were constructed from these varied perspectives.

After each interaction, the participants were interviewed separately, in their first language, to explore their perceptions of the effectiveness of the communication. The post-interaction ("exit") interviews were conducted by A L (for English speakers) and B M (for Yolgnu speakers). Semi-structured, in-depth interviews were also conducted with most staff and patients to develop a greater understanding of their backgrounds and wider experience.

Informed consent was obtained from all participants before videotaping. B M obtained verbal and written consent in the patients' own language.

Analysis

The data from all sources were integrated to explore the extent of miscommunication; the cultural, linguistic and systemic factors influencing communication; the effectiveness of communication strategies being used; and possible strategies for improving communication.

The video descriptions and interview transcripts were entered into QSR NVivo,[15] a computer software package that assists in managing qualitative data. Categories used in analysis were derived primarily from the data and through sequential analysis. To strengthen the validity of our analysis, we used "triangulation" (the comparison of results from two or more different methods of data collection) and "respondent validation" (cross-checking *interim* findings with the participants).[17,18]

Ethical approval

The study was approved by the ethics committees of the Menzies School of Health Research at the Royal Darwin Hospital and the Northern Territory University.

1: The research process

* Obtaining consent, exit interviews, feedback and in-depth interviews were undertaken with participants in their first language

RESULTS

A picture emerged of serious miscommunication, often unrecognised by participants, regarding fundamental issues in diagnosis, treatment and prevention. Although there were many differences of goals and structure observed in the interactions, common themes relating to miscommunication emerged. Factors impeding communication included lack of control by the patient, differing modes of discourse, dominance of the biomedical model, lack of shared knowledge and understanding, cultural and linguistic

distance, lack of staff training in intercultural communication, and failure to call on trained interpreters (see Box 2 and Box 3).

Lack of control by the patient

In each interaction, it was the *staff* who controlled the time, place, participants, purpose, structure, topics and language, as well as the form and style of discourse. There were few opportunities for the patients to initiate or influence the agenda. The staff decided whether or not interpreters would be required, even when unaware of the patient's fluency in English.

Differing modes of discourse

Western modes of discourse dominated, with Yolngu modes being marginalised or excluded. Question-and-answer routines, central to Western discourse, do not feature commonly in Yolngu discourse, particularly in relation to personal topics. In Yolngu discourse, the question-and-answer approach is complicated by factors such as cultural restrictions on who may ask for, or give, specific information. It is generally considered impolite to directly contradict or to respond negatively, particularly in encounters of unequal power or when the participants lack a close relationship. The patients in our study repeatedly gave responses that they believed the staff wanted to hear, a practice known in linguistics as "gratuitous concurrence".[19] Triangulation showed that these responses did not represent the patient's true feelings or experience, but were attempts to give "required" or "correct" responses, as in the following example:

> *Physician:* How much are you drinking? How much water?
>
> *Patient:* Little bit water tea, little bit ga bilin ["that's it"].
>
> *Physician:* How much each day? Water, tea?
>
> *Patient:* Three cup, two cup, little bit [said very confidently].

The physician believed that the patient had a clear understanding of the question and was describing the amount of fluid drunk daily. However, it later became clear that the patient responded this way because she knew what was expected. Her understanding of fluid restriction was that she should drink only two cups of "fizzy drink" per day, but that drinking tea or water whenever she felt like it was acceptable. Questions requiring a "yes"/"no" response were particularly susceptible to gratuitous concurrence. A nurse made the following comment:

> I never even considered that they might be saying "yo" [yes] when they are really saying "no". I never even thought of it.

Dominance of the biomedical model

The discourse in the interactions focused on renal function, renal failure, monitoring of and adherence to dialysis, and dietary and medication regimens. Non-medical aspects were excluded or marginalised. Yolngu priorities, which emerged in subsequent interviews and informal discussions, were social, cultural and economic, relating primarily to (currently) unavoidable relocation to Darwin if patients wished to access necessary treatment. One patient illustrated her problems with living in Darwin:

> I told her [the staff member] the truth . . . that I wasn't getting enough [food]. When I get my allowance, they take all the money for accommodation and leave only $30 for food — that's not enough.

Yolngu priorities, which directly affect clinical management, were rarely raised, and, when raised, were either not pursued or were brushed aside. Patients had no explicit opportunities to discuss their own approaches to managing their health. For example, in two interactions, they attempted to talk about Yolngu knowledge and management practices (related to traditional foods), but their contributions were either not understood or not acknowledged.

2: Sample interaction (A)

Setting: The doctor's office in a remote community 500 kilometres from Darwin.

Participants: Mr "A", a 24-year-old man with chronic renal disease who recently had a prolonged admission to Royal Darwin Hospital, during which he required temporary dialysis. He lives with his mother and grandmother, and is fluent in Yolngu languages but not in English. He will need relocation to Darwin within two years for maintenance dialysis.

Dr "B", a 38-year-old male physician with many years' experience working with multicultural and Aboriginal patients.

The interaction: The 20-minute interaction, in English, was initiated by Dr B, who did most of the talking. The patient's mother and grandmother assisted with communication. Mr A and his family asked no questions and gave limited, non-verbal responses to the doctor's questions.

Communication goals: Dr B had clear goals:

I wanted to reinforce that [the patient] was at risk of progression to end-stage renal disease and that he would benefit from treatment, of blood pressure in particular . . . and treatment of other things like anaemia. . . . The main thing was that he doesn't need dialysis at the moment, but that he needed to be monitored and to take his tablets.

The expectations of Mr A and his family were unclear. It later became apparent that they believed that his disease had been cured during his admission. They had no appreciation of its chronicity and of his need for regular tests and medications.

The participants' assessment: Dr B was uncertain of the outcome of the interaction:

Perhaps his mother got some idea . . . I hope they at least understand he is at risk of needing more dialysis. I think they now understand he has kidneys that aren't working so well . . .

After the consultation, the Yolngu researcher discovered that the family's understanding of the doctor's advice was that Mr A should be taking medication. Despite Dr B's extended explanation of chronicity and prognosis, the interaction did not achieve a shared understanding of the state of the patient's kidneys, the significance of test results or the importance of blood pressure control. The family had understood little. This prompted the Yolngu researcher to recall Dr B to explain further, while she provided interpreting assistance.

Consequences for clinical management: Miscommunication reduced the ability to actively engage Mr A and his family in controlling his blood pressure, in retarding progression of his renal disease and in planning for future dialysis. Lack of effective communication about the need to relocate to Darwin for treatment, away from family and community, could result in the patient's reluctance to accept dialysis in the future.

3: Sample interaction (B)

Setting: The open waiting area at the dialysis unit.

Participants: Ms "C", a 50-year-old woman who had been on dialysis for five years. She speaks Yolngu languages and is fluent in conversational English. She has graduate qualifications as a teacher.

Sr "D", a 31-year-old female nurse with 10 years' experience in renal services, both as a nurse and patient educator, but with little formal training in cultural awareness.

The interaction: The interaction, in English, was initiated by Sr D. She determined the timing and location to fit in with her work program and with the patient's dialysis schedule. The nurse did most of the talking and the patient asked few questions.

Communication goals: Sr D aimed to provide education through feedback and discussion of routine monthly test results. She aimed to integrate information about dialysis, medication and diet, specifically related to the test results.

Neither participant mentioned what Ms C might have wanted to communicate.

The participants' assessment: Both believed that the communication had, to some extent, been effective. Ms C said, "I could see it all clearly. . . . I didn't have any misunderstanding."

However, through analysis of the video with each participant and with further discussion, evidence of extensive miscommunication emerged. The nurse had emphasised, during the interaction, that Ms C's haemoglobin level was low and had discussed its significance in terms of her health and the use of erythropoietin. In the exit interview, Ms C indicated that she believed that all her results were normal.

Sr D had discussed results of biochemical tests and the use of specific medications. She said, "[The patient] knows a lot about medication and dialysis treatment. . . . She knows what medication she's on."

However, at exit interview, it became clear that Ms C had not understood key issues relating to the results, that she was unable to name most of her medications, and that her understanding of their actions was completely different from the biomedical explanations she was given. The absence of shared understanding of key concepts relating to results and medications was seen as an important source of miscommunication.

Consequences for clinical management: Both participants had perceived the communication to be effective. The discrepancy between perception and reality became evident only through triangulation of the data. Standard assessments of quality of care by the measurement of staff and patient satisfaction, in this case relating to education and staff–patient interaction, would not have revealed the miscommunication. This has important implications for clinical management. Best outcomes in the management of end-stage renal disease require adherence to a complex treatment regimen of regular dialysis, repeated tests, dietary restriction and daily medications.

Lack of shared knowledge and understanding

Extensive prerequisite knowledge is essential for making sense of information about the management of end-stage renal disease. A shared understanding of kidney and heart function, and of the nature of the circulatory system (including, for example, the components and function of blood), is necessary for meaningful discussion about medication, fluid restriction and dialysis. As shared understanding of many of these concepts does not exist, effective communication is seldom achieved.

Cultural and linguistic distance

The vast cultural and linguistic distance between staff and patients in these interactions impeded communication. Staff use of culturally specific terminology was one difficulty. For instance, quantification was a constant problem. Key biomedical issues were expressed quantitatively, including percentage of renal function, number of drinks consumed, amount and frequency of medications, length of visits home, length of time without dialysis, high and low blood pressure, and blood test results. But litres, kilograms, hours, dates and percentages have little, if any, meaning for most Yolngu, while Yolngu ways of expressing quantity and spatial and temporal concepts were completely unknown to staff.

Lack of staff training in cross-cultural communication

None of the staff speak an Aboriginal language and none of their Yolngu patients speak English as a first language. Furthermore, none of the staff had received any formal training in intercultural communication. Even general cultural awareness training, which is increasingly available to staff, had been utilised to a limited extent and to minimal effect. One physician recalled his only training experience in cultural awareness:

> In Alice Springs, I probably had a day's training. It would have been a standard thing, and it was brief, and I have no memory of it.

And yet he found that intercultural communication was

> . . . an incredibly difficult aspect of working there. I knew that there was next to no communication between me and the patients, which had an obvious impact on what happened.

There were organisational barriers to formal training, as a renal nurse related:

> I haven't done a cross-cultural course at all. When I first came up [to Darwin], it wasn't compulsory and I've tried to get in several times over the years and it was either booked out or Renal couldn't relieve me because they didn't have enough staff at the time.

For most of the staff, learning occurred "on the job", but this had serious limitations, as a physician reflected:

> You become aware of the issues just through doing what you're doing. Which is poor. . . . You learn by obstacles and by . . . causing affront and problems.

Limited use of interpreters

Until recently, there was no alternative to attempting whatever communication was possible through the assistance of whoever was available. In the absence of professional interpreters, family members had to suffice — a seriously inadequate practice.[20] Although an Aboriginal Interpreter Service providing Yolngu language speakers now exists, changes in practice are occurring only slowly. In the interactions observed in our study, the closest any of the staff or patients came to seeking the assistance of a professional interpreter was to call on the assistance of a family member who had some informal interpreting experience.

4: Strategies for improving communication between non-Aboriginal healthcare staff and Aboriginal patients

- Train staff in intercultural communication. It is the staff's responsibility to make this accommodation to enable Aboriginal people to make informed choices in the context of their own language and cultural environment.
- Train Aboriginal interpreters to prepare them for work with healthcare workers.
- Promote strategies to monitor the effectiveness of communication and to repair miscommunication.
- Develop educational resources to facilitate a shared understanding of (a) physiological processes and treatment options; and (b) cultural, social and economic realities confronting Aboriginal patients and their families.

DISCUSSION

Our study demonstrates that renal staff and Yolngu patients rarely achieved a shared understanding of key concepts. Consequently, communication was seriously limited and quality of care compromised. There was little indication that either staff or patients had, before or during these encounters, considered the potential for miscommunication. Even if this had occurred, staff had no tools or guidelines for assessing its extent. Our findings suggest that any substantial improvement in communication, and in ensuing health outcomes, requires fundamental change in the delivery of healthcare — in particular, in constructing a shared understanding, from the perspectives of both staff and patients, of physiological processes, renal disease and treatment options.

Previous research has been based on interviews with service providers, and sometimes with Aboriginal patients, about their perception of communication issues. Our study, by contrast, involved direct observation of interactions, and then, with the input of all participants, sequential analysis. We have shown that miscommunication can easily go unrecognised.

While previous studies of communication breakdown have usually focused on the clinical interaction, we looked beyond this. Our findings enabled us to understand both sides and to see the clinical interaction within the social, cultural and political context relevant to the delivery of healthcare to Aboriginal people.

We believe the qualitative research methods we used were appropriate. It could be argued that our findings may not be generalisable to staff–patient communication in the entire renal unit in which the research occurred, nor transferable to other patient-care settings. However, we believe that the methods of triangulation, respondent validation and maximum variation sampling strengthen the validity of our findings.

Videotaping the interactions did not appear to fundamentally alter the communication strategies used by staff. In any case, we would expect any bias, arising through participants' knowledge of being observed, to be towards *more* effective rather than *less* effective communication. Our results support similar findings of miscommunication in other Aboriginal health research[8,11,21] and in international cross-cultural research.[1,3] We believe that our research findings are both credible and relevant to the delivery of healthcare to Aboriginal people, and that similar miscommunication problems are likely to exist in other healthcare settings in which there are people whose first language is not English.

Fundamental change is required to achieve effective communication with Aboriginal patients who have renal disease. We will not be able to deliver optimal care without striking a balance between the staff's medical priorities and the patients' social needs. Some strategies for improving communication are set out in Box 4. Planning and implementing such strategies for the Yolngu will require collaboration between staff, patients and patients' families. We are currently developing such a project. Short of such radical change, attempts to improve communication can meet with only limited success.

COMPETING INTERESTS

None declared.

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Single-dose azithromycin versus seven days of amoxycillin in the treatment of acute otitis media in Aboriginal children (AATAAC): a double blind, randomised controlled trial

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ABSTRACT

Objective: To compare the clinical effectiveness of single-dose azithromycin treatment with 7 days of amoxycillin treatment among Aboriginal children with acute otitis media (AOM) in rural and remote communities in the Northern Territory.

Design, setting and participants: Aboriginal children aged 6 months to 6 years living in 16 rural and remote communities were screened for AOM. Those diagnosed with AOM were randomly allocated to receive either azithromycin (30 mg/kg as a single dose) or amoxycillin (50mg/kg/day in two divided doses for a minimum of 7 days). We used a double-dummy method to ensure blinding. Our study was conducted from 24 March 2003 to 20 July 2005.

Main outcome measures: Failure to cure AOM by the end of therapy; nasal carriage of *Streptococcus pneumoniae* and non-capsular *Haemophilus influenzae* (NCHi).

Results: We followed 306 of 320 children (96%) allocated to the treatment groups. Single-dose azithromycin did not reduce (or increase) the risk of clinical failure (50% failure rate [82/165]) compared with amoxycillin (54% failure rate [83/155]) (risk difference [RD], –4% [95% CI, –15% to 7%]; $P = 0.504$). Compared with amoxycillin, azithromycin significantly reduced the proportion of children with nasal carriage of *S. pneumoniae* (27% v 63%; RD, –36% [95% CI, –47% to –26%]; $P < 0.001$) and NCHi (55% v 85%; RD, –30% [95% CI, –40% to –21%]; $P < 0.001$). Nasal carriage of *S. pneumoniae* with intermediate or full resistance to penicillin was lower (but not significantly so) in the azithromycin group (10% v 16%), but this group had significantly increased carriage of azithromycin-resistant *S. pneumoniae* (10% v 3%; RD, 7% [95% CI, 0.1% to 12%]; $P = 0.001$). Carriage of β-lactamase-producing NCHi was about 5% in both groups.

