Staphylococcus aureus bacteraemia: a major cause of mortality in Australia and New Zealand

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on behalf of the Australia New Zealand Cooperative on Outcomes in Staphylococcal Sepsis

ABSTRACT

Objective: To document the types of, and mortality from, *Staphylococcus aureus* bacteraemia in Australia and New Zealand, and determine factors associated with mortality.

Design and setting: Prospective observational study in 27 independent or hospital pathology laboratories in Australia (24) and New Zealand (3), employing a web-based database to prospectively record demographic features, selected risk factors, principal antibiotic treatment and mortality data on all patients with positive blood cultures for *S. aureus* from June 2007 to May 2008.

Main outcome measure: 30-day all-cause mortality.

Results: 1994 episodes of *S. aureus* bacteraemia were identified, and complete 30-day follow-up data were available for 1865. Most episodes had their onset in the community (60.8%; 95% CI, 58.7%–63.0%). Methicillin-resistant *S. aureus* (MRSA) caused 450 episodes (24.1%; 95% CI, 22.2%–25.9%), and 123 of these (27.3%) had a susceptibility profile consistent with community-associated MRSA. All-cause mortality at 30 days was 20.6% (95% CI, 18.8%–22.5%). On univariate analysis, increased mortality was significantly associated with older age, European ethnicity, MRSA infection, infections not originating from a medical device, sepsis syndrome, pneumonia/empyema, and treatment with a glycopeptide or other non-β-lactam antibiotic. On multivariable analysis, independent predictors of mortality were age, sepsis syndrome, pneumonia/empyema, device-associated infection with a secondary focus, left-sided endocarditis, and treatment with a glycopeptide such as vancomycin, but not MRSA infection.

Conclusions: *S. aureus* bacteraemia is a common infection in both the community and hospitals in Australia and New Zealand, and is associated with appreciable mortality. Invasive MRSA infection may be more life-threatening, partly because of the inferior efficacy of the standard treatment, vancomycin. National web-based surveillance of *S. aureus* bacteraemia and its outcomes is not only important but also easily achievable.

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Staphylococcus aureus sepsis, especially that caused by methicillin-resistant *S. aureus* (MRSA), is widely recognised by the public and the general medical community as a problem associated with health care. Less well recognised, but equally important, is invasive *S. aureus* infection arising in the community. The emergence of community strains of MRSA has added significant concern about community-onset infections.[1] Although there is a wide variety of manifestations of serious invasive infections caused by *S. aureus*, in the great majority of these cases, the organism can be detected in blood cultures. Therefore, *S. aureus* bacteraemia is considered a very useful marker for these serious invasive infections,[2] and in some cases, is the only initial manifestation of the infection.

Published reports of experience around the world show that mortality from infections associated with *S. aureus* bacteraemia can range from as low as 2.5% to as high as 40%.[3-5] Mortality rates are known to vary significantly with patient age, clinical manifestation and comorbidities. Until recently, there have been no national data on the incidence and outcomes of *S. aureus* bacteraemia in either Australia or New Zealand. Small, largely retrospective, studies have been conducted, usually in single institutions. A recent prospective study of *S. aureus* bacteraemia conducted in 17 sites across Australia found a crude mortality rate of 11.2% when measured at discharge or 7 days, whichever came sooner.[6] An earlier study that documented outcomes in six major centres in New Zealand found an all-cause mortality of 22.4% and an attributable mortality of 18.9%.[7]

The Australia New Zealand Cooperative on Outcomes in Staphylococcal Sepsis (ANZCOSS) was established in 2007 to prospectively examine mortality from infections associated with *S. aureus* bacteraemia in a more structured way and to determine some of the risk factors for poor outcomes.[8]

METHODS

Independent or hospital pathology laboratories in Australia and New Zealand were asked to join the cooperative on a voluntary basis. Invitations were extended to all members of the Australian Group on Antimicrobial Resistance,[6] and by open invitation to selected hospital laboratories in New Zealand. The entry criterion for participating sites was a single blood culture that tested positive for *S. aureus*, associated with clinical manifestations in the patient consistent with staphylococcal infection. The date of study entry was the date of collection of the first positive culture in an episode of infection. A new episode in the same patient was recorded if the bacteraemia had cleared, but a further culture of blood taken more than 14 days after the initial positive culture was again positive.

