Two nations: racial disparities in bloodstream infections recorded at Alice Springs Hospital, central Australia, 2001–2005

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ABSTRACT

Objective: To compare bloodstream infection (BSI) rates, pathogens and mortality among Indigenous and non-Indigenous adults in central Australia.

Design, participants and setting: Retrospective study of adult patients (aged ⩾ 15 years) admitted to Alice Springs Hospital (ASH) between 1 January 2001 and 31 December 2005. Patients were followed up until 30 June 2008.

Main outcome measures: Admission-based and population-based BSI rates and mortality rates for Indigenous and non-Indigenous adults.

Results: During the study period, there were 824 BSI episodes (Indigenous, 753; non-Indigenous, 71). The admission-based BSI rate for Indigenous patients was 26.5 (95% CI, 26.4–26.6) per 1000 adult admissions, compared with 5.2 (95% CI, 5.1–5.2) per 1000 adult admissions for non-Indigenous patients (infection rate ratio [IRR], 5.13 [95% CI, 5.10–5.18]). The population-based BSI rate was 1354.7 (95% CI, 1256.3–1460.8) per 100 000 persons per year among Indigenous patients and 69.9 (95% CI, 55.1–88.6) per 100 000 persons per year among non-Indigenous patients (IRR, 19.4 [95% CI, 15.1–24.9]). These differences were not explained by higher comorbidity levels among Indigenous patients. Human T-cell lymphotropic virus type 1 and *Strongyloides stercoralis* infected 43% and 35%, respectively, of Indigenous patients tested. The risk of death during the follow-up period was 32.1% for Indigenous and 13.4% for non-Indigenous patients (hazard ratio [HR], 2.69 [95% CI, 1.38–5.25]; $P = 0.004$). Mortality rates were higher among Indigenous patients who had more than a single BSI (HR, 1.86 [95% CI, 1.32–2.62]; $P < 0.001$). The mean age at death was 48.5 years (SD, 16.2 years) for Indigenous patients and 75.1 years (SD, 18.7 years) for non-Indigenous patients ($P < 0.001$).

Conclusion: Indigenous adults living in central Australia experience BSI rates that are among the highest reported in the world. These are associated with a high risk of death, and are a likely consequence of the poor socioeconomic circumstances of Indigenous people.

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Indigenous Australians continue to live in conditions of enormous socioeconomic disadvantage and this is reflected in a life expectancy 17 years less than that of their non-Indigenous peers.[1] Chronic diseases such as diabetes mellitus are thought to be the major cause of life-years lost for Indigenous people in the Northern Territory.[2] However, these conditions may have less influence on life expectancy in central Australia, where infection-related mortality rates are higher than the rates in some African countries before the arrival of the current HIV pandemic.[3]

High background rates of infectious diseases among Indigenous Australians may underlie racial disparities in infection-related mortality in central Australia. Incidence rates of invasive pneumococcal disease[4] and prevalence rates of bronchiectasis,[5] for example, are among the highest in the world. The latter is associated with infection with the human T-cell lymphotropic virus type 1 (HTLV-1),[5] which is endemic in central Australia but rare elsewhere.[6] HTLV-1 infection predisposes to infection with the parasite *Strongyloides stercoralis* and to crusted scabies, providing portals of entry for enteric and gram-positive organisms, respectively.[7,8] Deaths resulting from complicated strongyloidiasis have recently been reported at Alice Springs Hospital (ASH), in central Australia.[7] Bloodstream infections (BSIs) continue to be associated with short-term mortality rates of 13%–21%,[9-11] and 38% of infection-related deaths among Indigenous adults at ASH follow a BSI.[3] The aim of our study was to characterise the pathogens responsible for BSI in central Australia and to determine the extent to which racial background influences BSI rates and mortality.

METHODS

We conducted a retrospective review of all positive blood cultures (BCs) collected from patients admitted to ASH between 1 January 2001 and 31 December 2005. Data were obtained from the ASH microbiology database. Due to differences between adults and children in susceptibility to and outcomes of infection, microbiology and mortality data presented here pertain only to adult patients. Incidence rates for children (age < 15 years) are included to permit comparison with other studies.