Conclusion: Although azithromycin reduced nasal carriage of *S. pneumoniae* and NCHi, clinical failure was high in both treatment groups. The possibility of weekly azithromycin treatment in children with persistent AOM should be evaluated.

Trial registration: Australian Clinical Trials Registry ACTRN 12609000691246.

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Australian Aboriginal children are prone to frequent and severe episodes of acute otitis media (AOM). In a 2003 survey of over 600 children living in 29 Aboriginal communities throughout the Northern Territory, 20% had tympanic membrane perforation, despite 87% uptake of pneumococcal conjugate vaccine.[1] Such high rates of AOM with perforation (AOMwiP) and chronic suppurative otitis media (CSOM) are seldom reported in the medical literature. A contributing factor to disease progression may be failed detection of AOM due to the absence of acute symptoms (pain, irritability or fever) in the presence of a bulging tympanic membrane.[2] Thus, a diagnosis of AOM without perforation (AOMwoP) is recommended if a bulging tympanic membrane is present, whether symptoms are present or not.[3] There are few studies assessing antibiotic treatment in such high-risk populations.

We have found that, compared with placebo, long-term antibiotic treatment resolves middle ear effusion and reduces perforation.[4] However, these regimens are difficult to deliver effectively,[5] particularly to poor families in remote settings. Previous studies have shown that short-course azithromycin (for 3–5 days),[6-9] weekly single-dose azithromycin prophylaxis for 12 weeks[10] or a single dose of azithromycin[11,12] are effective for treating AOM and are less likely than amoxycillin clavulanate to be associated with gastrointestinal side effects.[8]

The primary aim of our study was to determine the clinical effectiveness of a single dose of azithromycin compared with a standard 7-day treatment course of amoxycillin[13] in a population at high risk of tympanic membrane perforation and CSOM.

Abbreviations

AOM	Acute otitis media
AOMwiP	Acute otitis media with (tympanic membrane) perforation
AOMwoP	Acute otitis media without (tympanic membrane) perforation
CSOM	Chronic suppurative otitis media
MIC	Minimum inhibitory concentration
NCHi	Non-capsular *Haemophilus influenzae*
RD	Risk difference

METHODS

Study design

We conducted a participant-blinded, care provider-blinded, assessor-blinded, randomised controlled trial — the Azithromycin versus Amoxycillin for Treatment of Acute Otitis Media in Aboriginal Children (AATAAC) study — comparing single-dose azithromycin with 7 days of twice-daily amoxycillin treatment. Treatment allocation was stratified by health centre, body weight and diagnosis (AOMwoP or AOMwiP), using randomly varying block sizes.

Participants

Our study commenced on 24 March 2003 and was completed on 20 July 2005. We screened Aboriginal children from 14 remote communities and two Aboriginal medical services in northern and central Australia. Our inclusion criteria were (a) age between 6 months and 6 years; (b) new diagnosis of AOMwoP or AOMwiP; and (c) willingness of parents to bring their child for a follow-up visit. Children were excluded if they had (a) previously been allocated to an intervention group in the same study; (b) received antibiotics in the previous 7 days;

(c) current severe illness requiring intravenous or intramuscular antibiotic treatment to be given within the following 7 days; (d) a known allergy to penicillin or azithromycin; or (e) perforation covering more than 2% of the tympanic membrane.

Interventions

Children with AOM were randomly allocated to receive either azithromycin (30 mg/kg as a single dose) and amoxycillin placebo, or amoxycillin (50 mg/kg/day in two divided doses for a minimum of 7 days) and azithromycin placebo. All children were scheduled to be examined on Day 0 and at the end of therapy (between Day 6 and Day 11). Children diagnosed with AOMwiP at the end of therapy received a second course of treatment and were also examined between Day 12 and Day 21.

Outcomes

Primary outcomes

In our clinical trial protocol, we prespecified two primary outcomes:

- *Clinical failure:* the proportion of children with persistent ear pain, a bulging tympanic membrane or middle ear discharge at the end of therapy (children lost to follow-up were assumed not to have improved); and
- *Failure to improve:* the proportion of examined children with no improvement in clinical signs at the end of therapy. Improvement in clinical signs was determined by healing of a tympanic membrane perforation or substantial reduction in tympanic membrane bulging. Improvement could occur despite clinical failure at end of therapy.

Secondary outcomes

We also compared the azithromycin and amoxycillin groups with regard to the following secondary outcomes:

- *Clinical outcomes:* the proportion of children with clinical failure or failure to improve when seen before Day 11 after commencement of therapy (per-protocol analysis); clinical failure according to baseline diagnosis (AOMwoP or AOMwiP); clinical failure according to age (< 2 years or ⩾ 2 years); clinical failure according to nasal carriage of otitis media pathogens (*Streptococcus pneumoniae* or non-capsular *Haemophilus influenzae* [NCHi]) at the end of therapy; and additional clinical observations (runny nose and skin sores). We also recorded new episodes of AOMwiP or pain.
- *Microbiological outcomes:* nasal carriage of *S. pneumoniae*, NCHi, resistant *S. pneumoniae* or β-lactamase-positive NCHi; and the proportion of ear discharge cultures positive for *S. pneumoniae* or NCHi.

Clinical assessment

All clinical assessments were made by ear health research officers. Otoscopic findings were recorded on a standardised form. Assessments were made using a GSI 38 tympanometer (Grason-Stadler, Eden Prairie, Minn, USA), a LumiView portable binocular microscope (Welch Allyn, Skaneateles Falls, NY, USA), a Siegle speculum for pneumatic otoscopy, and a videootoscope (Welch Allyn, Skaneateles Falls, NY, USA). Video recordings of the tympanic membranes were reviewed by an independent unblinded observer.

We categorised middle ear states into one of six diagnostic categories, using criteria based on recommendations for clinical practice in this population:[3]

- Normal;
- Otitis media with effusion (intact and non-bulging tympanic membrane and type B tympanogram);
- AOMwoP (any bulging of the tympanic membrane and type B tympanogram);
- AOMwiP (middle ear discharge observed and perforation recently healed or present for less than 6 weeks or covering less than 2% of the pars tensa of the tympanic membrane [CSOM is nearly always associated with larger perforations]);
- Dry perforation (tympanic membrane perforation without any discharge observed); or
- CSOM (middle ear discharge observed and perforation present for longer than 6 weeks and covering at least 2% of the pars tensa of the tympanic membrane).

The final middle ear diagnosis reflected the child's more severely affected ear (highest category).

The presence of nasal discharge was recorded if nasal discharge was visible from 1 metre away at any time during the clinical examination. Skin sores (raised crust or pus visible) were recorded if visible on the arms, legs or head.

Microbiology

Nasal swabs were taken at baseline and at follow-up examinations. Swabs of ear discharge were also collected on the days when nasal swabs were taken. All swabs were collected, transported, stored and cultured as previously described.[14] Azithromycin resistance was defined as a minimum inhibitory concentration (MIC) of ⩾ 2 μg/mL. High-level resistance was defined as MIC > 32 μg/mL.

Sample size

We estimated that in at least 50% of children receiving standard therapy for AOM the condition would fail to resolve within 8 days. With 150 children in each intervention arm, the study would have a power of 95% to detect an improved outcome in an additional 20% of children ($\alpha = 0.05$). This difference was chosen as the minimum improvement that would potentially result in a change in recommended therapy for AOM from amoxycillin to azithromycin.

All statistical analyses were conducted using Stata software, version 10 (StataCorp, College Station, Tex, USA). Assessment of statistical significance was based on the Fisher exact test.

Ethics approval and registration

Our study was approved by the human research ethics committees of the Menzies School of Health Research and the Royal Darwin Hospital and by the Central Australian Human Research Ethics Committee, and was registered with the Australian Clinical Trials Registry (ACTRN 12609000691246). An independent Data and Safety Monitoring Board chaired by Professor David Brewster and Associate Professor Alan Ruben of the NT Clinical School, Flinders University, reviewed the data.

RESULTS

Clinical observations, nasal carriage and ear discharge at baseline (Box 1)

Clinical observations. Of 320 children taking part in the trial, 165 were randomly allocated to the azithromycin treatment group and 155 to the amoxycillin treatment group. As seven children were lost to follow-up in each group, analysis was carried out on the remaining 306 children (Box 2). Most were less than 2 years of age, and about half were male. For each group at baseline, 85% had AOMwoP and 15% had AOMwiP, and 5% of mothers reported that they thought their child had ear pain. Nasal discharge was seen in 69% of children and skin sores in 6%–7%.

Nasal carriage. For both treatment groups, nasal carriage of *S. pneumoniae* and NCHi was 83% or more. About 14% of children were colonised by *S. pneumoniae* with intermediate or full resistance to penicillin (MIC ⩾ 0.12 μg/mL), and about 5% carried azithromycin-resistant *S. pneumoniae* (MIC ⩾ 2 μg/mL). Less than 10% of children swabbed carried β-lactamase-producing NCHi.

1 Baseline characteristics of children participating in our study

Characteristic	Azithromycin group (*n* = 165)	Amoxycillin group (*n* = 155)
Mean age in months (SD)	18 (11)	17 (12)
Age less than 2 years	125/165 (76%)	125 (81%)
Male	80/165 (48%)	89 (57%)
Clinical features		
Ear pain	9/165 (5%)	8 (5%)
AOMwoP	140/165 (85%)	131 (85%)
AOMwiP	25/165 (15%)	24 (15%)
Nasal discharge	114/165 (69%)	107 (69%)
Skin sores	11/165 (7%)	10 (6%)
Nasal carriage of otitis media pathogens*		
Streptococcus pneumoniae	136/164 (83%)	131/152 (86%)
NCHi	140/164 (85%)	129/152 (85%)
Any *S. pneumoniae* or NCHi	155/164 (95%)	144/152 (95%)
Resistant *S. pneumoniae*		
Penicillin (MIC ⩾ 0.12 μg/mL)	25/164 (15%)	20/152 (13%)
Azithromycin (MIC ⩾ 2 μg/mL)	8/164 (5%)	6/152 (4%)
Any resistant *S. pneumoniae*	31/164 (19%)	22/152 (14%)
β-lactamase-producing NCHi	15/164 (9%)	9/152 (6%)
Any resistant *S. pneumoniae* or NCHi	42/164 (26%)	29/152 (19%)
Culture of ear discharge from children with AOMwiP†		
S. pneumoniae	8/36 (22%)	11/34 (32%)
NCHi	13/36 (36%)	14/34 (41%)

AOM = acute otitis media. AOMwiP = AOM with (tympanic membrane) perforation. AOMwoP = AOM without (tympanic membrane) perforation. MIC = minimum inhibitory concentration. NCHi = non-capsular *Haemophilus influenzae*. * Figures are proportion (%) of children swabbed. † Figures are proportion (%) of ears swabbed. ◆

Ear discharge. In the azithromycin and amoxycillin groups, respectively, *S. pneumoniae* was cultured from 22% and 32% of ear discharge swabs, and NCHi from 36% and 41% of ear discharge swabs.

Clinical outcomes, nasal carriage and ear discharge cultures at the end of therapy

Clinical failure (Box 3). At the end of therapy, 50% of the azithromycin group and 54% of the amoxycillin group were clinical failures. Similar proportions (45% and 49%) failed to improve, and about 5% were worse in both groups (nine children developed new perforations). Differences between the azithromycin and amoxycillin groups in clinical failure rates were not significant.

No differences in clinical failure or failure to improve were indicated in a per-protocol analysis (children seen before Day 11 after commencement of treatment). Only four parents reported that their child had ear pain (three of the children were in the amoxycillin group). Fewer children in the azithromycin group than the amoxycillin group had runny nose (35% v 46%), but the difference was not significant, and about 4% of children in both groups had skin sores.

Clinical failure rates in children who had AOMwoP at baseline were 40% in the azithromycin group and 46% in the amoxycillin group. For children who had AOMwiP at baseline, clinical failure rates were 92% and 83%, respectively. (Clinical failure rates were 84% and 63%, respectively, at Day 12–21.)

There was no significant association between age group (< 2 years or ⩾ 2 years) and clinical failure.

No significant difference in clinical failure was detected between azithromycin and amoxycillin groups for carriers or non-carriers of *S. pneumoniae* or NCHi at the end of therapy. Carriage of resistant *S. pneumoniae* or NCHi at baseline did not predict greater clinical failure. The risk difference (RD) in clinical failure between children carrying susceptible organisms (*S. pneumoniae* or NCHi) at baseline and those carrying resistant organisms at baseline was −9%.

Complications and side effects. There were three serious adverse events that resulted in admission to hospital during the treatment period. The independent Data and Safety Monitoring Board found that these were not associated with the study interventions. No side effects (eg, allergic, gastrointestinal) led to cessation of treatment or withdrawal from the study.

New episodes of disease. In both groups, new perforations were detected in 3%–4% of children who had AOMwoP at baseline. Pain resolved in all children who had had pain at baseline, but four children developed "new" pain (Box 4).

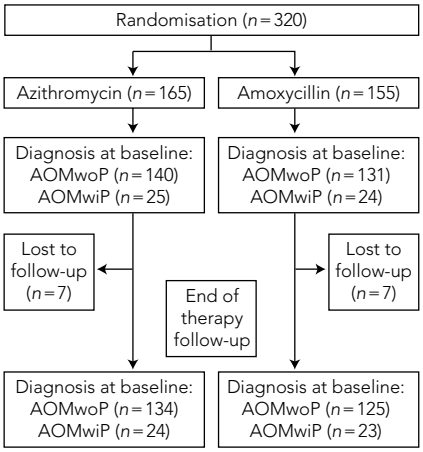
Nasal carriage. Compared with children in the amoxycillin group, children in the azithromycin group had significantly lower carriage of *S. pneumoniae* and *H. influenzae*, lower carriage of *S. pneumoniae* with intermediate or full resistance to penicillin (RD, −6% [but this difference was non-significant]), and significantly higher carriage of azithromycin-resistant *S. pneumoniae* (RD, 7%) (Box 5).

Ear discharge. The proportion of ear discharge cultures that were positive for *S. pneumoniae* and NCHi was about 25 percentage points lower in the azithromycin group (Box 5).

Comparison between baseline and end of therapy (Box 1 and Box 5)

Compared with baseline, nasal carriage of *S. pneumoniae* was significantly lower in both groups at follow-up (azithromycin group RD, −56% [95% CI, −65% to −47%]), and carriage of NCHi was also significantly reduced (azithromycin group RD, −31% [95% CI, −40% to −21%]). In the azithromycin group, compared with baseline, car-

2 Flowchart of children enrolled in the study



riage of *S. pneumoniae* with intermediate or full resistance to penicillin was lower at follow-up (10%) than at baseline (15%) (RD, −5% [95% CI, −12% to 2%]). In contrast, nasal carriage of azithromycin-resistant *S. pneumoniae* was higher at follow-up (10%) than at baseline (5%) (RD, 5% [95% CI, −1% to 10%]).

DISCUSSION

To our knowledge, this is the first randomised controlled trial of antibiotic treatment of AOM in a population with high rates of acute and chronic tympanic membrane perforation. Although our study was not designed or powered to show equivalence, our findings suggest that the two antibiotic regimens are likely to have similar efficacy in this population. However, in view of the substantial problems of adherence to twice-daily regimens in such populations, single-dose azithromycin treatment may be a more appropriate standard treatment option than a 7-day course of amoxycillin.

Azithromycin treatment reduced nasal carriage of both *S. pneumoniae* and NCHi. Although overall rates of antibiotic resistance were unchanged, azithromycin treatment was associated with an increase in carriage of azithromycin-resistant *S. pneumoniae*.