Approval to conduct the prospective data collection was given by the research ethics committee associated with each participating laboratory. A web-based data entry system was constructed to enable real-time data collection into a common database. To ensure patient anonymity, but to allow follow-up of discrepant results with each participating site, a record identifier unique to the participating site was used. Data were collected on age, sex, ethnicity, date of admission (if admitted), date of discharge, relationship of the infection to a medical device and its type, the principal clinical manifestation of the infection, the principal agent used for definitive initial treatment (usually intravenous), and mortality at 7 and 30 days from date of entry. To avoid interpretive bias, no attempt was made to assign attributable mortality. Participating sites

1 Definitions of in-vitro susceptibility profiles for *Staphylococcus aureus*

Penicillin-susceptible *S. aureus* (PSSA)	Susceptible to benzylpenicillin
Methicillin-susceptible *S. aureus* (MSSA)	Resistant to benzylpenicillin but susceptible to methicillin (and therefore flucloxacillin)
Multiresistant methicillin-resistant *S. aureus* (multi-R MRSA)	Methicillin-resistant and resistant to more than two non-β-lactam antimicrobial classes
United Kingdom epidemic methicillin-resistant *S. aureus* type-15-like (UK EMRSA-15-like)	Methicillin-resistant and additionally resistant to ciprofloxacin ± erythromycin only
Non-multiresistant methicillin-resistant *S. aureus* (non-multi-R MRSA)	Methicillin-resistant and susceptible to two or more non-β-lactam antimicrobial classes (other than the EMRSA-15 pattern) ◆

were asked to assign the susceptibility type of *S. aureus* to one of five types based on their locally generated antibiotic profile (Box 1). The place of onset of *S. aureus* infection was designated as the community (if the first positive blood culture in an episode of infection was collected before or within 48 hours of hospital admission) or hospital (if the first positive blood culture was collected more than 48 hours after admission).

Although data collection commenced in June 2007, some sites were able to include data that had been collected prospectively from January 2007. Regular audits for data discrepancies and potential duplicate entries were conducted and resolved with each of the participating sites on a regular basis during data collection. Analysis was conducted on all cases of *S. aureus* bacteraemia for which data entries had been completed by early May 2008.

Data analysis

Descriptive statistics were extracted in Microsoft Excel 2003 (Microsoft Corporation, Redmond, Wash, USA). Completed cases with mortality data available at 30 days were subjected to formal statistical analysis. Univariate analysis was conducted using χ^2 and Fisher's exact tests. Multivariable logistic regression analysis using a mixed effects model was undertaken using R version 2.8.1 (R Foundation for Statistical Computing; http://www.r-project.org/foundation/). The site of each laboratory was treated as a random effect, while all other predictors were treated as fixed effects. Independent variables included in the multivariable analysis were those identified as potentially significant on univariate analysis ($P < 0.2$).

Percentage mortality was also compared for each participating site by using the funnel plot comparative assessment tool. This graphic quality assessment tool uses the average mortality rate for all cases combined, and constructs lines two SDs and three SDs either side of the average that are determined by the number of deaths and the total number of cases observed by each participating site.[9]

RESULTS

At the time of data analysis in May 2008, 27 sites had entered patient data; 24 from Australia and three from New Zealand. Data had been entered into the database for 2297 episodes of *S. aureus* bacteraemia. Of these, 1994 had complete data entry for all fields; 1860 from Australia and 134 from New Zealand. Through a process of audit and data scanning, and referral of data gaps and anomalies back to participating sites for resolution, the data were considered near complete and of high quality. Seven-day mortality data were available for all 1994 completed cases, and 30-day mortality data were available for 1865 (93.5%). The all-cause mortality was 10.8% (95% CI, 9.4%–12.1%) at 7 days and 20.6% (95% CI, 18.8%–22.5%) at 30 days.