Potential contaminants in BCs, including coagulase-negative staphylococci and coryneform bacteria, were excluded unless isolated from more than one BC. Before 2006, viridans group streptococci were regarded as contaminants and excluded, as were *Acinetobacter* spp if not identified to species level.

A BC from which a pathogen was isolated defined a "BSI episode". Repeated instances of the same organism being cultured were excluded unless the episodes were more than 1 month apart. Population-based incidence rates were calculated for a given patient using only the index BSI episode. All episodes were included in the racial comparison of the responsible pathogens.

Demographic details were obtained for each patient from the ASH patient information database. For the years 2003–2005, dates of admission and morbidity codes were also recorded. Episodes during this period were categorised as "community-acquired" if BCs were drawn within 48 hours of admission or "health care-associated" if patients were receiving renal replacement therapy or chemotherapy, were admitted from a nursing home, or had a BC drawn more than 48 hours after admission.

Our study was approved by the Central Australian Human Research Ethics Committee.

Statistical analysis

Adult admission-based BSI rates (ie, number of BSIs per 1000 adult admissions) were calculated using the number of adult medical and renal patient admissions for each year as the denominator (admissions for day procedures or haemodialysis were excluded). Population-based BSI rates were calculated using age-stratified population data for the Alice

Springs region from the Australian Bureau of Statistics. All BSI rate comparisons were performed using Poisson regression. Survival rates for Indigenous and non-Indigenous adult patients with a BSI were compared using Kaplan–Meier plots, log-rank tests and Cox regression. The period of follow-up was to 30 June 2008. All analysis was performed using Stata software, version 10.1 (StataCorp, College Station, Tex, USA).

RESULTS

Between 2001 and 2005, 824 BSI episodes (753 in Indigenous and 71 in non-Indigenous patients) were recorded among 683 adults (614 Indigenous and 69 non-Indigenous) (Box 1). Health care-associated BSIs were uncommon in both groups, representing 45/485 episodes (9.3%) for Indigenous and 6/51 episodes (11.8%) for non-Indigenous patients. With the exception of malignancy and age, risk factors for sepsis were more common among Indigenous patients (Box 1).

The admission-based BSI incidence rate between 2001 and 2005 was 26.5 (95% CI, 26.4–26.6) per 1000 adult admissions for Indigenous adults compared with 5.2 (95% CI, 5.1–5.2) per 1000 adult admissions for non-Indigenous adults (infection rate ratio [IRR], 5.13 [95% CI, 5.10–5.18]) (Box 2). Population-based BSI rates for the same period were 1354.7 (95% CI, 1256.3–1460.8) per 100 000 persons per year for Indigenous adults compared with 69.9 (95% CI, 55.1–88.6) per 100 000 persons per year for non-Indigenous adults (IRR, 19.4 [95% CI, 15.1–24.9]) (Box 2). Among Indigenous adults, population-based BSI rates increased sharply in the 30–44-years age group and remained relatively constant thereafter (Box 3).

Pathogens

The pathogens isolated from Indigenous and non-Indigenous adults are summarised in Box 4. Among Indigenous patients tested, HTLV-1 western blots were positive for 43.0% and *S. stercoralis* serology was positive for 35.4% (Box 1). Of 78 HTLV-1 seropositive Indigenous adults who were also tested serologically for *S. stercoralis* infection, 37 (47.4%) were seropositive.

Mortality

Among 614 Indigenous and 69 non-Indigenous adults there were 197 Indigenous and nine non-Indigenous deaths. For Indigenous patients, the mean age of death was 48.5 years (SD, 16.2 years), compared with 75.1 years (SD, 18.7 years) for non-Indigenous patients ($P<0.001$). The risk of death during follow-up was 32.1% in Indigenous patients and 13.4% in non-Indigenous patients (hazard ratio [HR], 2.69 [95% CI, 1.38–5.25]; $P=0.004$) (Box 5, A). Short-term mortality rates were similar between Indigenous and non-Indigenous adults: 7-day HR, 1.32 (95% CI, 0.48–3.69) ($P=0.59$); 30-day HR, 1.47 (95% CI, 0.64–3.37) ($P=0.37$). Risk of death was higher for Indigenous patients between 1 month after a BSI and the end of follow-up (HR, 5.16 [95% CI, 1.64–16.22]; $P=0.005$). Indigenous adults were more likely than non-Indigenous adults to have at least two BSI episodes (16.5% v 5.0%; $P=0.019$) (Box 1), and mortality among these patients was significantly higher than for Indigenous adults who had only a single episode (Box 5, B).