In other published trials, an evidence summary and a meta-analysis that included azithromycin treatment of AOM, clinical response rates in groups receiving azithromycin have generally been 70% or higher.[6-8,11,12,15-18] In a Cochrane review of short-course antibiotic treatment for AOM, outcomes of short courses of agents such as ceftriaxone or azithromycin were comparable with outcomes of longer courses of other antibiotics.[8] Clinical trials assessing outcomes of single-dose azithromycin treatment[11,12] have reported similarly high success rates. The Cochrane meta-analysis summary odds ratio for primary outcomes after 3–5 days' treatment with azithromycin ($n = 1347$) compared with 10 days' treatment with another antibiotic ($n = 1246$) was 1.09 (95% CI, 0.86 to 1.38). Success rates were about 86% in both azithromycin groups and comparison groups.[8] In a single trial that reported outcome data for perforated or non-perforated eardrums of 28 evaluable patients with spontaneous perforation, there were more failures after 5 days of therapy than after 10 days of therapy.[19]

In our study, clinical success rates among Aboriginal children receiving azithromycin therapy for AOM were much lower than the rates reported in previous published stud-

3 Clinical outcomes at the end of therapy (between Day 6 and Day 11)

	Azithromycin group ($n = 165$)	Amoxycillin group ($n = 155$)	RD (95% CI)	*P*
Primary outcomes (intention-to-treat analysis)				
Clinical failure	82/165 (50%)	83/155 (54%)	−4% (−15% to 7%)	0.504
Failure to improve	71/158 (45%)	72/148 (49%)	−4% (−15% to 7%)	0.567
No change	63/158 (40%)	63/148 (43%)		
Worse	8/158 (5%)	9/148 (6%)		
Primary outcomes (per-protocol analysis*)				
Clinical failure	66/140 (47%)	72/135 (53%)	−6% (−18% to 6%)	0.335
Failure to improve	62/140 (44%)	68/135 (50%)	−6% (−18% to 6%)	0.335
Other clinical outcomes				
Pain	1/156 (1%)	3/147 (2%)	−1% (−4% to 1%)	0.358
Runny nose	55/158 (35%)	67/146 (46%)	−11% (−22% to 0.1%)	0.06
Skin sores	7/158 (4%)	4/146 (3%)	2% (−3% to 6%)	0.545
Clinical failure according to baseline diagnosis				
AOMwoP at baseline	53/134 (40%)	57/125 (46%)	−6% (−18% to 6%)	0.379
AOMwiP at baseline	22/24 (92%)	19/23 (83%)	9% (−10% to 28%)	0.416
Clinical failure according to age				
< 2 years of age	61/125 (49%)	68/125 (54%)	−6% (−18% to 7%)	0.448
⩾ 2 years of age	21/40 (53%)	15/30 (50%)	3% (−21% to 26%)	1.000
Clinical failure according to nasal carriage of otitis media pathogens at the end of therapy				
Positive for *Streptococcus pneumoniae*	21/42 (50%)	52/92 (57%)	−7% (−25% to 12)	0.575
Negative for *S. pneumoniae*	53/115 (46%)	23/53 (43%)	3% (−13 to 19%)	0.868
Positive for NCHi	46/86 (53%)	69/124 (56%)	−2% (−16% to 12%)	0.779
Negative or NCHi	28/71 (39%)	6/21 (29%)	11% (−12% to 33%)	0.446
Nasal carriage of otitis media pathogens at baseline (both treatment groups combined)	Susceptible pathogens at baseline†	Resistant pathogens at baseline‡		
S. pneumoniae	124/255 (49%)	27/51 (53%)	−4% (−19% to 11%)	0.646
S. pneumoniae or NCHi	112/237 (47%)	39/69 (57%)	−9% (−23% to 4%)	0.218

AOM = acute otitis media. AOMwiP = AOM with (tympanic membrane) perforation. AOMwoP = AOM without (tympanic membrane) perforation. NCHi = non-capsular *Haemophilus influenzae*. RD = risk difference. * Assessed before Day 11 after commencement of therapy. † Clinical failure rates in children carrying susceptible pathogens at baseline. ‡ Clinical failure rates in children carrying resistant pathogens at baseline. ◆

4 Persistent and new episodes of acute otitis media (AOM) at the end of therapy (between Day 6 and Day 11)

	Azithromycin group ($n = 165$)	Amoxycillin group ($n = 155$)	RD (95% CI)	*P*
AOMwoP if AOMwoP at baseline	49/140 (35%)	52/131 (40%)	−5% (−16% to 7%)	0.452
AOMwiP if AOMwiP at baseline	14/25 (56%)	11/24 (46%)	10% (−18% to 38%)	0.572
AOMwiP if no AOMwiP at baseline	4/140 (3%)	5/131 (4%)	−1% (−5% to 3%)	0.743
Pain if pain at baseline	0/9	0/8		
Pain if no pain at baseline	1/156 (1%)	3/147 (2%)	−1% (−4% to 1%)	0.358

AOMwiP = AOM with (tympanic membrane) perforation. AOMwoP = AOM without (tympanic membrane) perforation. RD = risk difference. ◆

5 Microbiological outcomes at the end of therapy (between Day 6 and Day 11)

	Azithromycin group (*n* = 157)	Amoxycillin group (*n* = 146)	RD (95% CI)	*P*
Nasal carriage of pathogens*				
Streptococcus pneumoniae	42/157 (27%)	92/146 (63%)	−36% (−47% to −26%)	< 0.001
NCHi	86/157 (55%)	124/146 (85%)	−30% (−40% to −21%)	< 0.001
S. pneumoniae and NCHi	26/157 (17%)	82/146 (56%)	−40% (−50% to −30%)	< 0.001
Any *S. pneumoniae* or NCHi	102/157 (65%)	134/146 (92%)	−27% (−36% to −18%)	< 0.001
Resistant *S. pneumoniae**				
Penicillin (MIC ⩾ 0.12 μg/ml)	16/157 (10%)	23/146 (16%)	−6% (−13% to 2%)	0.171
Azithromycin (MIC ⩾ 2 μg/mL)	15/157 (10%)	5/146 (3%)	7% (0.1% to 12%)	0.001
Azithromycin (MIC ⩾ 32 μg/mL)	9/157 (6%)	3/146 (2%)	4% (−1% to 8%)	0.140
Any resistant *S. pneumoniae*	25/157 (16%)	26/146 (18%)	−2% (−10% to 7%)	0.749
β-lactamase-producing NCHi	8/157 (5%)	7/146 (5%)	0.3% (−4% to 5%)	1.0
Any resistant *S. pneumoniae* or NCHi	33/157 (21%)	32/146 (22%)	−1% (−10% to 8%)	0.889
Culture of ear discharge from children with AOMwiP†				
S. pneumoniae	0/27	6/24 (25%)	−25% (−40% to −3%)	0.040
NCHi	6/27 (22%)	12/24 (50%)	−28% (−53% to −2%)	0.046

AOMwiP = acute otitis media with (tympanic membrane) perforation. NCHi = non-capsular *Haemophilus influenzae*. RD = risk difference. * Figures are proportion (%) of children swabbed. † Figures are proportion (%) of ears swabbed. ◆

ies. Reduced likelihood of cure is reported for patients with azithromycin-resistant *S. pneumoniae* (67%) compared with those with susceptible *S. pneumoniae* (90%).[17] The rates of antimicrobial resistance found in our study (about 21%) do not explain the high proportion of clinical failures observed.

Poor adherence to recommended treatment regimens could explain poor clinical outcomes in the amoxycillin group. However, as the single dose of azithromycin was given under supervision, compliance with azithromycin treatment was close to 100%. In another of our studies in which compliance with twice-daily amoxycillin therapy was monitored, we estimated that 17/30 participants took less than half their recommended treatment.[20] In that study, we found that AOM persisted in about three-quarters of participants and could not be explained by nasopharyngeal carriage of penicillin-resistant *S. pneumoniae* strains.[20]

The poor clinical outcomes associated with failure to eradicate NCHi in our study are consistent with clinical and bacteriological studies by Dagan et al showing clinical failure rates of 65%[18] and 53%[21] in patients with NCHi infections receiving azithromycin treatment.

We believe that persistent ear disease is related to high bacterial load in the nasopharynx.[22] Antibiotics that eradicate otitis media pathogens and reduce bacterial load are needed for clinical success. While azithromycin was more effective at eradicating AOM pathogens than amoxycillin, nasal carriage of any *S. pneumoniae* or NCHi remained high at the end of therapy (65% in the azithromycin group and 92% in the amoxycillin group). For children with AOMwiP, ear discharge cultures were less likely to be positive after treatment with azithromycin. The observed association of better clinical outcomes with pathogen clearance and the greater impact of azithromycin on carriage (particularly for NCHi) led us to believe that clinical trials of longer courses of azithromycin treatment are required. The potential benefits for other important child health problems (eg, skin sores, runny nose and trachoma) should also be evaluated.

In the AATAAC study, most children with AOM at enrolment had an intact bulging tympanic membrane. However, as few had ear pain, it is likely that many children in this population will have undiagnosed AOM. This may explain the high rate of tympanic membrane perforation in this population. Further research is needed to determine the benefits of early detection of at-risk children as well as predictors of which children will progress to perforation.

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COMPETING INTERESTS

None identified.

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Characteristics of the community-level diet of Aboriginal people in remote northern Australia

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Abstract

Objective: To describe the nutritional quality of community-level diets in remote northern Australian communities.

Design, setting and participants: A multisite 12-month assessment (July 2010 to June 2011) of community-level diet in three remote Aboriginal communities in the Northern Territory, linking data from food outlets and food services to the Australian Food and Nutrient Database.

Main outcome measures: Contribution of food groups to total food expenditure; macronutrient contribution to energy and nutrient density relative to requirements; and food sources of key nutrients.

Results: One-quarter (24.8%; SD, 1.4%) of total food expenditure was on non-alcoholic beverages; 15.6% (SD, 1.2%) was on sugar-sweetened drinks. 2.2% (SD, 0.2%) was spent on fruit and 5.4% (SD, 0.4%) on vegetables. Sugars contributed 25.7%–34.3% of dietary energy, 71% of which was table sugar and sugar-sweetened beverages. Dietary protein contributed 12.5%–14.1% of energy, lower than the recommended 15%–25% optimum. Furthermore, white bread was a major source of energy and most nutrients in all three communities.

Conclusion: Very poor dietary quality continues to be a characteristic of remote Aboriginal community nutrition profiles since the earliest studies almost three decades ago. Significant proportions of key nutrients are provided from poor-quality nutrient-fortified processed foods. Further evidence regarding the impact of the cost of food on food purchasing in this context is urgently needed and should include cost–benefit analysis of improved dietary intake on health outcomes.

Dietary improvement for Indigenous Australians is a priority strategy for reducing the health gap between Indigenous and non-Indigenous Australians.[1] Poor-quality diet among the Indigenous population is a significant risk factor for three of the major causes of premature death — cardiovascular disease, cancer and type 2 diabetes.[2] The 26% of Indigenous Australians living in remote areas experience 40% of the health gap of Indigenous Australians overall.[3] Much of this burden of disease is due to extremely poor nutrition throughout life.[4]

Comprehensive dietary data for Indigenous Australians are not available from national nutrition surveys or any other source. Previous reports on purchased food in remote Aboriginal communities are either dated,[5] limited to the primary store[5,6] and/or short-term or cross-sectional in design.[7,8] These studies have consistently reported low intake of fruit and vegetables, high intake of refined cereals and sugars, excessive sodium intake, and limited availability of several key micronutrients.

The aim of this study was to examine characteristics of the community-level diet in remote communities in the Northern Territory over a 12-month period.

Methods

We examined purchased food in three remote communities in relation to:

- food expenditure;
- estimated per capita intake;
- nutrient profile (macronutrient contribution to energy) and nutrient density (nutrient per 1000 kJ) relative to requirements; and
- major nutrient sources.

We collected information on community size, remoteness and availability of food in each community as well as community dietary data including all available foods with the exception of traditional foods and foods sourced externally to the community. Alcohol was prohibited in the three study communities at the time of our study.

Monthly electronic food (and non-alcoholic beverage) transaction data were provided by the community-owned store and independent stores in the three communities for July 2010 to June 2011. Food order data were collected from food suppliers for all food services in each of the three communities. All food and beverage items with their accompanying universal product code or store-derived product code, quantity sold, and dollar value (retail price) were imported to a purpose-designed Microsoft Access database[9] and linked to the Food Standards Australia New Zealand Australian Food and Nutrient survey specific (AUSNUT 1999 and AUSNUT 2007[10]) and reference (NUTTAB 06) databases (NUTTAB 06 has now been replaced by NUTTAB 2010). Folate dietary equivalent levels per 100 g were modified for bread and flour to equal NUTTAB 2010 levels since mandatory fortification was introduced. Unit weights were derived for all food and drink items and multiplied by the quantity sold to give a total item weight. Food items were categorised into food groups derived from the Australian Food and Nutrient Database AUSNUT 07 food grouping system[10] and beverages were further categorised to provide a greater level of detail (Appendix 1; all appendices are available online at mja.com.au). Several nutrient compositions for items not available in these databases were derived from the product's nutrition information panel, which is mandatory on all packaged foods in Australia, or from standard recipes. Nutrient availability was derived for 21 nutrients. Energy and nutrient content per 100 g edible portion was multiplied by the edible weight (primarily sourced from Australian Food and Nutrient data[10]) of each of the food and beverage items (adjusted for specific gravity to convert mL to g weight) to derive total energy and nutrient content for each food group.

Completeness of data and accuracy were ensured by: a check on monthly time periods reported, follow-up with providers where a food description or unit weight was not available or where a discrepancy was noted; checking of unit weights against unit dollar value;

1 Community characteristics

Community	Population, and age and/or sex distribution* 2006	2010	Estimated population†	Distance from food wholesaler; location‡	Access	Food stores	Food services
A	1697 (49% male; 703 residents <18 yrs)	2124 (50% male)	2286	> 500 km; island in Top End region	Regular daily flight	Community-owned store. Two independent stores	Aged care meals, child care, school canteen, school lunch program, breakfast program
B	250 (49% male; 94 residents <18 yrs)	210 (49% male)	202	> 400 km; central desert region	Sealed and unsealed road	Community-owned store	Aged care meals, school lunch program, child care
C	217 (43% male; 73 residents <18 yrs)	201 (49% male)	163	< 150 km; central desert region	Sealed and unsealed road	Community-owned store	Aged care meals, child care, school lunch program, breakfast program

*Based on Australian Bureau of Statistics (ABS) census data.[11,15] †2644 was derived for the total study population based on the total energy available in the purchased food supply and the weighted per capita energy requirement based on the total population age and sex distribution. This population size was used for analyses where data for all communities were combined rather than the total of 2651. ‡ All three communities are classified by the ABS Australian Standard Geographical Classification (http://www.health.gov.au/internet/otd/publishing.nsf/Content/locator) as RA5 (very remote). ◆

and a second person checking the matching of foods with nutrient composition data and assigning of food groups.

Data analysis

Data were grouped by community, food source, month and food group and transferred to Stata 10 (StataCorp) for analysis. Data for all food sources were combined (community food supply) and the average monthly and per capita daily weight and dollar value of each food group were calculated. Mean monthly and daily food weights were assumed to approximate mean monthly and daily dietary intakes for the data period.

The populations of each of the three remote communities and the three communities combined were estimated based on the total amount of energy provided through the community-level diet, and, assuming energy balance, were divided by the estimated weighted per capita energy requirement for each of the communities and the three communities combined. The estimated total population was verified against Australian Bureau of Statistics (ABS) estimates.[11] The weighted per capita energy requirement was determined for each community using the estimated energy requirement for each age group and sex, as stated in the Nutrient Reference Values for Australia and New Zealand[12] (with a physical activity factor of 1.6 [National Health and Medical Research Council — light activity[13]]) in conjunction with the population age and sex distribution as determined by the 2006 ABS population census for each of these three communities.