Patient and infection features

Among all 1994 cases with complete data, 1273 of the patients were male (63.8%; 95% CI, 61.7%–66.0%), more than a third were older than 70 years (36.6%; 95% CI, 34.5%–38.7%) and 84.1% were of European ethnicity (95% CI, 82.5%–85.7%). There was a reasonably representative spread of cases across the Australian states when compared with the population distribution, with Victoria slightly under-represented. The smaller number of cases from New Zealand involved patients almost exclusively from Auckland.

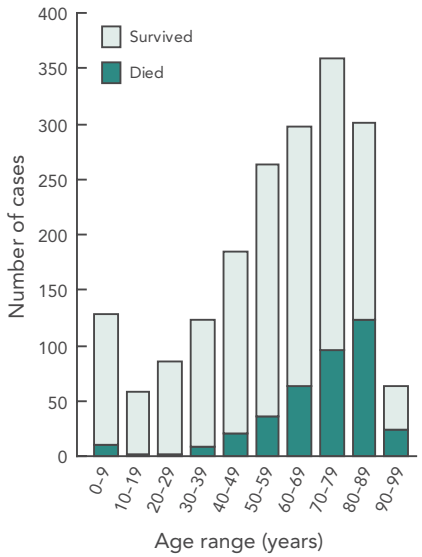
Most episodes of *S. aureus* bacteraemia had their onset in the community (60.8%; 95% CI, 58.7%–63.0%). A small proportion

2 Univariate analysis of demographic characteristics versus 30-day mortality for 1865 patients with *Staphylococcus aureus* bacteraemia

Demographic characteristic	Number	30-day all-cause mortality	*P**
Age			
⩽ 70 years	688 (36.9%)	12.9%	< 0.001
> 70 years	1177 (63.1%)	33.9%	
Sex			
Male	1194 (64.0%)	21.0%	0.633
Female	671 (36.0%)	20.0%	
Ethnicity			
European	1575 (84.5%)	22.2%	< 0.001
ATSI†	53 (2.8%)	5.7%	
Maori	31 (1.7%)	9.7%	
Other	206 (11.0%)	14.6%	
Jurisdiction			
New South Wales	643 (34.5%)	22.1%	
Queensland	317 (17.0%)	16.4%	
Victoria	272 (14.6%)	22.4%	
South Australia	197 (10.6%)	25.4%	
Western Australia	194 (10.4%)	19.1%	
New Zealand	132 (7.1%)	17.4%	
ACT, NT and Tasmania‡	110 (5.9%)	18.2%	
Participating sites ($n = 27$)			0.004

* By Pearson's χ^2 or Fisher's exact test. † Australian Aboriginal or Torres Strait Islander. ‡ Australian Capital Territory, Northern Territory and Tasmania combined. ◆

3 Number of cases of *Staphylococcus aureus* bacteraemia and patient survival, by age*



* Data not shown for one patient aged 100 years, who survived. ◆

of patients with community-onset *S. aureus* bacteraemia (6.3%) were not admitted to hospital, many being managed in home intravenous-therapy programs. Infection types conformed to recognised incidences; device-related infection without a metastatic focus was the most common infection type seen. Device-related infections accounted for 657 of all 1840 infections where information about the relationship to a device was known (35.7%; 95% CI, 33.5%–38.0%). A central venous line was the most common source, with peripheral lines and haemodialysis access contributing significant proportions.

