Prevalence of MRSA strains among *Staphylococcus aureus* isolated from outpatients, 2006

Report from the Australian Group for Antimicrobial Resistance

Geoffrey W Coombs, Graeme R Nimmo, Julie C Pearson, Keryn J Christiansen, Jan M Bell, Peter J Collignon, Mary-Louise McLaws and the Australian Group for Antimicrobial Resistance

Abstract

Biennial community-based *Staphylococcus aureus* antimicrobial surveillance programs have been performed by the Australian Group for Antimicrobial Resistance (AGAR) since 2000. Over this time the percentage of *S. aureus* identified as methicillin resistant has increased significantly from 10.3% in 2000 to 16% in 2006. This increase has occurred throughout Australia and has been due to the emergence of community-associated MRSA (CA-MRSA) clones. However, healthcare associated MRSA were still predominant in New South Wales/Australian Capital Territory and Victoria/Tasmania. In the 2006 survey CA-MRSA accounted for 8.8% of community-onset *S. aureus* infections. Although multiple CA-MRSA clones were characterised, the predominate clone identified was Queensland (Qld) MRSA (ST93-MRSA-IV) a Panton-Valentine leukocidin (PVL) positive MRSA that was first reported in Queensland and northern New South Wales in 2003 but has now spread throughout Australia. Several international PVL-positive CA-MRSA clones were also identified including USA300 MRSA (ST8-MRSA-IV). In addition, PVL was detected in an EMRSA-15 (ST22-MRSA-IV) isolate; a hospital associated MRSA clone that is known to be highly transmissible in the healthcare setting. With the introduction of the international clones and the transmission of Qld MRSA throughout the country, over 50% of CA-MRSA in Australia are now PVL positive. This change in the epidemiology of CA-MRSA in the Australian community will potentially result in an increase in skin and soft tissue infections in young Australians. As infections caused by these strains frequently results in hospitalisation their emergence is a major health concern. *Commun Dis Intell* 2009;33:10–20.

Keywords: *Staphylococcus aureus*, MRSA, healthcare-acquired infection, antimicrobial resistance, epidemiology

Introduction

The emergence of methicillin-resistant *Staphylococcus aureus* (MRSA) causing community-onset infections has represented a major change in the epidemiology of *S. aureus*. Community-onset MRSA (CO-MRSA) is a worldwide phenomenon with epidemics reported in many regions including Canada,[1] the United States of America[2] and Europe.[3] These reports have a number of findings in common including: lack of association with risk factors for healthcare-associated acquisition of MRSA; lack of resistance to non-β-lactam antibiotics; frequent association with Indigenous populations; and association with subcutaneous abscess formation and necrotising pneumonia.[3] The latter clinical conditions have been shown to correlate strongly with possession of the genes for Panton-Valentine leukocidin (PVL)+, an extracellular toxin that destroys leucocytes and causes tissue necrosis. Furthermore, unlike hospital-onset MRSA (HO-MRSA) epidemics, which are due to a relatively small number of MRSA clones, CO-MRSA epidemics are polyclonal. Although global transmission of some community-associated MRSA (CA-MRSA) clones has occurred, most clones are believed to have evolved independently of one another with little or no evidence of global transmission.

Australia has had a unique experience with CO-MRSA in that the first epidemic in Western Australia was documented earlier than in most countries.[5,6] It was due initially to a PVL-negative clone and subsequently to a great variety of clones, including some that are PVL-positive.[7] Epidemics initially developed quite separately with distinct clones in different parts of the country. This might be expected in a country with relatively few dense concentrations of population separated by large areas, often desert, with very sparse population.

The Australian Group for Antimicrobial Resistance (AGAR) has previously established that the major CA-MRSA clones circulating the community were WA MRSA-1 (ST1-MRSA-IV), the 'south-west Pacific' (SWP) clone (ST30-MRSA-IV), and the Qld clone (ST93-MRSA-IV), which were widely dispersed geographically.[8] Both the SWP and Qld clones usually carry PVL genes and are associated with abscess formation, bacteraemia and necrotising pneumonia.[9]

In this paper we report the prevalence of antimicrobial resistance in clinical isolates of *S. aureus* throughout Australia in an outpatient population, and describe changes in prevalence and geographic range of MRSA clones and the extent of PVL gene carriage in these strains.