1 Patient characteristics, outcomes and comorbid conditions for Indigenous and non-Indigenous adults admitted to Alice Springs Hospital with BSIs, 2001–2005

	Indigenous patients	Non-indigenous patients	*P*
Characteristics	(*n* = 614)	(*n* = 69)	
Mean age, in years (SD)	44.0 (15.2)	57.3 (20.4)	< 0.001
Age > 65 years, *n* (%)	64 (10.4%)	29 (42.0%)	< 0.001
Male, *n* (%)	271 (44.1%)	43 (62.3%)	< 0.001
Residence outside region, *n* (%)*	110 (17.9%)	24 (34.8%)	< 0.001
Outcomes			
30-day mortality, *n* (%)	76 (12.4%)	6 (8.7%)	0.38†
Overall mortality, *n* (%)	197 (32.1%)	9 (13.0%)	0.003†
Mean age at death, in years (SD)	48.5 (16.2)	75.1 (18.7)	< 0.001
Recurrent BSI, *n* (%)‡	91 (16.5%)	3 (5.0%)	0.019
Comorbid conditions, *n* (%)§	(*n* = 392)	(*n* = 44)	*P*¶
Diabetes mellitus	164 (41.8%)	6 (13.6%)	0.16
Alcohol dependence	152 (38.8%)	6 (13.6%)	0.21
Chronic renal failure	59 (15.1%)	2 (4.5%)	0.68
Haemodialysis	49 (12.5%)	0	< 0.001
Chronic liver disease	14 (3.6%)	0	< 0.001
Congestive cardiac failure	8 (2.0%)	1 (2.3%)	0.99
Malignancy	5 (1.3%)	4 (9.1%)	0.58
HTLV-1**	116/270 (43.0%)	nt	
Strongyloidiasis††	73/206 (35.4%)	nt	

BSI = bloodstream infection. HTLV-1 = human T-cell lymphotropic virus type 1. nt = not tested.
* Residence outside Alice Springs rural area. † Log-rank test. ‡ Among patients who survived > 30 days after initial BSI (Indigenous [*n* = 550]; non-Indigenous [*n* = 60]). § Comorbidities recorded for patients admitted over the period 2003–2005. ¶ Binomial test of proportions. ** Positive Western blot test for HTLV-1. †† Positive serology for *Strongyloides stercoralis*. ◆

DISCUSSION

Our study reports one of the highest BSI incidence rates worldwide among a population of Indigenous adults residing in central Australia. Population-based estimates suggest that, each year over the period 2001–2005, nearly 2% of all Indigenous adults aged over 29 years suffered a life-threatening BSI. The combined admission-based cumulative incidence for Indigenous adults and children reported here (22.7/1000) approximate those for community-acquired BSI in Nigeria before the HIV pandemic (23.0/1000)[12] and exceed those of hospitals in Thailand (9.5/1000)[13] and Vietnam (20.4/per 1000).[14] In contrast, admission-based (5.2/1000) and population-based (70/100 000 adult population per year) BSI rates for non-Indigenous adults were close to admission-based rates for adults with community-acquired BSI (4.7–5.4/1000)[10,11,15,16] and consistent with population-based rates for all-cause sepsis (240–275/100 000 adult population per year)[9,17] in other developed countries.