Methicillin-resistant strains of *S. aureus* were responsible for 450 episodes of bacteraemia (24.1%; 95% CI, 22.2%–25.9%). Multiresistant methicillin-resistant *S. aureus* (multi-R MRSA), the classical health-care associated phenotype, accounted for 61.3% (95% CI, 56.9%–65.6%) of these episodes, while non-multiresistant methicillin-resistant *S. aureus* (non-multi-R MRSA), the phenotype typical of community-acquired MRSA strains, accounted for 27.5% (95% CI, 23.5%–31.5%) of episodes. The remainder had the phenotype commonly associated with the introduced United Kingdom epidemic methicillin-resistant *S. aureus* type-15 clone. MRSA strains accounted for 17.8% (95% CI, 15.7%–20.0%) of community-onset infections, and 33.8% (95% CI, 30.5%–37.1%) of hospital-onset infections.

4 Univariate analysis of features of *Staphylococcus aureus* bacteraemia versus 30-day mortality in 1865 patients

Feature of *S. aureus* bacteraemia	Number	30-day all-cause mortality	*P**
Hospital admission			
Yes	1774 (95.1%)	20.6%	1.0
No	91 (4.9%)	20.9%	
***S. aureus* susceptibility†**			
PSSA	188 (10.1%)	21.8%	< 0.001
MSSA	1227 (65.8%)	17.0%	
Multi-R MRSA	278 (14.9%)	33.1%	
UK EMRSA-15-like	49 (2.6%)	30.6%	
Non-multi-R MRSA	123 (6.6%)	22.8%	
***S. aureus* methicillin susceptibility**			
Susceptible	1415 (75.9%)	17.7%	< 0.001
Resistant	450 (24.1%)	30.0%	
Place of onset of infection			
Hospital	1118 (59.9%)	23.9%	0.004
Community	747 (40.1%)	18.4%	
Device-related infection			
Yes	628 (33.7%)	16.7%	0.011
No	1096 (58.8%)	22.4%	
Not recorded	141 (7.6%)	24.1%	
Device type (if device-related)			
Central venous line	244 (13.1%)	20.5%	0.003
Peripheral line	142 (7.6%)	16.9%	
Haemodialysis access	117 (6.3%)	6.8%	
Orthopaedic	36 (1.9%)	8.3%	
Other	89 (4.8%)	22.5%	
Principal clinical manifestation			
Device without a metastatic focus	344 (18.4%)	12.8%	< 0.001
Skin and skin structure infection	317 (17.0%)	14.8%	
No focus found	242 (13.0%)	21.9%	
Osteoarticular infection‡	239 (12.8%)	11.7%	
Sepsis syndrome	201 (10.8%)	40.3%	
Pneumonia/empyema	145 (7.8%)	42.1%	
Left-sided endocarditis	67 (3.6%)	23.9%	
Right-sided endocarditis	51 (2.7%)	11.8%	
Deep abscess	48 (2.6%)	14.6%	
Device with a metastatic focus	46 (2.5%)	32.6%	
Central nervous system infection	28 (1.5%)	10.7%	
Other	137 (7.3%)	17.5%	
Treatment			
β-lactam	1151 (61.7%)	13.9%	
Glycopeptide	518 (27.8%)	25.7%	
None	73 (3.9%)	76.7%	
Other	123 (6.6%)	29.3%	< 0.001

PSSA = penicillin-susceptible *S. aureus*. MSSA = methicillin-susceptible *S. aureus*. Multi-R MRSA = multiresistant methicillin-resistant *S. aureus*. UK EMRSA-15-like = United Kingdom epidemic methicillin-resistant *S. aureus* type-15-like. Non-multi-R MRSA = non-multiresistant methicillin-resistant *S. aureus*.