Methods

Thirty laboratories from all 6 states, the Australian Capital Territory and the Northern Territory participated in the survey. Commencing on 10 June 2006, each laboratory collected up to 100 consecutive significant clinical isolates from patients attending primary care clinics, outpatient clinics, emergency departments or other outpatient settings, or residing in long-term care facilities. Dialysis and day surgery patients were excluded. Only 1 isolate per patient was tested and no isolates from screening swabs or from specimens received for the purpose of gathering surveillance data were included.

Species identification

S. aureus was identified by morphology and positive results of at least two of 3 tests: slide coagulase test, tube coagulase test, and demonstration of deoxyribonuclease production.[10] Additional tests such as fermentation of mannitol or growth on mannitol-salt agar may have been performed for confirmation.

Susceptibility testing methodology

Participating laboratories performed antimicrobial susceptibility tests using the Vitek2® AST-P545 card (BioMerieux, Durham, NC). Antimicrobials tested were benzylpenicillin, oxacillin, cefazolin, vancomycin, rifampicin, fusidic acid, gentamicin, erythromycin, clindamycin, tetracycline, trimethoprim/sulphamethoxazole (cotrimoxazole), ciprofloxacin, quinupristin/dalfopristin (Synercid®), teicoplanin, linezolid, imipenem, and nitrofurantoin. Results were interpreted for non-susceptibility according to Clinical and Laboratory Standards Institute (CLSI) breakpoints.[11] Penicillin susceptible strains were tested for β-lactamase production using nitrocefin. A cefoxitin disc diffusion test was used to confirm methicillin-resistance. Mupirocin and cefoxitin were tested by disc diffusion using the CLSI or calibrated dichotomous sensitivity (CDS) methods.[11,12] The tigecycline minimum inhibitory concentration of all isolates was determined by Etest® (AB Biodisk, Solna, Sweden).

Characterisation of methicillin-resistant *Staphylococcus aureus*

Resistogram typing was performed by disk diffusion against a panel of 6 chemicals and dyes as previously described.[13,14] Coagulase gene restriction fragment length polymorphism typing was performed as described elsewhere.[15] Pulsed-field gel electrophoresis (PFGE) of chromosomal DNA was performed using the CHEF DRIII System (Bio-Rad Laboratories, Sydney, NSW) and interpreted as described elsewhere.[16,17] Representative isolates were characterised by multilocus sequence typing (MLST) and staphylococcal chromosomal cassette *mec* (SCC*mec*) typing with results interpreted as described previously.[8,18,19]

Clones are reported with their common names (e.g. WA MRSA-I) followed by the sequence type (ST), methicillin resistance phenotype, and SCC*mec* type (I to V) (e.g. ST1-MRSA-IV). Clones are classified into 2 groups on the basis of previously published evidence: those implicated in healthcare-associated infection and those implicated in community-associated infection.

MRSA isolates were assayed for the presence of PVL genes using polymerase chain reaction (PCR) primers for a 1554-bp region from the *lukS-PV* and *lukF-PV* genes.[20]

Statistical analysis

The proportions and 95% confidence intervals (CI) were calculated for MRSA by laboratory, state or territory, age, source, invasiveness of infection (blood, sterile site or cerebrospinal fluid isolates) and antibiogram. Odds ratio for the association of age and MRSA was examined after age of patient was categorised into one of 5 age groups. All descriptive and inferential statistics were calculated using Epi Info version 6.0.4 (Centers for Disease Control and Prevention, Atlanta, Georgia, USA) with the alpha level set at the 5% level for 2-sided tests for significance.

Results

Participating laboratories (26 public and 4 private) were located in New South Wales (8), the Australian Capital Territory (1), Queensland (5), Victoria (6), Tasmania (2), the Northern Territory (1), South Australia (3) and Western Australia (4). To ensure institutional anonymity data have been combined for New South Wales and the Australian Capital Territory, Victoria and Tasmania and for Queensland and the Northern Territory (Table 1). There were 2,979 isolates included in the survey with the majority (76.7%) of isolates contributed by New South Wales/Australian Capital Territory (30.0%), Victoria/Tasmania (26.5%) and Queensland/Northern Territory (20.1%).