An increased susceptibility to infection as a result of comorbid conditions cannot fully account for the marked racial disparities in

BSI rates in central Australia. Diabetes and alcohol dependence were the most frequent comorbidities among Indigenous adults, yet these are associated with only modest increases in the risk of invasive bacterial infection.[18,19] Rates of diabetes mellitus among African Americans presenting with sepsis, for example, may approach those reported here,[20] but this is associated with only a two-fold increase in sepsis risk relative to white Americans.[9,17] In Australia, by contrast, profound differences exist between racial groups in their socioeconomic circumstances and resultant environmental exposure that might explain the markedly higher risk of BSI among Indigenous Australians. In some remote Indigenous communities the mean number of people living per house continues to be as high as 17;[21] nearly half these houses do not have functioning sanitation, and opportunities to maintain skin hygiene are limited.[22] In such environments, exposure to bacterial pathogens and *S. stercoralis* is likely, and HTLV-1 infection may further increase risk by predisposing people to complicated strongyloidiasis[7] and crusted scabies.[8]

Short-term mortality rates were comparable between racial groups, but Indigenous adults were more than five times more likely to die from 1 month after a BSI, at a mean age that was 26.6 years lower than that of their non-Indigenous peers. We were unable to attribute cause of death, but our results suggest that reinfection may have been a major contributor to out-of-hospital mortality. Consistent with their very high background BSI rates, Indigenous adults were three times more likely to be readmitted with a further BSI; and compared with Indigenous adults who had only a single BSI episode, those experiencing recurrence had nearly double the risk of death.

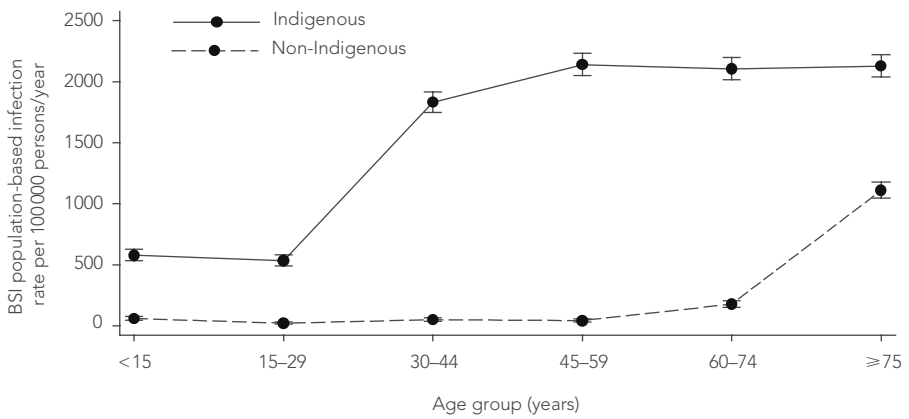
A limitation to our study lay in our inability to precisely determine population-based rates. The Indigenous population of central Australia is extremely mobile and, even when people have relocated to Alice Springs, ASH continues to record the originating community as the primary place of residence. Some visitors to Alice Springs will therefore have been included in our population-based estimates. However, excluding all Indigenous patients associated with a community outside the Alice Springs region would reduce the population-based BSI rate by only 17.9%. Moreover, at least 65% of Indigenous people reside in remote communities whose health clinics do not use ASH microbiology services, and patients with

2 Infection rates and infection rate ratios for Indigenous and non-Indigenous patients admitted to Alice Springs Hospital with BSIs, 2001–2005