* By Pearson's χ^2 or Fisher's exact test. † See Box 1 for definitions of susceptibility types. ‡ Includes osteomyelitis, septic arthritis and discitis. ◆

5 Multivariable analysis of risk factors for 30-day all-cause mortality for patients with *Staphylococcus aureus* bacteraemia

Risk factor	*P*	Odds ratio	95% CI
Age	< 0.001		
Age squared	< 0.001		
Ethnicity			
European	—	1	
ATSI*	0.226	0.455	0.127–1.628
Maori	0.766	0.814	0.210–3.156
Other	0.701	0.913	0.573–1.453
***S. aureus* susceptibility**†			
PSSA	—	1	
MSSA	0.219	0.767	0.501–1.171
Non-multi-R MRSA	0.413	0.753	0.381–1.484
Multi-R MRSA	0.899	1.039	0.578–1.864
UK EMRSA-15-like	0.513	0.754	0.323–1.758
Place of onset of infection			
Hospital	—	1	
Community	0.012	0.695	0.323–1.758
Device-related infection			
Yes	—	1	
No	0.045	0.649	0.425–0.990
Not recorded	0.230	0.736	0.445–1.215
Principal clinical manifestation			
Osteoarticular infection‡	—	1	
Central nervous system infection	0.866	1.117	0.310–4.013
Deep abscess(es) (excluding central nervous system abscesses)	0.366	1.594	0.617–4.117
Device infection with secondary focus	0.011	2.997	1.280–7.012
Device infection without secondary focus	0.602	1.186	0.624–2.251
Left-sided endocarditis	0.006	2.801	1.340–5.853
Right-sided endocarditis	0.575	1.334	0.487–3.649
No focus found	0.601	1.159	0.666–2.015
Other	0.676	1.154	0.590–2.255
Pneumonia/empyema	< 0.001	3.890	2.207–6.853
Sepsis syndrome	< 0.001	4.011	2.339–6.874
Skin and skin structure infection	0.904	0.968	0.565–1.655
Treatment			
β-lactam	—	1	
Glycopeptide	0.020	1.641	1.082–2.489
None	< 0.001	19.679	9.884–37.170
Other	0.008	1.984	1.200–3.280

PSSA = penicillin-susceptible *S. aureus*. MSSA = methicillin-susceptible *S. aureus*. Non-multi-R MRSA = non-multiresistant methicillin-resistant *S. aureus*. Multi-R MRSA = multiresistant methicillin-resistant *S. aureus*. UK EMRSA-15-like = United Kingdom epidemic methicillin-resistant *S. aureus* type-15-like.
* Australian Aboriginal or Torres Strait Islander. † See Box 1 for definitions of susceptibility types.
‡ Includes osteomyelitis, septic arthritis and discitis. ◆

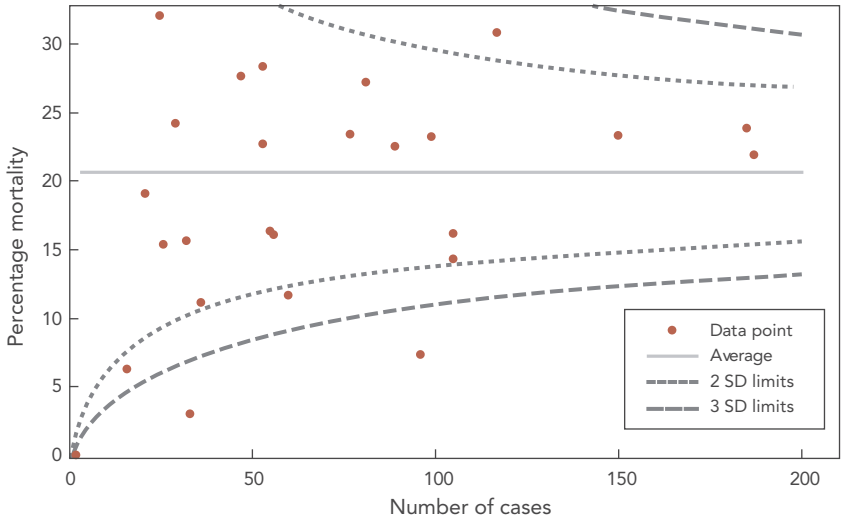
While most of the 1514 patients with methicillin-susceptible strains were treated with flucloxacillin (65.0%), almost 10% were treated with the glycopeptide vancomycin (9.7%); the remainder were treated with other β-lactams (14.8%) or other drug classes (6.6%), or were not treated for a range of reasons (3.7%), such as terminal underlying illness. Vancomycin was the most widely used agent for MRSA infections (81.0%). The other available glycopeptide, teicoplanin, was used in only 1.0% of cases. Other MRSA infections were treated with a range of other agents, including lincomycin or clindamycin (4.6%), while 6.0% were not treated. Among the 1865 patients for whom data were available, there was a 30-day mortality rate of 22.1% (95% CI, 15.3%–29.0%) in the 140 patients with methicillin-susceptible strains who were treated with vancomycin, compared with 13.2% (95% CI, 11.0%–15.4%; $P = 0.005$) mortality among the 932 treated with flucloxacillin.