Specimen source

Skin and soft tissue infections (SSTI) specimens contributed the majority (81.0%, 95% CI 79.6–

Table 1. Number of institutions and *Staphylococcus aureus* isolates collected in state or territory

Region	Number of institutions	Total	%
New South Wales/ Australian Capital Territory	9	895	30.0
Queensland/ Northern Territory	6	600	20.1
South Australia	3	299	10.0
Victoria/Tasmania	8	788	26.5
Western Australia	4	397	13.3
Total	30	2,979	100.0

Table 2. Number and proportion of isolates associated with specimen types (where known)

Specimen source	Number	% (95% CI)
Skin and soft tissue	2,414	81.0 (79.6–82.4)
Respiratory	177	5.9 (5.1–6.9)
Ear	109	3.7 (3.0–4.4)
Blood	106	3.6 (2.9–4.3)
Urine	96	3.2 (2.6–3.9)
Eye	48	1.6 (1.2–2.1)
Sterile site	28	0.9 (0.96–1.4)
Total	2,978	
Invasive	134	4.5 (3.8–5.3)
Non-invasive	2,844	95.5 (94.7–96.2)

CI – confidence interval

82.4%) of isolates followed by respiratory specimens (5.9%, 95% CI 5.1%–6.9%) while blood culture isolates contributed only 3.6% (95% CI 2.9%–4.3%) of the total with significantly ($P<0.0001$) more isolates causing non-invasive (95.5%) than invasive (4.5%) infections (Table 2).

Susceptibility and typing results

The proportion of MRSA was 16.0% (95% CI 14.7%–17.3%) nationally (Table 3), which was not significantly different from the proportion identified in 2004 (15.3%) ($P = 0.55$). At a regional level the proportions of MRSA identified in 2004 and 2006 were stable in New South Wales/Australian Capital Territory (19.8% in 2004 to 23.0%, NS), South Australia (10.3% to 12.0%, NS), Victoria/Tasmania (10.7% to 12.7%, NS) and Western Australia (13.0% to 11.3%, NS), while Queensland/Northern Territory showed a significant decrease (19.8%–14.8%, $P = 0.0494$).[9] The proportion of invasive isolates (blood/sterile sites) that were MRSA was 10.4% overall and did not vary significantly ($P = 0.6563$) between regions. Urinary isolates included a significantly ($P<0.0001$, $X^2=42.59$) greater proportion of MRSA (33.3%, 95% CI 24.2%–43.8%) than any other specimen types (Table 4).

Of the 476 *S. aureus* identified as MRSA, 462 were referred to the WA Gram-positive Bacteria Typing and Research Unit for epidemiological typing.

The proportion of MRSA that were healthcare-associated MRSA (HA-MRSA) clones varied markedly between regions, ranging from 11.4% in Western Australia to 57.1% in Victoria/Tasmania ($P<0.0001$) (Figure 1). More than half of all MRSA in Victoria/Tasmania (56/98, 57.1%) and New South Wales/Australian Capital Territory (110/198, 55.6%) were HA-MRSA, whereas a quarter or less were HA-MRSA in South Australia (9/36, 25.0%), Queensland/Northern Territory (20/86, 23.3%) and Western Australia (5/44, 11.4%). Of HA-MRSA, Eastern Australian (EA)-MRSA (ST239-MRSA-III) predominated in the eastern regions ranging from 52.7% in New South Wales/Australian Capital Territory to

Table 3. Proportion of *Staphylococcus aureus* that are methicillin-resistant, by region and source

	% (95%Confidence interval) (n/N)						Difference across regions X^2 P
	NSW/ACT	Qld/NT	SA	Vic/Tas	WA	Aus	
All	23.0 (20.3–25.9) (206/895)	14.8 (12.1–18.0) (89/600)	12.0 (8.7–16.4) (36/299)	12.7 (10.5–15.3) (100/788)	11.3 (8.5–15.0) (45/397)	16.0 (14.7–17.3) (476/2,979)	49.79 <0.0001
Invasive	15.4 (5.0–35.7)| (4/26)	12.5 (4.1–29.9) (4/32)	0.0 (0.0–40.2) (0/8)	10.9 (4.5–22.9) (6/55)	0.0 (0.0–28.3) (0/13)	10.4 (6.0–17.2) (14/134)	3.284 0.6563
Non–invasive	23.2 (20.5–26.2) (202/869)	15.0 (12.2–18.2) (85/568)	12.4 (8.9–16.8) (36/291)	12.8 (10.5–15.5) (94/733)	11.5 (8.6–15.2) (44/383)	16.2 (14.9–17.6) (461/2,844)	47.95 <0.0001