Nutrient density was calculated for each nutrient by dividing the total nutrient weight by the energy value of the community food supply. Population-weighted nutrient density requirements were derived using estimated average requirements (EARs).[12] The EAR for nutrients is stated as a daily average and varies by age and sex. EARs are estimated to meet the requirements of half the healthy individuals of a particular age group and sex and are used to assess the prevalence of inadequate intakes at a population level.[12] A nutrient density level below the weighted EAR per 1000 kJ was considered insufficient in meeting the population's requirements.

Adequate intake (AI) values were used for nutrients for which no EAR was available (potassium, dietary fibre and vitamin E α-tocopherol equivalents). The midpoint of the AI range for sodium was used. Macronutrient profiles (the proportions of dietary energy from protein, total fat, saturated fat, carbohydrate and total sugar) were compared with acceptable macronutrient distribution ranges.[14] Major food sources were defined as foods contributing 10% or more of a specific nutrient.

Ethics approval was provided by the Human Research Ethics Committee of Menzies School of Health Research and the Northern Territory Department of Health and the Central Australian Human Research Ethics Committee. Written informed consent was gained from all participating communities, food businesses and food services.

Results

The estimated total population was 2644. Community populations ranged in estimated size from 163 to 2286 residents of mostly Aboriginal ethnicity and were comparable with regard to age and sex distributions.[15] The distance from each community to the nearest food wholesaler ranged from 130 km to 520 km. Variation between the communities in remoteness, size, and number of food outlets is shown in Box 1.

Expenditure patterns

Average per capita monthly spending on food and non-alcoholic beverages in communities A, B and C, respectively, was $394 (SD, $31), $418 (SD, $82) and $379 (SD, $80). About one-quarter of all money spent on food and beverages was on beverages (combined communities, 24.8%; SD, 1.4%), with soft drinks contributing 11.6%–16.1% to sales across the three communities (combined communities, 15.6%; SD 1.2%) (Appendix 2). This compares to less than 10% in total spent on fruit and vegetables in each of the three communities (7.3%, 9.1% and 8.9%; combined communities, 2.2% [SD, 0.2%] on fruit and 5.4% [SD, 0.4%] on vegetables) (Appendix 2).

Per capita daily intake

Based on population estimates, there appeared to be differences in the daily per capita volume of many food groups between community A compared with

2 Estimated energy availability and macronutrient profile, overall and by community

Energy intake	Community A	Community B	Community C	All communities	
Estimated per capita energy intake based on 2010 census population (kJ)	9845	9119	7623	9608	
Estimated per capita energy intake, based on estimated energy requirement* (kJ [SD])	9147 (927)	9480 (1644)	9400 (1740)	9212 (856)	
Macronutrient distribution as a proportion of dietary energy (% [SD])					**Recommended range[14]**
Protein	12.5% (0.3)	14.1% (0.8)	13.4% (0.6)	12.7% (0.3)	15%–25%
Fat	24.5% (0.6)	31.6% (1.5)	33.5% (1.1)	25.7% (0.6)	20%–35%
Saturated fat	9.4% (0.3)	11.6% (0.6)	12.1% (0.3)	9.7% (0.3)	< 10%
Carbohydrate	62.1% (0.8)	53.3% (1.8)	52.1% (1.1)	60.7% (0.8)	45%–65%
Sugars	34.3% (0.8)	28.9% (2.2)	25.7% (1.8)	33.4% (0.7)	< 10%†

* Estimated energy requirements were calculated by age group (1–3 years; 4–8 years; 9–13 years; 14–18 years; 19–30 years; 31–50 years; 51–70 years; > 70 years) and sex based on Nutrient Reference Values for Australia and New Zealand, tables 1–3.[11] For age 19 to > 70 years, the midpoint height and weight of each adult age group was used. For < 18 years, the midpoint of the estimated energy requirement range across each age and sex category was used. Energy expenditure was estimated at 1.6 basal metabolic rate overall. We estimated 8% of women aged 14–50 years were pregnant and 8% were breastfeeding, based on Australian Bureau of Statistics 2006 births data, table 9.2[16] and 2006 census data for women aged 13–54 years.[15] † Recommendation for "free sugars" — all monosaccharides and disaccharides added to foods by the manufacturer, cook or consumer, plus sugars naturally present in honey, syrups and fruit juices.[17] ◆

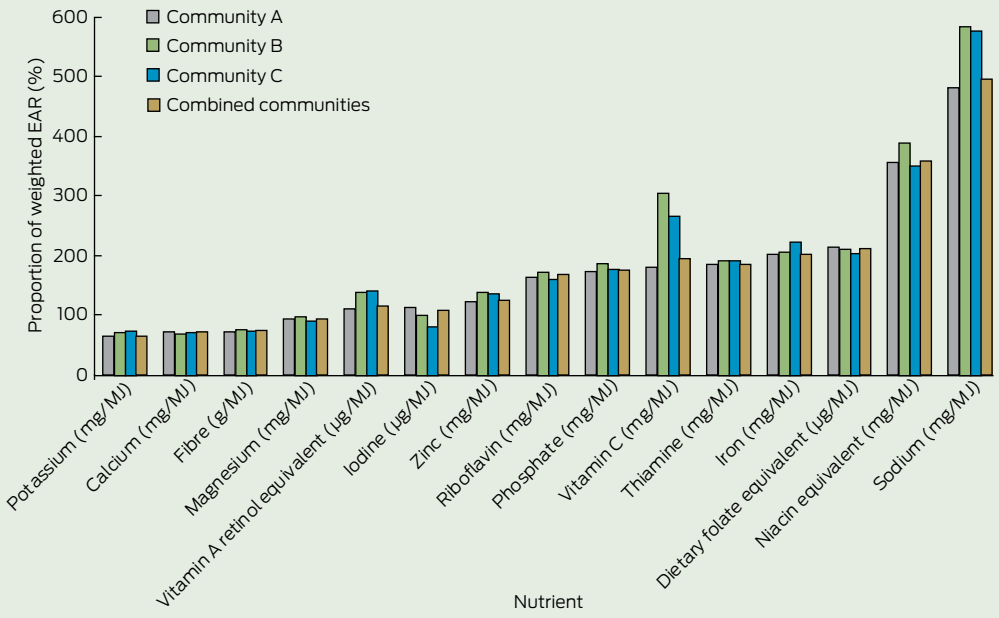
communities B and C and less notable differences between communities B and C (Appendix 3).

On average, per capita daily intake of beverages (including purchased water and liquid tea) was 1464 g (SD, 130.5 g) with sugar-sweetened soft drinks comprising 298–497 g across communities (Appendix 3). Liquid tea constituted most of the remaining beverage volume. Daily per capita fruit and vegetable intake in community A (122 g) was just over half that of communities B (222 g) and C (247 g) (Appendix 3).

Macronutrient profile

For community A, the proportion of dietary energy as carbohydrate was at the higher end of the recommended range; for communities B and C it was within the recommended range. Sugars contributed 25.7%–34.3% of the total proportion of dietary energy across the three communities (Box 2), 71% of which was table sugar and sugar-sweetened beverages. The proportion of dietary energy from fat was within the acceptable range for each community, and lower in community A compared with communities B and C. The proportion of dietary energy as saturated fat was within the recommended range for community A and higher than recommended for communities B and C. The proportion of dietary energy as protein was lower than the recommended minimum in all three communities (Box 2).

3 Nutrient per 1000 kJ as a percentage of weighted estimated average requirement (EAR) per 1000 kJ,* overall and by community



* Adequate intake values were used for nutrients for which no EAR was available (potassium, dietary fibre, vitamin E α-tocopherol equivalents, sodium). ◆

Micronutrient density

With reference to weighted EARs (or AIs) per 1000 kJ and nutrients measured, in all three communities the diet was insufficient in calcium, magnesium, potassium and fibre (Box 3). Iron, vitamin C and folate equivalents were all around double the weighted EAR per 1000 kJ and niacin equivalents were nearly four times the EAR (Box 3). Sodium was the nutrient provided in the greatest excess, at nearly six times the midpoint of the average intake range (Box 3). Most nutrient density values appeared lower in community A compared with communities B and C (Appendix 4).

Major nutrient sources

In all three communities, white bread fortified with fibre and a range of micronutrients was a major source of protein, fibre, iron, sodium, calcium, dietary folate, potassium, magnesium and B-group vitamins (Appendix 5). Sugar and sugar-sweetened beverages provided 65%–72% of total sugars (Appendix 5). Bread, salt and baking powder were major sources of sodium in all three communities. Major food sources of all nutrients were similar across the three communities (Appendix 5).

Discussion

Our comprehensive assessment of the community diet averaged over a 12-month period showed a high intake of refined cereals and added sugars, low levels of fruit, vegetables and protein, limiting key micronutrients, and excessive sodium intake. Our findings confirm recent and past reports of dietary quality in remote Aboriginal communities.[5,8] We report food expenditure and dietary patterns that are similar to those reported previously using store sales data alone,[5,6,8] as are the limiting nutrients (protein, potassium, magnesium, calcium and fibre).[8]

A striking finding from our study is the high expenditure on beverages and corresponding high intake of sugar-sweetened beverages coupled with low expenditure (and low intakes) of fruit and vegetables.

The level of sugar-sweetened soft drinks reported for communities B and C is in line with what we have previously reported for 10 NT communities from store data alone.[6] The apparently substantially higher per capita volume reported for community A warrants further investigation, which could include examining variation in regional consumption, food delivery systems and food outlets. Similarly high per capita consumption of sugar-sweetened beverages has been reported among Aboriginal and Torres Strait Islander children in regional New South Wales (boys, 457 g/day; girls, 431 g/day) and for children at the national level (364.7 g/day).[18,19] The high volume of tea purchased is also of concern, as tea is generally consumed as a sugar-sweetened beverage.

The low daily fruit and vegetable intake reported for the three study communities (which on average equated to 0.3 to 0.7 serves of fruit and 1.1 to 2.1 serves of vegetables) is in range with the reported average of 0.4 serves of fruit and 0.9 serves of vegetables per person per day sold through 10 NT community stores in 2009,[6] but lower than intakes self-reported among other Aboriginal populations in remote Queensland and regional NSW.[18,20,21] Our estimates do suggest improved intakes compared with the low levels of fruit and vegetable intake reported nearly three decades earlier for six remote NT communities.[5] Caution needs to be applied in making comparisons with past studies owing to use of different methodologies. It has been estimated that increasing fruit and vegetable consumption to up to 600 g per day could reduce the global burden of ischaemic heart disease and stroke by 31% and 19%, respectively.[22] The benefits for the Indigenous population are likely to be much greater, considering their currently low intake of fruit and vegetables and high burden of disease.

A further disturbing aspect of the diet is that fibre-modified and fortified white bread is providing a large proportion of key nutrients, including protein, folate, iron, calcium and magnesium, and unacceptably high levels of sodium. Similarly, among Aboriginal and Torres Strait Islander children in regional NSW, bread was also reported to be a major dietary source of energy, salt and fibre.[18] It is alarming that white bread is providing a large percentage of dietary protein when it is a poor protein source. Considering the high-quality protein foods traditionally consumed by Aboriginal Australians,[23] this apparent shift to a low-protein and high-carbohydrate diet needs investigation. Traditional foods, such as fish and other seafood, eggs and meat provide high-quality protein, but are unlikely to be significant at the population level if not accessed frequently and by a substantial proportion of the population.

The extremely high rates of preventable chronic disease experienced among Aboriginal people in remote Australia and the high intake of sugar-sweetened beverages, unacceptably low levels of fruit and vegetables, and limiting essential nutrients, provide a compelling rationale that more needs to be done to improve diet and nutrition. Poverty is a key driver of food choice[24-26] and although most Indigenous people living in remote communities are in the low income bracket, a standard basket of food costs, on average, 45% more in remote NT communities than in the NT capital.[27] People in the study communities spend more on food ($379 to $418 per person per month) compared with the expenditure estimated for other Australians ($314 per person per month with 2.6 persons per household).[28] Our study provides the only available estimate of remote community food and drink expenditure that we know of. Household expenditure data are not available for very remote Australia, representing a gap in information on food affordability, a major determinant of health.

Our study highlighted some important differences in dietary quality between the study communities, with the dietary profile for community A being generally poorer. This may be indicative of intercommunity or regional differences, such as community size, number of food outlets, location and remoteness, access to food outlets, level of subsistence procurement and use of traditional foods, climate, housing or water quality, and warrants broader investigation.

As with individual-level dietary assessment, there are limitations in estimating community-level dietary intake. An inherent issue in community-level per capita measures in research is the difficulty of determining the population for the study period, so caution is required in using the values presented here; however, the total population (2644) was verified against ABS predicted estimates for the 2011 Australian remote Indigenous population (2638) and was within 4% of the later released ABS census data collected in 2010 for the three study communities (2535). Further, monthly per capita dietary intake estimations were averaged over a 12-month period and are likely to take into account the fluctuations in population that occur in remote communities seasonally and over time. A strength of our study is that expenditure patterns based on proportional spending, macronutrient profile and nutrient density provide an assessment of dietary quality that are entirely independent of population size estimates. Furthermore, as dietary data are derived from food sales records rather than self-reported data, they provide an objective assessment of diet quality. Limitations in using food sales data as a measure of dietary intake have been reported previously.[8] Estimated per capita energy intakes for communities A and B differed by less than 10% from per capita requirements derived from 2010 ABS census population figures, indicating completeness in food sale data. Estimated energy intakes for community C were lower than required but 81% of per capita requirements.

Reports on dietary quality are also limited by the accuracy of food composition databases. For example, the range of nutrients presented for each food in the Australian food composition database

varies depending on the analytical data available. Nutrient levels reported in this study are based on currently available nutrient composition data.[29]

A limitation in assessing the nutritional quality of the community-level diet using purchased food data is the exclusion of traditional food intake. It is assumed that traditional food contributes minimally to community-level dietary intake, as not all families have access to traditional foods and procurement usually does not occur on a regular basis. However, the contribution of traditional food to dietary intake has not been investigated. We recognise it would be important in future studies to quantify the contribution of traditional foods to total food intake. The low expenditure on (and therefore low intake of) high-quality protein foods suggests that either these foods are not affordable, or that possibly these foods are accessed through subsistence procurement. However, mean daily energy intake estimates based on 2010 census data indicate that the great majority of energy required is provided through the imported food supply.

Despite these limitations, this study provides an objective, contemporary and comprehensive assessment of the community-level diet in three remote Indigenous communities without the inherent limitations of individual-level dietary intake assessment. It provides evidence on key areas of concern for dietary improvement in remote Aboriginal communities.

Very poor dietary quality has continued to be a characteristic of community nutrition profiles in remote Indigenous communities in Australia for at least three decades. Significant proportions of a number of key micronutrients are provided as fortification in a diet derived predominantly from otherwise poor-quality, highly processed foods. Ongoing monitoring (through use of food sales data) of community-level diet is needed to better inform community and wider level policy and strategy development and implementation. Low income is undoubtedly a key driver of diet quality. Further evidence regarding the impact of the cost of food on food purchasing in this context is urgently needed and the long-term cost benefit of dietary improvement needs to be considered.

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Rheumatic heart disease in Timor-Leste school students: an echocardiography-based prevalence study

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The known Rheumatic heart disease (RHD) is a disease of poverty that is highly prevalent in resource-limited settings and among Indigenous Australians and New Zealand Māori. The prevalence of RHD in Timor-Leste has not been described.