	Number of BSIs	Infection rate (95% CI)*	Infection rate ratio (95% CI)
Admission-based BSIs*			
Adults and children			
Non-Indigenous	87	4.82 (4.79–4.85)	1.0
Indigenous	896	22.70 (22.65–22.74)	4.71 (4.68–4.74)
All adults			
Non-Indigenous	71	5.2 (5.1–5.2)	1.0
Indigenous	753	26.5 (26.4–26.6)	5.13 (5.10–5.18)
Population-based BSIs*			
Adults and children			
Non-Indigenous	84	67.7 (54.7–83.9)	1.0
Indigenous	819	1097.6 (1024.9–1175.4)	16.2 (12.9–20.3)
All adults			
Non-Indigenous	68	69.9 (55.1–88.6)	1.0
Indigenous	676	1354.7 (1256.3–1460.8)	19.4 (15.1–24.9)
Adults 15–29 years			
Non-Indigenous	6	22.0 (9.9–49.0)	1.0
Indigenous	114	533.8 (444.3–641.3)	24.2 (10.7–55.1)
Adults 30–44 years			
Non-Indigenous	18	50.7 (31.9–80.5)	1.0
Indigenous	282	1832.2 (1630.4–2059.1)	36.1 (22.4–58.2)
Adults 45–59 years			
Non-Indigenous	11	43.1 (23.9–77.9)	1.0
Indigenous	173	2140.0 (1843.8–2483.9)	49.6 (27.0–91.2)
Adults 60–74 years			
Non-Indigenous	13	178.4 (103.6–307.2)	1.0
Indigenous	82	2105.8 (1696.0–2614.7)	11.8 (6.8–21.2)
Adults ⩾ 75 years			
Non-Indigenous	20	1109.9 (716.0–1720.3)	1.0
Indigenous	25	2127.7 (1437.7–3148.8)	1.9 (1.1–3.5)

BSI = bloodstream infection. * Infection rates are BSIs per 1000 adult admissions (admission-based BSIs) or BSIs per 100 000 adult population per year (population-based BSIs). ◆

3 Population-based BSI incidence rates among Indigenous and non-Indigenous patients admitted to Alice Springs Hospital, by age, 2001–2005



4 Pathogens isolated from blood cultures taken from adult patients admitted to Alice Springs Hospital, 2001–2005*

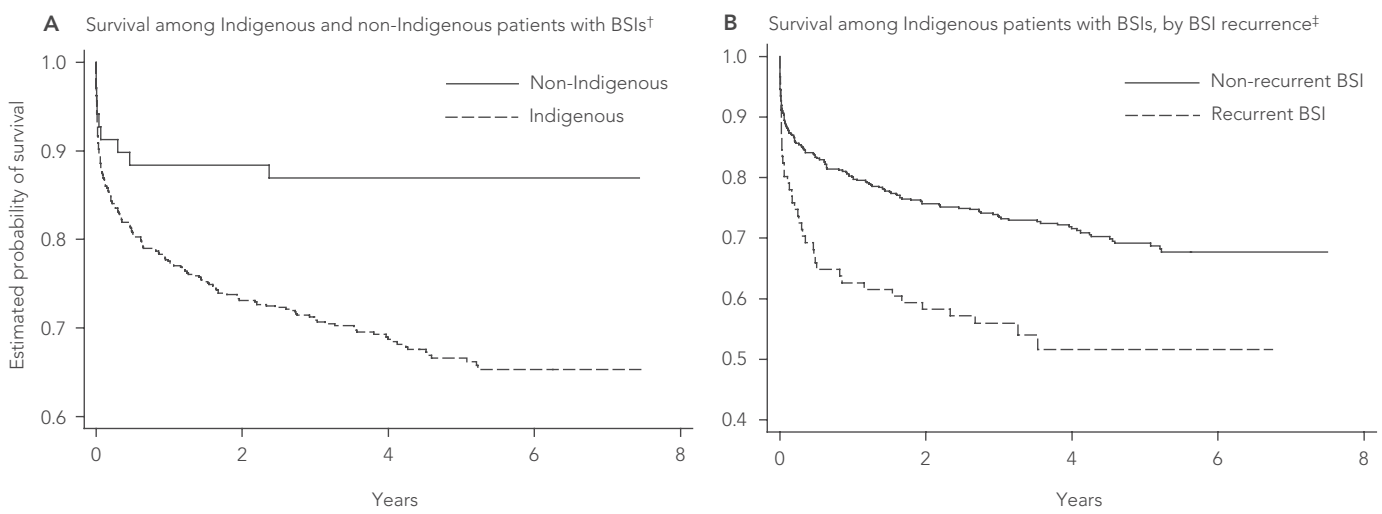
Pathogen	Indigenous patients ($n = 753$)†	Non-Indigenous patients ($n = 71$)‡	*P*
Gram-negative bacteria	384 (51.0%)	28 (39.4%)	0.08
Enterobacteriaceae	277 (72.1%)	22 (78.6%)	0.35
Escherichia coli	187 (67.5%)§	17 (77.3%)§	0.86
Klebsiella pneumoniae	50 (18.1%)§	0	0.025
Anaerobes	23 (6.0%)	2 (7.1%)	0.83
Enteric pathogens¶	19 (4.9%)	0	0.16
Pseudomonas spp	16 (4.2%)	4 (14.3%)	0.03
Haemophilus influenzae	15 (3.9%)	0	0.21
Neisseria spp**	11 (2.9%)	0	0.29
Other	10 (2.6%)	0	0.31
Acinetobacter baumannii	6 (1.6%)	0	0.43
Gram-positive bacteria	369 (49.2%)	44 (62.0%)	0.08
Staphylococcus aureus	147 (39.8%)	17 (38.6%)	0.4
Methicillin-resistant *S. aureus*	43 (29.3%)††	1 (5.9%)††	0.06
Streptococcus pneumoniae	110 (29.8%)	7 (15.9%)	0.18
β-haemolytic streptococci	59 (16.0%)	12 (27.3%)	0.02
Enterococcus spp	23 (6.2%)	4 (9.1%)	0.35
Streptococcus anginosus group	12 (3.3%)	2 (4.5%)	0.58
Other	12 (3.3%)	2 (4.5%)	0.58
Streptococcus bovis	6 (1.6%)	1 (2.3%)	0.64
Mycobacteria	1 (0.1%)	0	0.78
Mycobacterium mucogenicum	1 (0.1%)	0	0.78
Fungi	6 (0.8%)	2 (2.8%)	0.12
Cryptococcus neoformans	1 (0.1%)	0	0.78
Candida spp	5 (0.7%)	2 (2.8%)	0.08