X2 – chi-square *P* – probability

80.0% in Queensland/Northern Territory but was not isolated in Western Australia. Queensland/Northern Territory had the lowest proportion (4/20, 20.0%) of EMRSA-15 (ST22-MRSA-IV).

Table 4. Proportion of *Staphylococcus aureus* that are methicillin-resistant, by source (where known)

Specimen source	% MRSA	95% CI
Skin and soft tissue	16.0 (387/2,414)	14.6–17.6
Respiratory	19.8 (35/177)	14.3–26.6
Ear	3.7 (4/109)	1.2–9.7
Blood	11.3 (12/106)	6.2–19.3
Urine	33.3 (32/96)	24.2–43.8
Eye	6.3 (3/48)	1.5–17.5
Sterile site	7.1 (2/28)	1.9–23.7

CI – confidence interval

Among the CA-MRSA strains, the Qld clone (ST93-MRSA-IV) was predominant in New South Wales/Australian Capital Territory (49/88, 55.7%) and Queensland/Northern Territory (20/66, 30.3%), while Western Australia MRSA-1 (ST1-MRSA-IV) accounted for approximately half of the isolates in Western Australia (19/39, 48.7%) and South Australia (15/27, 55.6%) (Figure 2). Victoria/Tasmania had the greatest diversity of community clones and was unique in having a large proportion (19/39, 21.4%) of the Victorian MRSA clone (ST45-MRSA-IV).

Resistance in *S. aureus* to non-β-lactam antimicrobials with the exception of rifampicin and fusidic acid varied significantly between states (Table 5). Resistance to gentamicin, tetracycline and trimethoprim/sulphamethoxazole was highest in New South Wales/Australian Capital Territory, Victoria/Tasmania, and Queensland/Northern Territory, reflecting the higher proportion of MRSA isolates in these regions that were EA-MRSA (Figure 1).

Resistance to non-β-lactam antimicrobials in MRSA clones with more than 30 isolates is shown in Table 6. EA-MRSA had very high levels of resistance to erythromycin, cotrimoxazole, tetracycline, ciprofloxacin and gentamicin, but low levels of

Figure 1. Proportion of healthcare associated methicillin-resistant *Staphylococcus aureus* clones, by regions

