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

Lloyd J Einsiedel and Richard J Woodman

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.


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.

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

DEFINING THE GAP — RESEARCH
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 3). 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).

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) and exceed those of hospitals in Thailand (9.5/1000) and Vietnam (20.4/1000). 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) and consistent with population-based rates for all-cause sepsis (240–275/100 000 adult population per year) 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.\textsuperscript{18,19} Rates of diabetes mellitus among African Americans presenting with sepsis, for example, may approach those reported here,\textsuperscript{20} but this is associated with only a two-fold increase in sepsis risk relative to white Americans.\textsuperscript{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,\textsuperscript{21} nearly half these houses do not have functioning sanitation, and opportunities to maintain skin hygiene are limited.\textsuperscript{22} In such environments, exposure to bacterial pathogens and \textit{S. stercolaris} is likely, and HTLV-1 infection may further increase risk by predisposing people to complicated strongyloidiasis\textsuperscript{7} and crusted scabies.\textsuperscript{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

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

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

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).
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 sanitation22 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 approach23,24 based on dialogue, cultural understanding and the develop-
REFERENCES

Correspondence:

2 Department of General Practice, Flinders University, Alice Springs, NT.

Richard J Woodman, PhD, Infectious Diseases Physician

Lloyd J Einsiedel, PhD, FRACP, Infectious Diseases Physician

Richard J Woodman, PhD, Statistician

Department of General Practice, Flinders University, Alice Springs, NT.

ACKNOWLEDGEMENTS

We wish to thank Liselle Fernandes and Sheela Joseph for providing the comorbidity data. We received funding from the Northern Territory Rural Clinical School, an initiative of the Australian Department of Health and Ageing.

COMPETING INTERESTS

None identified.

AUTHOR DETAILS

Lloyd J Einsiedel, PhD, FRACP, Infectious Diseases Physician

Richard J Woodman, PhD, Statistician

1 Department of Medicine, Alice Springs Hospital, Alice Springs, NT.

2 Department of General Practice, Flinders University, Adelaide, SA.

Correspondence: lloyd.einsiedel@nt.gov.au

REFERENCES


(Received 19 Jun 2009, accepted 16 Nov 2009)