* Figures are number (%) of isolates. † 91 organisms excluded as potential contaminants (viridans group streptococci, 30; *Acinetobacter* spp, 20). ‡ Eight organisms excluded as potential contaminants (viridans group streptococci, 3). § As a percentage of Enterobacteriaceae. ¶ *Salmonella*, *Campylobacter* and *Shigella* spp. ** *N. meningitidis* and *N. gonorrhoeae*. †† As a percentage of *S. aureus*. ◆

sepsis from these communities are heavily pretreated with antibiotics before retrieval, reducing the chance of isolating a pathogen at ASH. Some of the BC isolates excluded from our study, such as viridans group streptococci and *Acinetobacter* spp, may also have been true pathogens. We are therefore unlikely to have overestimated actual BSI rates in the Indigenous population.

Previously, attempts to improve public health in remote Indigenous communities have largely focused on providing infrastructure.[23] Inadequate housing and sanitation[22] undoubtedly facilitate pathogen transmission in these communities, and further improvements are imperative. However, despite attempts to improve infrastructure, BSI rates for the Indigenous population of central Australia remain higher than those of some developing countries. The failure to reduce rates of bacterial infection in Indigenous communities may be comparable to the situation in developing countries in which the provision of water and sanitation alone does not improve health outcomes, owing to multiple routes of disease transmission, poor hygiene and a heavily contaminated environment.[23,24] In such a setting, attempts to drive change externally by improving health infrastructure in isolation will fail.[24] Consistent with the previous recommendations of the National Aboriginal Health Strategy,[25] sustained improvement will require a coordinated approach[23,24] based on dialogue, cultural understanding and the develop-

5 Survival of adult patients after admission to Alice Springs Hospital with a BSI, 2001–2005*



BSI = bloodstream infection. * Follow-up was to 30 June 2008. † Hazard ratio (HR) (Indigenous v non-Indigenous), 2.69 (95% CI, 1.38–5.25); $P = 0.004$. ‡ HR (recurrent v non-recurrent BSI), 1.86 (95% CI, 1.32–2.62); $P < 0.001$. Recurrent BSI was defined as at least two separate BSI episodes caused by the same pathogen or different pathogens isolated more than 1 month apart. ◆

ment of locally specific solutions by Indigenous people themselves.

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COMPETING INTERESTS

None identified.

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Collaborative work by Mangkaja Arts, Fitzroy Crossing, WA *(see page 572)*