Univariate and multivariable analyses

Univariate and multivariable analyses were performed on the 1865 cases for which 30-day all-cause mortality data were available. In the univariate analysis, demographic risk factors for increased mortality (Box 2) were age over 70 years and European ethnicity. As shown in Box 3, age-related mortality was a striking feature. Other significant factors on univariate analysis were some participating sites (Box 2); MRSA infection (other than non-multi-R MRSA); hospital-onset infection; infection that did not originate from a medical device; the clinical manifestations of sepsis syndrome, pneumonia/empyema and device infection with a metastatic focus; and treatment with a glycopeptide or other non-β-lactam (either for methicillin-susceptible *S. aureus* or MRSA infection) (Box 4).

Multivariable logistic regression using a mixed effects model showed that, of the significant risk factors identified in the univariate analysis, only age, place of onset of infection, a diagnosis of device infection with a secondary focus, principal clinical manifestation and treatment remained in the model (Box 5). Age was the strongest predictor of higher mortality, and both linear and quadratic terms in age were used to model the parabolic relationship between the variables. Pneumonia/empyema and sepsis syndrome were associated with higher mortality. Notably, we did not find evidence that infection with MRSA was an independent predictor of higher mortality. By contrast, glycopeptide treatment was predictive of higher mortality, with an odds ratio of 1.6 (95% CI, 1.1–2.7). Device-related infection and device type, when causing infection, were not independent predictors of reduced mortality in the multivariable analysis.

6 Funnel plot of 30-day percentage mortality from *Staphylococcus aureus* bacteraemia for patients identified by the 27 participating institutions



Each data point represents the percentage mortality at a single institution, ordered on the *x* axis by the number of cases observed. The straight line represents the overall average 30-day mortality. The inner curves represent two SDs either side of overall average 30-day mortality, while the outer curves represent three SDs either side of the overall average. The SD (confidence) limits are wide when few cases are observed, and narrow with increasing numbers of cases. The plot allows each participating institution to compare its mortality rate against the overall average and with institutions with similar numbers of cases. ◆

Comparative assessment

There was wide variation in mortality rates between participating sites. The results of the funnel plot comparison are shown in Box 6. Four participating sites had 30-day mortality rates below the 2 SD limit. Two sites had 30-day mortality rates below the 3 SD limit; for one of these, the finding could readily be attributed to age profile. One site had a 30-day mortality rate above the 2 SD limit. Investigation revealed a small spike of mortality attributable to late patient transfer to their site, while the subsequent mortality rate was close to the overall average.