The new The prevalence of echocardiography-detected RHD in Timor-Leste is comparable with the highest rates in the world. All cases of RHD had previously been undiagnosed.

The implications A public health response for preventing and managing RHD in Timor-Leste is urgently needed, including improving the recognition of acute rheumatic fever. Further investigation is required to determine the best approaches for managing RHD detected by echocardiography.

Abstract

Objectives: To determine the prevalence of rheumatic heart disease (RHD) in school-aged children and young people in Timor-Leste.

Design: Prospective cross-sectional survey. Echocardiography was performed by Australian cardiologists to determine the presence of RHD. Demographic data were also collected. Patients in whom RHD was detected were entered into a register to allow monitoring of adherence to secondary prophylaxis; the first dose of benzathine penicillin G (BPG) was administered on the day of screening.

Setting: Schools in urban (Dili) and rural (Ermera) Timor-Leste.

Participants: School students aged 5–20 years.

Outcome measures: Definite and borderline RHD, as defined by World Heart Federation echocardiographic criteria.

Results: 1365 participants were screened; their median age was 11 years (IQR, 9–14 years), and 53% were girls. The estimated prevalence of definite RHD was 18.3 cases per 1000 population (95% CI, 12.3–27.0 per 1000), and of definite or borderline RHD 35.2 per 1000 (95% CI, 26.5–46.4 per 1000). Definite (adjusted odds ratio [aOR], 3.5; 95% CI, 1.3–9.4) and definite or borderline RHD (aOR, 2.7; 95% CI, 1.4–5.2) were more prevalent among girls than boys. Eleven children (0.8%) had congenital heart disease. Of the 25 children in whom definite RHD was identified, 21 (84%) received education and a first dose of BPG on the day of screening; all 25 have since received education about primary care for RHD and have commenced penicillin prophylaxis.

Conclusions: The rates of RHD in Timor-Leste are among the highest in the world, and prevalence is higher among girls than boys. Community engagement is essential for ensuring follow-up and the effective delivery of secondary prophylaxis.

Rheumatic heart disease (RHD) is a preventable condition that is now extremely rare in high income countries, although its prevalence in indigenous, migrant and refugee populations remains high.[1-4] The prevalence of RHD is also high in low and middle income countries in sub-Saharan Africa, South America, Asia, and the Pacific, where high rates of *Streptococcus pyogenes* (group A streptococcus [GAS]) transmission have been documented.[5,6] Group A streptococcal infections are associated with poverty, and cause a spectrum of disease ranging from skin infections and pharyngitis to invasive disease, including bacteraemia.[5] Acute rheumatic fever (ARF) is a multisystem immune-mediated condition that can follow infection with GAS; GAS pharyngitis is associated with ARF, whereas a link between GAS pyoderma and ARF has been suggested but not confirmed.[7] In RHD, chronic valvular damage results from carditis associated with repeated episodes of ARF. ARF recurrences and the associated progression of valvular disease in established RHD can be effectively prevented by 4-weekly injections of long-acting benzathine penicillin G (BPG).[8,9]

After 24 years of occupation, the people of Timor-Leste voted for independence from Indonesia in 1999; following 3 years of transitional administration by the United Nations, it achieved independence in 2002. During and immediately following the referendum and the subsequent withdrawal of the Indonesian army, 70% of the national infrastructure was destroyed, including 35% of health facilities; the vast majority of clinicians and health managers fled the country.[10] Timor-Leste has been reconstructing its health system ever since. In a setting of limited resources such as Timor-Leste, the importance of research for identifying needs and driving improvements in health service delivery is clear.[11]

ARF is not notifiable in Timor-Leste, and its incidence is unknown. The limited capacity of the country for diagnostic microbiology means that the incidence of GAS infections is also unknown, although skin infections (including impetigo) are common.[12] Anecdotal reports from clinicians in Timor-Leste suggest that the number of patients presenting with ARF to heath care facilities is small, but that the burden of RHD among children and young people is significant. Many patients present to local health services with end-stage heart disease, but access to surgery is limited. Some East Timorese can undergo cardiac surgery in other countries, funded by charitable organisations such as the East Timor Hearts Fund (ETHF) and Rotary Oceania Medical Aid for Children (ROMAC), but many succumb to their disease while still young.[13]

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1 World Heart Federation criteria for echocardiographic diagnosis of rheumatic heart disease (RHD) in people under 21 years of age (2012)[15]

Definite rheumatic heart disease
At least one of:

- Pathological mitral regurgitation and at least two morphological features of RHD of the mitral valve
- Mitral stenosis mean gradient ≥ 4 mmHg*
- Pathological aortic regurgitation and at least two morphological features of RHD of the aortic valve†
- Borderline disease of both the aortic and mitral valves‡

Borderline rheumatic heart disease
At least one of:

- At least two morphological features of RHD of the mitral valve without pathological mitral regurgitation or mitral stenosis
- Pathological mitral regurgitation
- Pathological aortic regurgitation

Normal echocardiographic findings
All of:

- Mitral regurgitation that does not meet all four Doppler echocardiographic criteria (physiological mitral regurgitation)
- Aortic regurgitation that does not meet all four Doppler echocardiographic criteria (physiological aortic regurgitation)
- An isolated morphological feature of RHD of the mitral valve (for example, valvular thickening) without any associated pathological stenosis or regurgitation
- Morphological feature of RHD of the aortic valve (for example, valvular thickening) without any associated pathological stenosis or regurgitation

* Congenital mitral valve anomalies must be excluded. † Bicuspid aortic valve, dilated aortic root, and hypertension must be excluded. ‡ Combined aortic and mitral regurgitation in high prevalence regions and in the absence of congenital heart disease is regarded as rheumatic. ◆

No published studies have described the burden of RHD in Timor-Leste. We therefore conducted an echocardiography screening study with the aims of describing the prevalence of RHD in school-aged children and young people in Timor-Leste, and of establishing an RHD register for coordinating secondary prophylaxis for East Timorese people with RHD.

Methods

Schools were recruited in the capital city of Dili and the inland district of Ermera to obtain a sample population representative of the different geographic regions and a spectrum of socio-economic status in Timor-Leste. A consultation visit to the schools was made 4 months prior to the commencement of screening. A plain language information sheet in Tetum was distributed to parents and families, giving them the opportunity to decline screening of their children. An opt-out approach to consent was adopted because it was strongly preferred by local school principals and community members; this method has been applied successfully in other studies.[14]

All students aged 5–20 years who attended school on the screening days were eligible to participate in the study. Demographic data for all students were collected on a standard form to reduce measurement bias; to maximise accuracy, only study staff fluent in the local language recorded the data. Date of birth, age, number of people in their household, and number of rooms in their home were based on student self-report. All participants were examined for evidence of impetigo or scabies; these results will be reported in a separate article. As the capacity for diagnostic microbiology in Timor-Leste is limited, samples for microbiological testing were not collected.

An echocardiogram was performed by one of five cardiologists (including two paediatric specialists) with a Vivid I or Q machine (GE Healthcare). All children had full screening echocardiograms, including parasternal long axis, parasternal short axis, apical four-chamber, and apical five-chamber views (2D and Doppler). RHD was classified as borderline or definite according to World Heart Federation criteria (Box 1).[15] Abnormal echocardiograms were flagged during the screening process and immediately reviewed by a paediatric cardiologist, as well as by all five cardiologists at the end of the screening week; a diagnosis of borderline or definite RHD was confirmed only after consensus was reached by at least three of the cardiologists involved in the study. Normal echocardiograms were not reviewed.

Statistical analysis

Data were entered into an Access 2016 database (Microsoft), and statistical analysis was conducted in Stata 13 (StataCorp). Descriptive statistical analysis was undertaken and the prevalence of RHD estimated with 95% confidence intervals (CIs). Continuous variables that were not normally distributed were compared in Mann–Whitney rank sum tests; differences in binary variables were assessed in χ^2 tests (univariate analysis). Relative risks (RRs) and 95% CIs were calculated. For multivariate analyses, logistic regression was employed to estimate adjusted odds ratios (aORs) with 95% CIs. Sex, age, and location of screening were included in the logistic regression model because these variables could plausibly influence the prevalence of RHD. $P < 0.05$ was deemed statistically significant.

Patients with RHD were entered into a register to facilitate monitoring of adherence to subsequent secondary prophylaxis, with the first dose of BPG administered on the day of the study. Patients with heart disease amenable to surgery were referred to ROMAC for possible surgery in Australia.

Ethics approval

Ethics approval for the study was obtained from the Human Research Ethics Committee of the Northern Territory Department of Health and Menzies School of Health Research (reference, 2016-2546) and the Instituto Nacional de Saúde in Timor-Leste (reference, MS-INS/DF/DP/V/2016/220). Permission to undertake screening was granted by the Ministério da Educação in Timor-Leste and by the principals of the schools involved.

2 Demographic data for people under 21 years of age screened for rheumatic heart disease in Dili or Ermera, Timor-Leste

	Dili	Ermera	Total
Total number	505	860	1365
Sex			
Girls	255 (50.5%)	470 (54.7%)	725 (53.1%)
Boys	250 (49.5%)	390 (45.3%)	640 (46.9%)
Age (years)			
5–9	233 (46.1%)	180 (20.9%)	413 (30.3%)
10–14	269 (53.3%)	351 (40.8%)	620 (45.4%)
15–20	3 (0.6%)	329 (38.3%)	332 (24.3%)
People per household, median (interquartile range)	6 (4–8)	8 (6–9)	7 (6–9)

Results

A total of 1365 participants aged 5–20 years were screened by echocardiography. Their median age was 11 years (interquartile range [IQR], 9–14 years) and 725 (53%) were girls; the sex distribution was similar for all age groups (data not shown). Five hundred and five children (37%) were screened in Dili, 860 (63%) in the district of Ermera. All students knew their age, but fewer than half were able to report their date of birth. None knew whether they were allergic to penicillin. The median number of residents per household was 7 (IQR, 6–9) (Box 2).

A total of 25 definite and 23 borderline cases of RHD were detected by echocardiography. The estimated prevalence of definite RHD was 18.3 cases per 1000 population (95% CI, 12.3–27.0 per 1000); that of definite or borderline RHD was 35.2 cases per 1000 population (95% CI, 26.5–46.4 per 1000). None of the children in whom RHD was detected had previously been diagnosed with RHD; one had a history of ARF without evidence of carditis and of poor adherence to penicillin prophylaxis. All cases were classified as either mild or moderate, none as severe (Box 3). All borderline and definite cases included mild or moderate mitral regurgitation. No mitral stenosis was detected. Three children also had mild or moderate aortic regurgitation. Most of the children in whom definite or borderline RHD was detected lived in Ermera, where the estimated prevalence was 43.0 cases per 1000 population (95% CI, 31.2–58.9) (Box 3).

3 Estimated prevalence of rheumatic heart disease in people under 21 years of age in Dili or Ermera, Timor-Leste

	Dili		Ermera		Total	
	Cases	Prevalence, per 1000 (95% CI)	Cases	Prevalence, per 1000 (95% CI)	Cases	Prevalence, per 1000 (95% CI)
People with rheumatic heart disease						
Definite	7	13.9 (0.6–28.9)	18	20.9 (13.1–33.1)	25	18.3 (12.3–27.0)
Borderline	4	7.9 (0.2–21.0)	19	22.1 (14.0–34.5)	23	16.8 (11.1–25.3)
Definite or borderline	11	21.8 (11.7–39.0)	37	43.0 (31.2–58.9)	48	35.2 (26.5–46.4)
Severity of definite or borderline rheumatic heart disease						
Mild	10		32		42	
Moderate	1		5		6	
Severe	0		0		0	

CI = confidence interval. ◆

Eleven children (0.8%) had congenital heart disease. Two were referred for surgery (one case of severe aortic stenosis, one of Ebstein's anomaly); nine children had relatively minor cardiac anomalies, including three with haemodynamically insignificant atrial septal defects, two with mitral valve prolapse, two with patent ductus arteriosus, one with dextrocardia, and one with mild pulmonary stenosis.

There was a clear sex difference in prevalence of RHD. There were 27.6 cases of definite RHD per 1000 girls (95% CI, 17.7–42.5) and 7.8 cases per 1000 boys (95% CI, 2.8–18.7) (aOR, 3.6; 95% CI, 1.3–9.5; $P = 0.012$); there were 49.7 cases of definite or borderline RHD per 1000 girls (95% CI, 35.9–68.2) and 18.8 cases per 1000 boys (95% CI, 10.4–32.9) (aOR, 2.7; 95% CI, 1.4–5.2; $P = 0.004$).

Students screened in Ermera appeared more likely to have definite or borderline RHD in the univariate analysis (RR, 2.0; 95% CI, 1.0–3.8; $P = 0.040$), but the multivariate analysis (adjusted for age and sex) detected no significant association between RHD and age or location (Box 4).

Twenty-one children with definite RHD (84%) received BPG and education on the day of screening, and all children were entered into the RHD register for follow-up. Children whose parents were not available on the day of screening did not initially receive BPG, but all children and families have subsequently received education about RHD and the importance of secondary prophylaxis, and have commenced treatment with BPG.

Discussion

Our echocardiography-based screening study identified a very high prevalence of RHD among young people in Timor-Leste, comparable with the highest documented rates of RHD elsewhere (Box 5).[1,6,16-24] The prevalence of definite RHD in our study (18 cases per 1000 population) is higher than the rate for Fiji (7 cases per 1000), and similar to that in other regional neighbours, including the Top End of Australia (15 cases per 1000 for Indigenous Australians at high risk).[1,18] The prevalence in Timor-Leste may be higher than estimated; we did not detect any cases of mitral stenosis or other forms of severe RHD, and it is possible that students with severe RHD were too unwell to attend school, and therefore not screened. It is also possible that children and young people from poorer families, at greater risk of RHD, do not regularly attend school. The absence of severe RHD in our study contrasts with anecdotal reports of fatal cases managed by Timor-Leste health services, and with the experience of visiting cardiology services,[13] but the morbidity and mortality of severe RHD in Timor-Leste has not been formally documented.