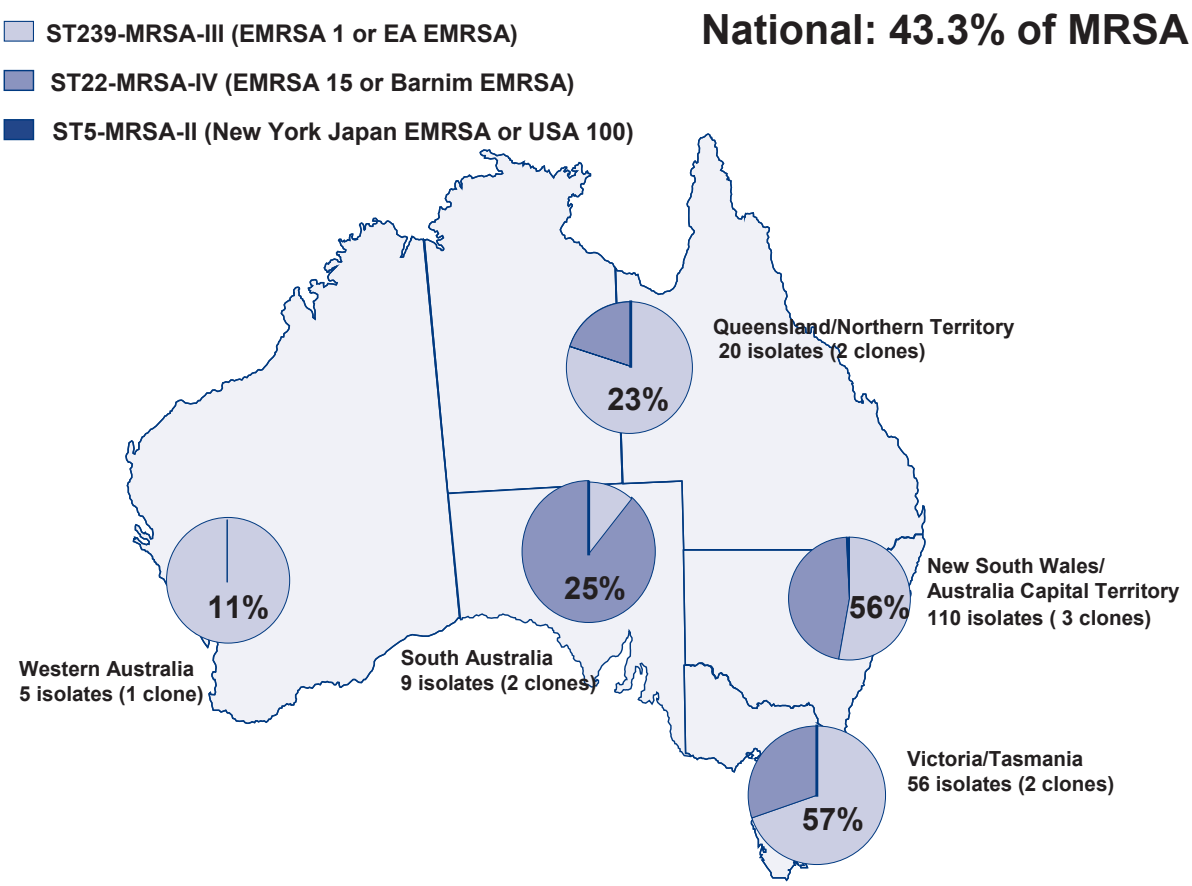


Figure 2. Proportion of community associated methicillin-resistant *Staphylococcus aureus* clones, by region