DISCUSSION

This study provides the first reliable national estimate of percentage mortality from *S. aureus* bacteraemia for Australia, and provides indicative mortality for New Zealand. That so many institutions were willing to participate in the cooperative with no additional resources indicates the importance that those in the medical community attach to this problem. For Australia at least, we estimate that we have captured between a third and half of all cases over the period of data collection (about 1 year on average), based on previous estimates of the national incidence in Australia of around 6500 cases.[5] The average 30-day mortality rate of around 20% is more than double that of bacterial diseases with a much higher public health profile, such as invasive meningococcal disease, and its incidence in the community is also substantially higher.[10]

In conducting this surveillance, we aimed to make data entry as simple and reliable as possible. To these ends, we chose web-based data entry because of its ready accessibility and consistency, and we kept the number of fields requiring data low. Further, we attempted to collect easily accessible data that required only a small additional effort above normal clinical activities, and to avoid, as far as possible, collecting data that required subjective interpretation. In particular, we did not ask participating sites to provide attributable mortality, which, in many respects, is a subjective judgement, particularly in cases with complex comorbidities. Some studies have estimated that attributable mortality is around 65% of crude mortality at 30 days.[11] As an indication, the 7-day mortality rate of nearly 11% would almost all be attributable to staphylococcal sepsis.

Risk factors for mortality were readily identified in the univariate and multivariable analyses. As in most previous studies, age was the most important determinant of poor outcome, with increasing mortality with each decade. Noticeable, however, was the higher mortality in the first decade of life compared with the second and third decades. Some, but not all of these deaths, occurred in the first few months of life, presumably in hospitalised neonates. The findings of significantly increased mortality with left-sided endocarditis, device infection with a secondary focus, pneumonia/empyema and sepsis syndrome are also not unexpected.

More surprising is the fact that MRSA infection was not an independent risk factor for increased mortality in the multivariable analysis. A number of studies and meta-analyses have shown that MRSA is associated with poorer outcomes, including increased mortality.[12] Our analysis suggests that this may be attributed to the antimicrobial agent used for treatment, almost always vancomycin. The likely reason that we obtained this result is that sufficient patients infected with methicillin-susceptible strains were treated with vancomycin instead of a β-lactam, and this allowed us to demonstrate a higher 30-day mortality with vancomycin than with flucloxacillin treatment. There has been increasing evidence of the inferior efficacy of vancomycin in treating staphylococcal infection,[13] including recent compelling evidence of its inferiority to β-lactam treatment in treating methicillin-susceptible *S. aureus* infection.[14] So, it may be better to consider MRSA as a major threat mostly because the drug of choice for serious invasive infection is less effective than β-lactams are against methicillin-susceptible strains. Unfortunately, new agents for treating serious MRSA infection, such as linezolid and daptomycin, have so far not shown predictable superiority over vancomycin in prospective randomised controlled trials.[13] We have begun a retrospective review to determine why vancomycin does not perform as well as should be expected. Factors to consider include the pharmacokinetics and pharmacodynamics of the drug and the emergence of reduced susceptibility, especially of the heterogeneous type, in some strains of *S. aureus*.

A limitation of our study was that no data were collected on comorbidities. These have been shown to influence mortality significantly.[11] Data such as these require more intensive efforts at collection, which we did not regard as conducive to recruitment or compliance with data collection. Moreover, the studies that have examined comorbidities have commonly used non-discriminatory measures, such as the Charlson Comorbidity Index, many components of

which have no plausible link to poor outcomes for serious staphylococcal infection. Further studies are required to determine which aspects of comorbidities predict poor outcomes. It is possible that differences in comorbidities may have accounted in part for the differences in mortality rates we observed between participating sites.

Clearly, invasive *S. aureus* infection is a major disease in Australia and New Zealand, with substantial mortality. The substantial proportion of infections that are caused by MRSA, and the suboptimal antimicrobial agents available for MRSA treatment exacerbate the problem. There has been a perception that serious staphylococcal infection, especially that caused by MRSA, is a problem largely confined to hospitals. However, our data show that it is also a major problem in the community, where methicillin-susceptible strains are a significant cause of morbidity and mortality, and where non-multiresistant strains of MRSA are now adding to the burden of disease in a substantial way. Continuous surveillance of *S. aureus* bacteraemia and its outcomes should be a priority in Australia and New Zealand, so that change can be driven, interventions designed and implemented, and their effects measured.

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COMPETING INTERESTS

None identified.

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