Further investigation of the morbidity and mortality of RHD in Timor-Leste are needed, and, as RHD had not previously been diagnosed in any of the young people we screened, active case detection and close follow-up is warranted. The World Health Organization has recently renewed its resolution to tackle RHD as an important international health problem requiring a multisectoral

4 Univariate and multivariate analysis of prevalence of rheumatic heart disease in people under 21 years of age in Dili and Ermera, Timor-Leste

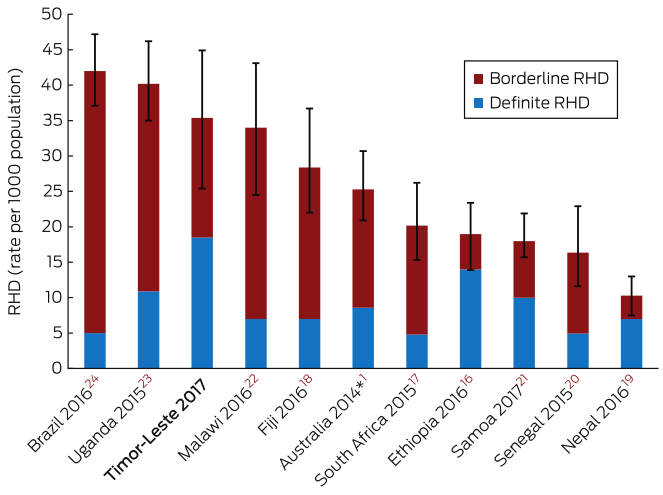
	Definite rheumatic heart disease					Definite and borderline rheumatic heart disease				
	Cases	Relative risk (95% CI) univariate	*P*	Adjusted odds ratio* (95% CI) multivariate	*P*	Cases	Relative risk (95% CI) univariate	*P*	Adjusted odds ratio* (95% CI) multivariate	*P*
Sex			0.007		0.012			0.002		0.004
Boys	5 (0.8%)	1		1		12 (1.9%)	1		1	
Girls	20 (2.8%)	3.5 (1.3–9.4)		3.6 (1.3–9.5)		36 (5.0%)	2.6 (1.4–5.0)		2.7 (1.4–5.2)	
Age (years)										
5–9	5 (1%)			1		9 (2%)			1	
10–14	15 (2.4%)			1.9 (0.7–5.3)	0.22	24 (3.9%)			1.7 (0.8–3.7)	0.20
15–20	5 (2%)			1.0 (0.3–3.6)	0.97	15 (4.5%)			1.6 (0.7–3.9)	0.31
Location								0.040		
Dili	7 (1%)	1	0.35	1	0.34	11 (2.2%)	1		1	0.13
Ermera	18 (2.1%)	1.5 (0.6–3.6)		1.6 (0.6–4.0)		37 (4.3%)	2.0 (1.0–3.8)		1.8 (0.8.–3.7)	

CI = confidence interval. * Adjusted for other two factors in table. ◆

response. The WHO recognises that RHD affects a substantial proportion of the world's most vulnerable populations, and that antibiotic treatment of group A streptococcal infections and secondary prophylaxis for preventing ARF are cost-effective strategies that should be implemented in high burden settings.[25]

Efforts to reduce the prevalence of RHD in Timor-Leste should focus on primordial (reducing risk factors for GAS infection), primary (treating GAS infections to prevent ARF), and secondary prevention (penicillin prophylaxis for people with a history of ARF or established RHD). Access to cardiac surgery (tertiary prevention) is limited to cases amenable to international referral, usually to Australia, Indonesia or Singapore, as cardiac surgery is not currently performed in Timor-Leste.

5 Prevalence in recent studies of definite and borderline rheumatic heart disease in young people detected by echocardiography according to World Heart Federation criteria



* Indigenous Australian population at high risk of rheumatic heart disease. All studies reported prevalence for people under 21 years of age, except the South African study (24 years or younger). ◆

Household sizes in this cohort were large, consistent with census data from Timor-Leste, and domestic crowding is a known risk factor for RHD.[26] Access to health care services is limited, particularly in regional areas. Ermera, like much of Timor-Leste, is a mountainous area with very poor roads in some parts. Delivering BPG regularly is challenging, but has been successfully undertaken since our screening study. Our study has shown that it is possible to screen for and diagnose RHD, immediately enter patients into a register, provide education, and commence BPG prophylaxis on the same day. However, we relied on volunteer cardiologists with specialist skills in the echocardiographic diagnosis of RHD, and it would be difficult to reproduce this approach to screening on a large scale.

Registries for the surveillance and follow-up of patients with established RHD have been shown to be effective in reducing morbidity and mortality, especially when incorporated into national disease surveillance networks.[25] Increasing the available information on disease burden by active surveillance could provide the impetus for providing more resources to the Timorese health care system, leading to long term improvement of local cardiac services, as well as to improving the chances of those with established RHD of undergoing surgery overseas. Most importantly, an effective register facilitates the delivery of penicillin prophylaxis that can halt the progression of RHD and prevent the sequelae of heart failure and death.[8,9]

We found that that risk of RHD in Timor-Leste is greater for girls. This is similar to findings in Australia and other parts of the world,[1,27] although a significant sex difference was not detected by a recent meta-analysis.[6] The cause of the sex difference is yet to be determined. The risk of morbidity and mortality during pregnancy and childbirth is very high in women with RHD, and increases with successive pregnancies.[5] Catholicism is common in Timor-Leste, a country with a median household size of 5.7 people;[28] access to contraception is limited, and societal opposition to its use significant.[29] The prevalence of RHD among pregnant women in Timor-Leste is unknown, but multiple pregnancies in a setting of high prevalence RHD may place women and babies at increased risk of adverse outcomes and death.

There were several limitations to our study. Screening was only conducted in two districts of Timor-Leste. The prevalence of RHD

in other districts may be different, but we expect that enrolling subjects from both an urban and a rural district resulted in a sufficiently representative sample for producing meaningful results. We enrolled only children and young people attending school, and those from poorer families or too sick to go to school will not have been included in our sample. The children screened in Ermera were older than those screened in Dili, but neither age nor location were significant factors in the multivariate analysis.

The cardiologists performing the echocardiograms provided a diagnosis during screening, and there was no external review of the images by a blinded cardiologist. However, a diagnosis was only provided after consensus among three of the five cardiologists was reached; it is very unlikely that the burden of RHD was overestimated. On the other hand, unremarkable echocardiograms were not reviewed, so it is possible that some cases were missed and the prevalence therefore underestimated. Some uncertainty regarding the clinical significance of borderline RHD remains, but recent studies have found that children with borderline RHD are more likely to have ARF, progression of their valvular lesions, and later development of definite RHD.[30] Nevertheless, echocardiography screening for RHD case detection remains controversial, as there is no evidence that screening leads to improved outcomes.

Conclusion

We found a significant burden of undetected RHD in our sample of young people, particularly among girls and young women. Our finding of a large burden of undetected disease indicates that active case detection is needed in Timor-Leste. The health system of Timor-Leste needs to be improved to increase the capacity of health workers to recognise and manage ARF and RHD. The nascent RHD register needs to be expanded, and further investigations in Timor-Leste and elsewhere are required to better define the role and practice of echocardiography screening, and to guide secondary prophylaxis for patients in whom RHD is detected by screening.

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Perspective

Emerging diabetes and metabolic conditions among Aboriginal and Torres Strait Islander young people

Intersectoral collaboration is needed to engage communities and design effective culturally and age-appropriate interventions

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The gap between the health of Aboriginal and Torres Strait Islander and non-Indigenous Australians is well documented, with many policies and programs currently working towards improving outcomes. Despite these efforts, life expectancy is 10–11 years less than that of non-Indigenous Australians,[1] and 65% of deaths occur before 65 years of age, compared with 19% in the non-Indigenous population.[1]

Cardiovascular and metabolic diseases are responsible for most of the gap in life expectancy and are associated with higher hospitalisation and mortality rates.[1] In 2013, hospitalisation rates for cardiovascular disease were 1.6–2.5 times higher in Indigenous people depending on age, and Indigenous adults are six times as likely to die from diabetes as non-Indigenous Australians.[1] Indigenous adolescents with type 2 diabetes are over ten times more likely to be hospitalised than non-Indigenous adolescents.[2] While we acknowledge that hospitalisations are a poor indicator of the true prevalence of diabetes-related complications within the community, they suggest that the burden of disease associated with a diagnosis of diabetes is greater in the Aboriginal and Torres Strait Islander population.[1]

Importantly, the age of onset of cardiometabolic conditions, such as diabetes, obesity and cardiovascular disease, is significantly younger in Indigenous than in non-Indigenous Australians, and the incidence of these conditions continues to increase in Indigenous youth.[3] With limited national data currently available, however, the prevalence of type 2 diabetes among Indigenous young people is not known. Studies from different states consistently report higher rates, from a younger age, of youth onset type 2 diabetes in the Indigenous Australian population.[3–5] While we have seen an increasing prevalence among all Australian youth over the past 20 years, Indigenous young people have experienced a much greater rise in new diagnosis rates. Western Australian data indicate a disturbing discrepancy, with a type 2 diabetes incidence of 12.6 per 100 000 person years in Indigenous youth aged 16 years or less, compared with an incidence of 0.6 in non-Indigenous youth — a striking 20-fold difference.[3] A 2007 New South Wales study suggested that type 2 diabetes was 6.1 times as common among Indigenous young people aged 0–19 years than among non-Indigenous young people,[4] and a 2013 Northern Territory study reported that Indigenous youth represented 88% of youth type 2 diabetes diagnoses.[5]

These statistics indicate the extent of the issue despite likely diagnostic underestimation, as many children with type 2 diabetes may be managed in primary care rather than by paediatric or diabetes services, or are not diagnosed until a later age.[5] The increasing incidence in the young Indigenous Australian population parallels the global context, where type 2 diabetes in youth has been described as an avoidable "disease of poverty" concentrated in populations of socio-economically disadvantaged young people.[6] Risk factors for diabetes and metabolic syndrome have been reported at a significantly earlier age and at much higher frequency among the Indigenous Australian population,[7,8] suggesting that the prevalence of diabetes will continue to rise exponentially in the future without intervention.

In Indigenous Australian young people with type 2 diabetes, there are also higher rates of comorbidities, with 59% also having hypertension, 24% having dyslipidaemia and 61% having obesity.[3] These comorbidities will have a significant impact on the future burden of disease, and may lead to renal, cardiac, neurological and ophthalmological complications. Canadian data demonstrated that 45% of patients with youth onset type 2 diabetes had reached end-stage renal failure, requiring renal replacement therapy, 20 years after diagnosis, compared with zero people with type 1 diabetes.[9] Youth onset type 2 diabetes was associated with a 23 times higher risk of kidney failure and 39 times higher risk of need for dialysis, compared with young people without diabetes.[9] This implies that many young people who are being diagnosed with diabetes now will be on dialysis by 30 years of age, with significant effects on Aboriginal and Torres Strait Islander families and communities.

Global reports also suggest that the pathophysiology, phenotype, treatment response and prognosis of youth onset diabetes differ significantly from later onset diabetes.[10] This contributes to greater concern regarding the future health of young people, as youth onset diabetes (defined as that diagnosed before the age of 25 years) appears to progress more rapidly, be more difficult to treat, have more comorbidities, and have a higher risk of complications. There is also a greater risk of mental health concerns, and the diagnosis of diabetes appears to worsen any pre-existing comorbidities,[11] which is of great concern in an already very vulnerable population. Type 2 diabetes is known to carry a burden of stigma, potentially further isolating young people,[6,10] and there has been a lack of research focusing on strengths or resilience factors that would enhance engagement and self-management.[6]

Experience internationally from other First Nations populations suggests a stark trajectory ahead for Australia, as emerging cardiometabolic disease develops among Indigenous Australian young people, with associated early morbidity and mortality. Among First Nations peoples of Canada, 43% of children born to mothers diagnosed with type 2 diabetes before 18 years of age developed diabetes by 10–19 years of age and 25% developed diabetes by 7 years of age.[11] Intergenerational and epigenetic factors have been suggested as important potential contributory factors in understanding increasing diabetes rates in Indigenous young people.[12,13] Changes in DNA methylation may be induced by the in utero environment, with lifelong metabolic risk possibly influenced by the timing of exposure, although limited longitudinal data are currently available.[14]

Work among the Pima Indian peoples of Arizona has explored intergenerational risk. A 3.7-fold higher risk of type 2 diabetes was reported in Pima Indian children exposed to intrauterine hyperglycaemia compared with siblings who were born before the mother developed diabetes, and these children also had a higher body mass index, suggesting that the intrauterine environment may be more important than underlying genetic risk.[13] The risk of diabetes at 10–14 years of age was 16.9 (95% CI, 4.1–70.8) times higher in overweight American Indian children than normal weight children, and children who already demonstrated impaired glucose tolerance at 5–9 years of age had a 188.2 (95% CI, 43.8–808.0) times greater risk of developing diabetes by 10–14 years of age, although the small numbers of incident type 2 diabetes cases should be noted.[15] It has been suggested that 47% of type 2 diabetes in youth may be subsequent to intrauterine exposure to maternal diabetes and obesity, with each generation developing cardiometabolic disease at a younger age than the preceding generation.[16] However, the limitations of such studies should be acknowledged, including the inability to measure general societal changes contributing to increased prevalence of cardiometabolic conditions in some populations.

Comprehensive strategies, action plans and both funding and better communication across sectors (health, education, infrastructure and local government) and departments are required to address obesity, diabetes and metabolic risk among Indigenous young people in Australia. It requires a radical rethinking of our current approach which is failing Aboriginal and Torres Strait Islander young people and communities, and a commitment to reconsider the paradigm, to be open to innovative approaches and the involvement of multiple sectors.

It is critical that we act now to prevent these emerging public health issues, with engagement of Indigenous communities in the design of interventions being crucial. A suite of interventions across the life course are required, targeting children and young people before they develop disease, particularly childhood obesity, as well as targeting their parents to prevent intergenerational transmission of metabolic risk. The in utero period and first 5 years of life are influential in terms of the long term risk of chronic disease, and we propose that identifying and improving childhood metabolic health be a targeted priority of health services. Key time points for intervention to prevent intergenerational metabolic disease are before conception (optimising general health of women of child bearing age), during pregnancy (optimising health and diagnosing and managing any chronic disease early), after pregnancy (optimising health and improving rates of breastfeeding to reduce risk of obesity and diabetes in children of women with diabetes), and in early childhood (encouraging healthy feeding practices and physical activity, and preventing childhood obesity).

Public health approaches and intersectoral collaboration are needed to address these issues, engage communities, and design effective and culturally and age appropriate interventions. Simple public health messages, such as breastfeeding initiation and continuation for more than 4 months, and smoking cessation and prevention of uptake in young people offer possible points of intervention to reduce later metabolic risk, with limited breastfeeding and early smoking both shown to increase the risk of young onset obesity and diabetes.[17,18] International studies demonstrate that interventions targeting high risk 7–10-year-old children and engaging families and school staff, as well as peers as mentors, in culturally appropriate ways can be effective in reducing risk factors for type 2 diabetes and metabolic disease.[18] In conjunction with targeted programs, the political commitment for broad system changes across sectors, with adequate resourcing, is required in order to address entrenched socio-economic inequities.

Collaboration between clinicians and researchers across Australia is required to establish the true prevalence and disease burden of type 2 diabetes among Indigenous young people. We need to hear from young people and families as to how diabetes and health are conceptualised amid the many competing priorities of life, aiming to improve models of care and educational strategies. Despite the high risk of cardiometabolic diseases within the Indigenous population, and our knowledge that this occurs from a young age, annual screening rates for type 2 diabetes vary from 0 to 43% across Australia, with particularly

low rates reported in younger adults.[19] Increased screening rates, especially in areas of established high risk, may contribute to earlier diagnosis and management of diabetes, and so improve long term outcomes. The "diabetes story" needs to truly engage with Indigenous people and be seen as a national priority. We need to act now to prevent a further increase in cardiometabolic conditions among children and young people, which will have long lasting impacts on Aboriginal and Torres Strait Islander communities.

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Guideline summary

The 2020 Australian guideline for prevention, diagnosis and management of acute rheumatic fever and rheumatic heart disease

Anna P Ralph[1], Sara Noonan[2], Vicki Wade[2], Bart J Currie[1,3]

Abstract

Introduction: Acute rheumatic fever (ARF) and rheumatic heart disease (RHD) cause significant morbidity and premature mortality among Australian Aboriginal and Torres Strait Islander peoples. RHDAustralia has produced a fully updated clinical guideline in response to new knowledge gained since the 2012 edition. The guideline aligns with major international ARF and RHD practice guidelines from the American Heart Association and World Heart Federation to ensure best practice. The GRADE system was used to assess the quality and strength of evidence where appropriate.

Main recommendations: The 2020 Australian guideline details best practice care for people with or at risk of ARF and RHD. It provides up-to-date guidance on primordial, primary and secondary prevention, diagnosis and management, preconception and perinatal management of women with RHD, culturally safe practice, provision of a trained and supported Aboriginal and Torres Strait Islander workforce, disease burden, RHD screening, control programs and new technologies.