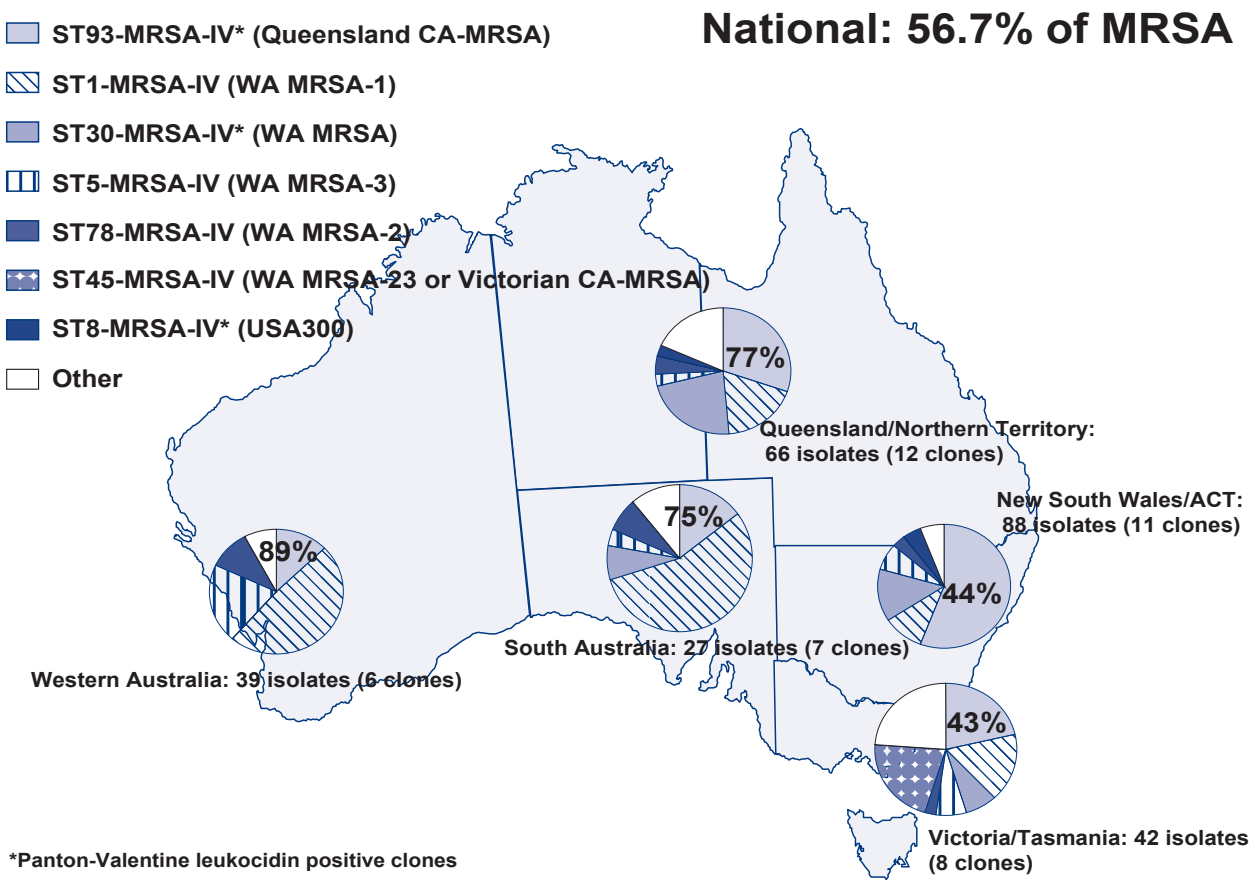


Table 5. Proportion (and number) of *Staphylococcus aureus* non-susceptible to non-β-lactams

Drug	NSW/ACT		Qld/NT		SA		Vic/Tas		WA		Aus		Difference across regions	
	%	n	%	n	%	n	%	n	%	n	%	n	X^2	*P*
Total isolates	895		600		299		788		397		2,979			
Erythromycin	20.1	180	16.3	98	16.7	50	16.4	129	13.1	52	17.1	509	10.79	0.0557
Tetracycline	10.4	93	5.5	33	2.0	6	8.9	70	3.3	13	7.2	215	40.71	<0.0001
Trimethoprim-sulphamethoxazole	8.9	80	3.8	23	1.3	4	7.4	58	0.8	3	5.6	168	54.56	<0.0001
Ciprofloxacin	14.6	131	4.5	27	4.3	13	10.2	80	2.0	8	8.7	259	84.65	<0.0001
Gentamicin	7.6	68	4.3	26	0.7	2	5.8	46	0.3	1	4.8	143	46.61	<0.0001
Fusidic acid	3.5	31	5.8	35	4.3	13	4.3	34	4.5	18	4.4	131	4.831	0.4369
Mupirocin	1.3	12	3.3	20	0.7	2	1.1	9	2.3	9	1.7	52	14.00	0.0156
Rifampicin	1.0	9	1.2	7	0.0	0	0.4	3	0.0	0	0.6	19	9.848	0.0797

X^2 – chi-square *P* - probability

resistance to rifampicin, fusidic acid and mupirocin. For EMRSA-15 almost all isolates were ciprofloxacin resistant and approximately half were erythromycin resistant, while resistance to other agents was uncommon. Of the major community-associated clones, the Qld clone was unique in being invariably susceptible to non-β-lactam antimicrobials except erythromycin. Approximately a third of WA MRSA-1 isolates were resistant to erythromycin and fusidic acid. Both WA MRSA-1 and SWP MRSA had much higher levels of resistance to mupirocin than was seen in other clones. The prevalence of clindamycin resistance approximates that of erythromycin as most erythromycin resistance in *S. aureus* in Australia is due to the inducible MLS_B mechanism.