Changes in management as a result of the guideline: Key changes include updating of ARF and RHD diagnostic criteria; change in secondary prophylaxis duration; improved pain management for intramuscular injections; and changes to antibiotic regimens for primary prevention. Other changes include an emphasis on provision of culturally appropriate care; updated burden of disease data using linked register and hospitalisations data; primordial prevention strategies to reduce streptococcal infection addressing household overcrowding and personal hygiene; recommendations for population-based echocardiographic screening for RHD in select populations; expanded management guidance for women with RHD or ARF to cover contraception, antenatal, delivery and postnatal care, and to stratify pregnancy risks according to RHD severity; and a priority classification system for presence and severity of RHD to align with appropriate timing of follow-up.

Acute rheumatic fever (ARF) is an autoimmune disease triggered in some children and young adults by infection with group A streptococci.[1] Repeated or severe ARF leads to rheumatic heart disease (RHD), with high morbidity and mortality. Group A streptococcal infection risk is associated with socio-economic factors such as household crowding.[2] High rates occur in Australian Aboriginal and Torres Strait Islander populations, especially in rural or remote settings. Prevalence estimates for definite RHD in Australian children range from < 1 per 1000 population in low risk children, to 33[3] to 50[4] per 1000 people in high risk populations. High rates of disease also occur among Māori and Pacific Islander populations.[5]

Given this high burden of disease in Australian subpopulations, yet rarity in the broader population, clinical practice guidelines are essential. Australian ARF and RHD guidelines were first produced in 2007[6] and revised in 2012.[7] The 2020 guideline, developed in accordance with the National Health and Medical Research Council standards for guidelines[8] by RHDAustralia (the national support unit for ARF and RHD), builds on these and incorporates new evidence from trials and other research, new medication options — such as expanded roles for corticosteroids, and use of non-vitamin K antagonist oral anticoagulants — and new expert consensus opinion including revised parameters for ARF and RHD diagnosis. The guideline[9] is freely available online, accompanied by a video summary of changes, key information, useful tables and figures, an app for smart devices containing a condensed version, and an interactive ARF diagnosis calculator.[10] All electronic resources align with the 2020 edition and the 2015 American Heart Association (AHA) revised Jones criteria for diagnosis of ARF[11] to ensure consistency and best practice.

Methods

A guideline steering committee was formed comprising RHDAustralia members and partners, ARF and RHD experts, and Aboriginal and Torres Strait Islander advisors. The steering committee provided high level strategic direction and advice, content support and endorsement of the final version.

RHDAustralia's Senior Aboriginal Cultural Advisor led a review of all content. A sociocultural framework highlighted social and emotional wellbeing, and ensured that recommendations adhered to culturally safe practice. An Aboriginal and Torres Strait Islander advisory group provided expert cultural advice, with consumer input from community members (Champions4Change[12]). Insights from the Champions introduce each chapter, such as: "You need to understand the community and the problems that they are facing and then, and only then, you can help them to get rid of RHD".[9]

A targeted health workforce survey was conducted to inform the format and scope of the new edition. The 196 respondents (53% urban, 18% rural, 29% remote) indicated that a freely available digital version as well as print copies was desired, with a quick guide format as additional detail. Each chapter structure therefore comprises a key information section followed by an evidence-based discussion and, where relevant, case studies.

The steering committee developed chapter headings and invited multidisciplinary experts (Indigenous and non-Indigenous medical, nursing, research and allied health specialties) from among Australian and New Zealand topic authorities (Supporting Information, Table 1). Authors reviewed relevant chapters from the 2012 edition (unless developing a new chapter), conducted literature reviews using MEDLINE and PubMed Central, and considered in-process citations, research underway and grey literature. The lived experience of ARF and RHD was represented through patient stories and case studies.

The Grading of Recommendations Assessment, Development and Evaluation (GRADE) system[13] was applied by writing

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groups where appropriate. Quality of evidence was classified from A (high) to D (very low) and the strength of the recommendations graded as 1 (strong) or 2 (weak). For example, GRADE 1A indicates that the recommendation should be applied to most patients without reservation, while GRADE 2D indicates that evidence is lacking but expert consensus weakly supports the recommendation. New recommendations not firmly supported by evidence or where evidence was contentious were discussed until consensus was reached, or until an acceptable majority position was obtained considering the available evidence. The aim was to present feasible rather than highly aspirational guidance for ARF and RHD control in such cases; examples include definition of ARF risk groups, duration of secondary prophylaxis, and recommendations for community echocardiogram screening for active case finding in high risk communities. Changes from the previous edition are summarised in Box 1.

Feedback was invited from multidisciplinary content experts in Australia and New Zealand, and recommendations were incorporated where appropriate. Endorsement was sought from peak health policy, advocacy and training organisations (Supporting Information, Table 2). The editors reviewed the

1 Summary of changes from the 2012 edition

Chapter	Changes
Primary prevention	• Updated definition of high risk groups for empirical treatment of throat and skin infections. • New recommendations for management of group A streptococcal skin infections to prevent ARF. • BPG dosing streamlined to three dose bands for simplicity compared with the previously recommended five dose bands. • Option for tablet dosing of cotrimoxazole to treat group A streptococcal impetigo if syrup is in short supply.
Diagnosis of ARF	• In low risk populations, subclinical carditis is now a major criterion. • In low risk populations, ESR as a minor criterion is now ≥ 60 mm rather than ≥ 30 mm. • In low risk populations, fever as a minor criterion is now ≥ 38.5°C rather than ≥ 38.0°C. • For all populations, a definite recurrent episode of ARF after documented history of ARF or RHD now requires two major, or one major and two minor, or three minor criteria (plus evidence of preceding group A streptococcal infection) rather than two major, or one major and one minor, or three minor criteria.
Management of ARF	• Probable ARF – "highly suspected" renamed "probable" ARF. • Probable ARF – "uncertain" renamed "possible" ARF. • Suspected ARF refers to presentations not yet allocated to a category of definite, probable, possible ARF or no ARF. • Non-steroidal anti-inflammatory drugs naproxen and ibuprofen are now recommended first line in children for joint pain, ahead of aspirin. • A revised hierarchy of therapeutic approaches for Sydenham chorea is provided.
Diagnosis of RHD	• World Health Federation guidelines for the diagnosis of RHD, validated in high and low prevalence populations, are endorsed and described. • Echocardiographic features of severity are aligned with updated international guidelines for valvular heart disease (European Society of Cardiology 2017[14] and American Heart Association/American College of Cardiology 2014[15]). • Exercise stress testing has been included as an additional testing modality in determining severity of RHD and planning for intervention. • A new section on distinguishing RHD from other valvular pathology is provided. • The role of new echocardiography technology in RHD diagnosis, including hand-held and portable options, is described.
Secondary prophylaxis	• New recommendations are made for the duration of secondary prophylaxis for some groups. • A focus is provided on the responsibility of health services to provide a culturally safe service, and for staff to be culturally competent in the management of secondary prophylaxis. • The new term benzathine benzylpenicillin G replaces benzathine penicillin G. • BPG is given in units, not weight (g or mg) in keeping with Therapeutic Goods Administration requirements for labelling. • Instructions for intramuscular injection of BPG at the ventrogluteal site are provided. • New approaches are given to managing injection pain, fear and distress, including the option of medically prescribed lidocaine (lignocaine) and procedural sedation.
Management of RHD	• Updated priority definitions have been developed to align with appropriately timed follow-up. • Transcatheter valve replacement is discussed as an option in younger individuals. • A focus on transition from paediatric to adult services is provided. • A definition of non-valvular atrial fibrillation is provided and the role of CHA_2DS_2-VA score to determine thromboembolic risk described. • The role of non-vitamin K antagonist oral anticoagulants (novel oral anticoagulants) in RHD is detailed.
Women and girls with RHD	• Care pathways for girls and women with RHD are provided. • Care pathways and a referral algorithm are provided for pregnant women with RHD. • A new section on transition to adult care, reproductive health, and preconception care is included. • Strategies for a well-planned pregnancy and delivery are discussed. • Anticoagulation during pregnancy has been revised and expanded.
New chapters	• Culture and workforce • Burden of ARF and RHD • Primordial prevention and social determinants of ARF • Primary prevention • Screening for RHD • RHD control programs • New technologies

ARF = acute rheumatic fever; BPG = benzathine benzylpenicillin G; CHA_2DS_2-VA = congestive heart failure, hypertension, age, diabetes, stroke, vascular, age; ESR = erythrocyte sedimentation rate; RHD = rheumatic heart disease. ◆

semi-final draft to ensure inclusion of any recently published or in-press literature, consistency across chapters, clarity for practitioners, and alignment with other Australian and international guidelines. Where recommendations departed from other local guidelines (eg, Australian Therapeutic Guidelines: Antibiotic [https://www.tg.org.au] or the Central Australian Rural Practitioners Association Standard Treatment Manual[16]), this was communicated to respective editors to encourage alignment in their next edition.

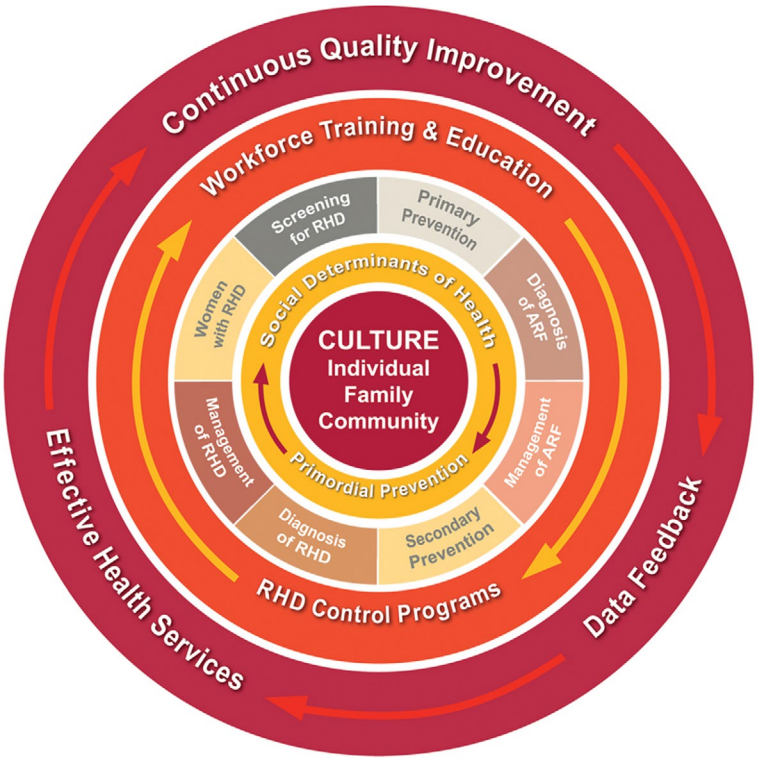
Culture and workforce

The guideline is underpinned by a strong focus on the provision of culturally safe care, in line with national recommendations.[17] Cultural safety requires health care providers and institutions to recognise their own culture and ameliorate approaches which diminish, demean or disempower the cultural identities and wellbeing of patients.[18] A socio-ecological model was developed for the guideline (Box 2), describing influences shaping Aboriginal and Torres Strait Islander peoples' health care interactions. The value of fostering a strong Aboriginal health workforce in delivering care is highlighted to support effective client engagement.[19,20]

Burden of disease

We developed a new chapter on burden of disease, using original data from the End RHD in Australia Study of Epidemiology (ERASE) Project,[21] Australian Institute of Health and Welfare data[22] and other sources. Since the early 1990s, ARF has occurred almost exclusively in young Aboriginal and Torres Strait Islander people (94% of cases; 33% affecting 5–14-year-olds). The recognised female predominance in rates of ARF (56%) and RHD (61%) is reiterated.[22]

2 Socio-ecological model underpinning the guidelines



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Recommendations

Primordial prevention and social determinants of ARF

Primordial prevention reduces community-based risk factors to prevent the occurrence of a disease. ARF is attributable to social determinants of health, including quality of housing, level of household occupancy, and access to health hardware including washing facilities.[2,23] The Nine Healthy Living Practices[24,25] are simple recommendations to reduce the risk of injury, communicable diseases and environmental diseases in household settings. They are used as the framework for this chapter. Each Practice was reviewed and the evidence graded regarding their likely association with reducing streptococcal infection at community level. The two practices for which the evidence is graded as strong are "washing people" and "reducing negative impacts of overcrowding".

Primary prevention

People at high risk of ARF (Box 3) require empirical antibiotic treatment for sore throat, with penicillin the first line choice (GRADE 1B).[26–28] Impetigo caused by group A streptococcus is very common among Aboriginal and Torres Strait Islander children living in remote areas, with almost one in two affected at any time.[29] Identification, treatment and prevention of group A streptococcal skin infections may help reduce ARF burden (GRADE 1B).[30–34] Group A streptococcal skin infections should be treated with cotrimoxazole orally or benzathine benzylpenicillin G intramuscularly (GRADE 1A).[35] Dosing regimens are provided in the full guideline (Table 5.3).[9]

3 Risk groups for acute rheumatic fever (ARF) and rheumatic heart disease (RHD)*

Risk	Setting
High risk	• Living in an ARF-endemic setting† • Aboriginal and Torres Strait Islander peoples living in rural or remote settings • Aboriginal and Torres Strait Islander peoples, and Māori and/or Pacific Islander peoples living in metropolitan households affected by crowding and/or lower socio-economic status • Personal history of ARF/RHD and age < 40 years
May be high risk	• Family or household recent history of ARF/RHD • Household overcrowding (> 2 people/bedroom) or low socio-economic status • Migrant or refugee from low or middle income country and their children
Additional considerations which increase risk	• Prior residence in a high ARF risk setting • Frequent or recent travel to a high ARF risk setting • Aged 5–20 years (peak years for ARF)

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4 2020 Australian criteria for acute rheumatic fever (ARF) diagnosis*

	High risk groups†	Low risk groups
Definite initial episode of ARF	• 2 major manifestations + evidence of preceding group A streptococcal infection, OR • 1 major + 2 minor manifestations + evidence of preceding group A streptococcal infection‡	
Definite recurrent‡ episode of ARF in patient with documented history of ARF or RHD	• 2 major manifestations + evidence of preceding group A streptococcal infection, OR • 1 major + 2 minor manifestations + evidence of preceding group A streptococcal infection,‡ OR • 3 minor manifestations + evidence of a preceding group A streptococcal infection‡	
Probable or possible ARF (first episode or recurrence§)	A clinical presentation in which ARF is considered a likely diagnosis but falls short in meeting the criteria by either: • one major or one minor manifestation, OR • no evidence of preceding group A streptococcal infection (streptococcal titres within normal limits or titres not measured) Such cases should be further categorised according to the level of confidence with which the diagnosis is made: • probable ARF (previously termed "probable: highly suspected") • possible ARF (previously termed "probable: uncertain")	
Major manifestations	• Carditis (including subclinical evidence of rheumatic valvulitis on echocardiogram) • Polyarthritis¶ or aseptic monoarthritis or polyarthralgia • Sydenham chorea** • Erythema marginatum†† • Subcutaneous nodules	• Carditis (including subclinical evidence of rheumatic valvulitis on echocardiogram) • Polyarthritis¶ • Sydenham chorea** • Erythema marginatum†† • Subcutaneous nodules
Minor manifestations	• Fever‡‡ ≥ 38°C • Monoarthralgia§§ • ESR ≥ 30 mm/h or CRP ≥ 30 mg/L • Prolonged PR interval on ECG¶¶	• Fever ≥ 38.5°C • Polyarthralgia or aseptic monoarthritis§§ • ESR ≥ 60 m/h or CRP ≥ 30 mg/L • Prolonged PR interval on ECG¶¶

CRP = C-reactive protein; ECG = electrocardiogram; ESR = erythrocyte sedimentation rate; RHD = rheumatic heart disease.*Reproduced with permission from Menzies School of Health Research, Charles Darwin University, Darwin, Australia, which holds copyright: https://www.rhdaustralia.org.au/resources/2020-guideline-prevention-diagnosis-and-management-acute-rheumatic-fever-and-rheumatic.[9] †High risk groups are those living in communities with high rates of ARF (incidence > 30/100 000 per year in 5–14-year-olds) or RHD (all-age prevalence > 2/1000). Aboriginal and Torres Strait Islander peoples living in rural or remote settings are known to be at high risk. Data are not available for other populations but Aboriginal and Torres Strait Islander peoples living in urban settings, Māori and Pacific Islanders, and potentially immigrants from developing countries, may also be at high risk. ‡Elevated or rising antistreptolysin O or other streptococcal antibody, or a positive throat culture or rapid antigen or nucleic acid test for group A streptococcal infection. §Recurrent definite, probable or possible ARF requires a time period of more than 90 days after the onset of symptoms from the previous episode of definite, probable or possible ARF. ¶A definite history of arthritis is sufficient to satisfy this manifestation. Note that if polyarthritis is present as a major manifestation, polyarthralgia or aseptic monoarthritis cannot be considered an additional minor manifestation in the same person. ** Chorea does not require other manifestations or evidence of preceding group A streptococcal infection, provided other causes of chorea are excluded. ††Care should be taken not to label other rashes, particularly non-specific viral exanthems, as erythema marginatum. ‡‡In high risk groups, fever can be considered a minor manifestation based on a reliable history (in the absence of documented temperature) if anti-inflammatory medication has already been administered. §§If polyarthritis is present as a major criterion, monoarthritis or arthralgia cannot be considered an additional minor manifestation. ¶¶If carditis is present as a major manifestation, a prolonged PR interval cannot be considered an additional minor manifestation. ◆

ARF diagnosis

There is now alignment between Australian diagnostic criteria for ARF and the AHA revised Jones criteria[11] (Box 4), outlining differences for high and low risk populations (Box 3). The changes to diagnostic criteria in low risk groups include a higher temperature (≥ 38.5°C rather than ≥ 38°C), higher erythrocyte sedimentation rate (≥ 60 mm/h rather than ≥ 30 mm/h), and echocardiographic evidence of valvulitis with carditis. A combination of major and minor criteria is used to diagnose ARF; major criteria including arthritis, carditis, chorea and skin manifestations are strongly associated with ARF, while minor criteria such as fever and raised inflammatory markers support the diagnosis. For all populations, definite recurrent ARF now requires two major, or one major and *two minor*, or three minor criteria, rather than two major, or one major and one minor, or three minor criteria. Alignment with the AHA is important to promote a consistent approach to ARF diagnosis globally, and the changes also improve specificity of ARF diagnosis, especially in low risk populations where ARF is very uncommon.

All patients with suspected ARF should be hospitalised, investigated with electrocardiography and echocardiography, and have differential diagnoses excluded (GRADE 1B). Each episode should be categorised as initial or recurrent ARF, with certainty of diagnosis indicated as definite, probable or possible:

- *definite ARF* meets revised Jones criteria with alternative diagnoses excluded;
- *probable ARF* is an acute presentation not fulfilling criteria, missing one major or one minor criterion or lacking evidence of preceding streptococcal infection, but where ARF is still considered the most likely diagnosis; and
- *possible ARF* applies to the same presentation type as probable ARF, but where ARF is considered uncertain but cannot be ruled out.

ARF management

The pillars of ARF management are eradication of the inciting group A streptococcal infection using penicillin (or an alternative if allergic to penicillin) and management of symptoms with analgesic–antipyretic agents as needed (GRADE 1B). The guideline discusses the use of corticosteroids as a potential disease-modifying agent in severe rheumatic carditis (GRADE 2B), and to reduce severity of Sydenham chorea (GRADE 2B).

For definite ARF, a priority grade based on the severity of any accompanying RHD should be assigned, using a revised priority classification (Supporting Information, Table 3). The time since ARF and the severity of RHD determine the duration of secondary prophylaxis (Box 5) and the priority grade determines frequency of reviews and echocardiograms. People diagnosed with ARF must be notified to the local public health

Guideline summary

5 Recommended duration of secondary prophylaxisfor acute rheumatic fever (ARF) and rheumatic heart disease (RHD)*

Diagnosis	Definition	Duration of prophylaxis	Conditions for ceasing prophylaxis†	Timing of echocardiography after cessation‡
Possible ARF (no cardiac involvement)	Incomplete features of ARF with normal echocardiogram and normal ECG§ throughout ARF episode	12 months (then reassess)	• No signs and symptoms of ARF within the previous 12 months • Normal echocardiogram	At 1 year
Probable ARF	Highly suspected ARF with normal echocardiogram	Minimum of 5 years after most recent episode of probable ARF, or until age 21 years (whichever is longer)	• No probable or definite ARF within the previous 5 years • Normal echocardiogram	At 1, 3 and 5 years
Definite ARF (no cardiac involvement)	ARF with normal echocardiogram and normal ECG§ throughout ARF episode	Minimum of 5 years after most recent episode of ARF, or until age 21 years (whichever is longer)	• No probable or definite ARF within the previous 5 years • Normal echocardiogram	At 1, 3 and 5 years
Definite ARF(with cardiac involvement)	ARF with carditis or RHD on echocardiogram, or with atrioventricular conduction abnormality on ECG§ during ARF episode	According to relevant RHD severity		
Borderline RHD (≤ 20 years of age only)	Borderline RHD on echocardiogram without a documented history of ARF	Not usually recommended¶		Medical review and repeat echocardiogram at 1, 3 and 5 years after diagnosis
Mild RHD††	Echocardiogram showing: • mild regurgitation or mild stenosis of a single valve, OR • atrioventricular conduction abnormality on ECG§ during ARF episode	• If documented history of ARF: minimum of 10 years after the most recent episode of ARF, or until age 21 years (whichever is longer) • If NO documented history of ARF and aged <35 years: ‡‡ minimum of 5 years following diagnosis of RHD or until age 21 years (whichever is longer)	• No probable or definite ARF within the previous 10 years, no progression of RHD • Stable echocardiographic features for 2 years	At 1, 3 and 5 years
Moderate RHD††,§§	Echocardiogram showing: • moderate regurgitation or moderate stenosis of a single valve, OR • combined mild regurgitation and/or mild stenosis of one or more valves Examples: • mild mitral regurgitation and mild mitral stenosis • mild mitral regurgitation and mild aortic regurgitation	• If documented history of ARF: minimum of 10 years after the most recent episode of ARF or until age 35 years (whichever is longer) • If no documented history of ARF and aged < 35 years: ‡‡ minimum of 5 years following diagnosis of RHD or until age 35 years (whichever is longer)	• No probable or definite ARF within the previous 10 years • Stable echocardiographic features for 2 years	Initially every 12 months
Severe RHD§§,¶¶	Echocardiogram showing: • severe regurgitation or severe stenosis of any valve, OR • combined moderate regurgitation and/or moderate stenosis of one or more valves Examples: • moderate mitral regurgitation and moderate mitral stenosis • moderate mitral stenosis and moderate aortic regurgitation, OR • past or impending valve repair or prosthetic valve replacement	• If documented history of ARF: minimum of 10 years after the most recent episode of ARF or until age 40 years (whichever is longer) • If no documented history of ARF:††† minimum of 5 years following diagnosis of RHD or until age 40 years (whichever is longer)	• Stable valvular disease/ cardiac function on serial echocardiogram for 3 years, OR • Patient or family preference to cease due to advancing age and/or end of life care	Initially every 6 months

AV = atrioventricular; ECG = electrocardiogram.*Reproduced with permission from Menzies School of Health Research, Charles Darwin University, Darwin, Australia, which holds copyright: https://www.rhdaustralia.org.au/resources/2020-guideline-prevention-diagnosis-and-management-acute-rheumatic-fever-and-rheumatic.[9] †All people receiving secondary prophylaxis require a comprehensive clinical assessment and echocardiogram before cessation. Risk factors including future exposure to high streptococcal burden environments need to be considered. ‡Echocardiography may be more frequent based on clinical status and specialist review. §Normal ECG means no AV conduction abnormality during the ARF episode — including first degree heart block, second degree heart block, third degree (complete) heart block and accelerated junctional rhythm. ¶Secondary prophylaxis may be considered in some circumstances, including family preference, family history of rheumatic heart valve surgery, or suspected retrospective history of ARF. If prophylaxis is commenced, consider ceasing after 1–3 years if no history of ARF and if echocardiographic features have resolved or not progressed to definite RHD. ††Prophylaxis may be considered for longer in women considering pregnancy who live in high risk circumstances for ARF. ‡‡If diagnosed with mild or moderate RHD aged ≥ 35 years (without ARF), secondary prophylaxis is not required. §§Rarely, moderate or severe RHD may improve on echocardiogram without valve surgery. In these cases, the conditions for ceasing prophylaxis can change to follow the most relevant severity category. For instance, if moderate RHD improves to mild on echocardiogram, recommendations for mild RHD can then be instigated. ¶¶Risk of ARF recurrence is low in people aged ≥ 40 years; however, lifelong secondary prophylaxis is usually recommended for patients who have had, or are likely to need, heart valve surgery. †††If diagnosed with severe RHD aged ≥ 40 years (without ARF), specialist input is required to determine the need for secondary prophylaxis. ◆

unit in accordance with Australian state and territory legislation and be registered with the jurisdictional RHD control program (GRADE 1B).

Diagnosis of RHD

The guideline provides more detail on the use of echocardiogram in accordance with World Heart Federation recommendations on echocardiographic diagnosis of RHD,[36] which provide criteria distinguishing pathological RHD from physiological changes (GRADE 1B). Exercise testing or stress echocardiography is recommended when severity of symptoms and echocardiographic findings are discordant (GRADE 1B). Transoesophageal echocardiography may help in planning surgical intervention (GRADE 1B).

RHD is also notifiable in Western Australia, South Australia, Northern Territory, Queensland, and New South Wales (RHD for people aged < 35 years).[5]

Screening for RHD

Population screening for RHD may provide more accurate estimates of disease burden and an opportunity to initiate management for people with previously unrecognised RHD. Population-based screening using auscultation, inaccurate for detecting RHD, is not recommended (GRADE 1A). Screening using echocardiography can accurately detect previously undiagnosed RHD (GRADE 1A). Echocardiographic screening procedures have evolved using different technologies and operators with varying levels of expertise.[4] Echocardiographic screening for RHD meets some but not all public health criteria for community screening.[37] The disease does place a significant burden on at-risk populations, there is a latent stage that can be identified, and there is treatment in the form of secondary prophylaxis and cardiological or surgical intervention. However, the impact of secondary prophylaxis on the trajectory of screen-detected RHD is not yet defined, and feasible community screening tools have thus far demonstrated inadequate sensitivity and specificity.[4] While there remains insufficient evidence to recommend routine population-level echocardiographic screening for RHD in Australia as a method of disease detection and control (GRADE 2B), it is recognised that echocardiographic community screening is valuable under specific circumstances such as clusters of ARF or suspected extreme rates of RHD.[4]

Secondary prophylaxis ARF

Secondary prophylaxis comprises regular administration of antibiotics after diagnosis of ARF or RHD to prevent future group A streptococcal infections and ARF recurrence. Group A streptococcus does not develop resistance to penicillin, although one instance of acquisition of reduced ampicillin susceptibility has been reported.[38] Long acting benzathine benzylpenicillin G delivered every 28 days is the first line recommendation for ARF prophylaxis (GRADE 1B). Previously, secondary prophylaxis was recommended in Australia for at least 10 years after the most recent episode of ARF or until 21 years of age, whichever comes later. The 2020 guideline recommends secondary prophylaxis for 5 years after the most recent episode of ARF or until 21 years of age if there has been no acute cardiac involvement evident on electrocardiograph or echocardiogram during ARF, and follow-up and end-of-treatment echocardiograms confirm ongoing absence of valvular involvement (Box 5). This is more aligned with international guidelines[39,40] and is supported by Australian register data.

Management of RHD

Every patient with RHD should have access to specialist paediatric or adult cardiology services, and coordinated transition from paediatric to adult care (GRADE 2A). Non-vitamin K antagonist oral anticoagulants can be used in patients with RHD-related atrial fibrillation or elevated CHA_2DS_2-VA (congestive heart disease, hypertension, age, diabetes, stroke, vascular) score, even if valvular disease is present, provided there is no mitral stenosis of moderate or greater severity and no mechanical valve in situ (GRADE 2B). For patients with moderate or severe mitral stenosis and atrial fibrillation, vitamin K antagonists (eg, warfarin) currently remain the only indicated oral anticoagulant (GRADE 1B).[41]

Surgical decision making must take into consideration a patient's personal, social and cultural situation. Early engagement of a multidisciplinary team is essential to determine the appropriate choice and timing of intervention. Surgical options include repair, bioprosthetic or mechanical valve replacement, and transcatheter valve replacement. Key considerations are the patient's age, risks of anticoagulation, anticipated adherence, plans for future pregnancy, and durability of valve repair and prosthesis.

Antibiotic prophylaxis for endocarditis prevention with amoxycillin (first line) is recommended in all people with RHD undergoing invasive procedures as defined in Table 11.5 of the guideline[9] (GRADE 1C).

Females with RHD

About 61% of RHD cases in Australia occur in females.[5] Women with moderate or greater mitral stenosis, severe mitral or aortic regurgitation, severe aortic stenosis, pulmonary hypertension or heart failure are at high risk of cardiac events during pregnancy and have an elevated chance of adverse fetal outcomes. A left ventricular ejection fraction < 30% or reduced systolic function with New York Heart Association class III–IV symptoms is associated with high maternal morbidity or mortality, and pregnancy is strongly discouraged.[42] Conversely, selected women with mild RHD can safely conceive and have children. In 2–3% of annually recorded pregnancies among Aboriginal women in the Northern Territory, the women have RHD. Women with mild RHD may be able to give birth on Country, an important cultural practice for many Aboriginal and Torres Strait Islander people.[43]

Pre-conception diagnosis of RHD is critical to optimise management including potential surgery. Long acting, reversible contraceptives (eg, intra-uterine contraceptive devices, etonogestrel implants) are recommended for women who agree to avoid pregnancy after advice. Oestrogen-containing contraceptives are associated with elevated risk of thrombosis (GRADE 1A) and should be avoided if additional thrombosis risks are present.

A pregnant woman in a high risk group for ARF and RHD who presents with breathlessness, orthopnoea, wheeze or worsening fatigue should be investigated with an echocardiogram (GRADE 1A). Normal vaginal delivery is generally preferred for women with RHD. Epidural anaesthesia (after appropriately timed, short term cessation of any anticoagulation) may be indicated to reduce tachycardia and hypertension that can precipitate acute heart failure during delivery.

RHD control programs

Comprehensive RHD control programs can provide effective approaches to reducing the burden of RHD (GRADE 1B). RHD control programs in Australia maintain register and recall systems

for secondary prophylaxis and optimum clinical management; support patient care and education about ARF and RHD through workforce education and training; promote primary prevention aimed at preventing initial episodes of ARF; and provide jurisdiction-wide data for epidemiological reporting (https://www.rhdaustralia.org.au/control-programs).

New technologies

Research underway in Australasia aims to discover alternatives to or improvement in delivery of benzathine benzylpenicillin G, develop a group A streptococcus vaccine, and develop a diagnostic test for ARF.

Conclusion

The 2020 ARF and RHD guideline places person and culture at the centre of care and synthesises the current evidence to provide expert clinical guidance from prevention through to tertiary care.

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Supporting Information

Additional Supporting Information is included with the online version of this article.

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