Table 6. Methicillin-resistant *Staphylococcus aureus* clones: proportion non-susceptible and mean age

Clone	MLST/SCC*mec*Σ	n	Ery	Tet	Tmp-SXT	Cip	Gen	Fus	Mup	Rif	Mean age (95% CI)
EA-EMRSA	ST239/III	114	99.1	96.5	97.4	94.7	93.9	1.8	0.9	4.4	64.7 (61.0–68.4)
Qld MRSA	ST93/IV	87	9.2	0.0	0.0	0.0	0.0	0.0	0.0	0.0	30.5 (26.5–34.5)
EMRSA-15	ST22/IV	85	52.9	2.0	0.0	97.6	1.2	0.0	1.2	2.4	71.9 (67.9–75.9)
WA MRSA-1	ST1/IV	62	33.9	1.6	0.0	3.2	8.1	33.9	8.1	1.6	49.1 (41.6–56.5)
SWP MRSA	ST30/IV	31	3.2	3.2	0.0	0.0	0.0	0.0	6.0	0.0	31.5 (23.9–39.0)
Other clones	various	83	43.4	12.0	3.6	20.5	6.0	2.4	2.4	3.6	47.8 (41.4–54.2)

MLST – multi-locus sequence type, SCCmec – staphylococcal chromosomal cassette, mec Ery – erythromycin, Tet – tetracycline Tmp-SXT – trimethoprim-sulphamethoxazole, Cip – Ciprofloxacin, Gen – gentamicin, Fus – fusidic acid, Mup – mupirocin, Rif – rifampicin, CI – confidence interval

No resistance was detected to vancomycin, teicoplanin, quinupristin-dalfopristin or linezolid. Seven of 2,979 (0.2%) isolates were classified as resistant to tigecycline using the US FDA and EUCAST breakpoints of 0.5mg/L. The regional level of resistance to penicillin and non-β-lactam antimicrobials in methicillin-susceptible *Staphylococcus aureus* (MSSA) is shown in Table 7.

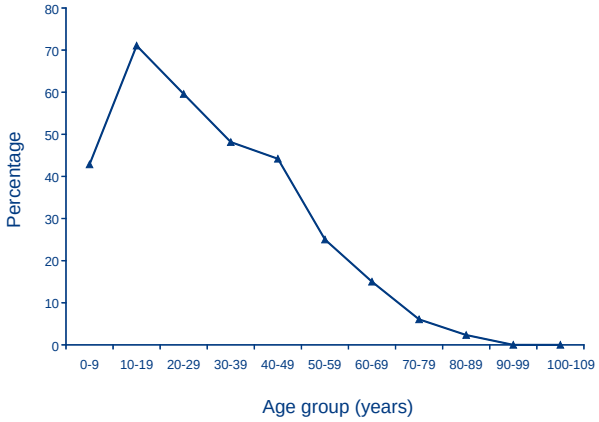
Prevalence of Panton-Valentine leukocidin

One isolate belonging to a HA-MRSA clone (EMRSA-15) was PVL positive (Table 8). The Qld clone, which was the predominant community-associated clone overall with 87 isolates, was invariably PVL positive. Other PVL positive clones included SWP, WA MRSA-1 (indistinguishable from USA400 by standard typing methods), USA300 (ST8-MRSA-IV), the European clone (ST80-MRSA-IV) and the Taiwanese clone (ST59-MRSA-V_T).

Relationship of age to methicillin-resistant *Staphylococcus aureus* prevalence

The mean age of patients with MRSA (52.2 (95% CI 49.7–54.7)) was significantly higher ($P<0.0001$) than that for patients with MSSA (44.7 (95% CI 43.6–45.7)). The mean age of patients with various MRSA clones also differed significantly: the mean ages for HA-MRSA clones (EA-MRSA and EMRSA-15) were highest, those for the PVL positive CA-MRSA clones (Qld and SWP) were lowest and the PVL negative community-associated WA MRSA-1 and the minor clones were in between (Table 6). The proportion of MRSA that was PVL positive was much higher in the young, peaking in the 10–19 year age group and falling steadily to zero in the 90–99 year age group (Figure 3). Of those patients presenting with SSTIs due to MRSA, a PVL positive clone was the most likely cause for those patients in the 10–19 (26/35 74.3% (95% CI 57.6%–86.0%) and the 20–29 year age groups (26/41 63.4% (95% CI 48.0%–76.5%).

Figure 3. Proportion of methicillin-resistant *Staphylococcus aureus* that are Panton-Valentine leukocidin positive, by age group



Discussion

Biennial community-based *S. aureus* antimicrobial surveillance programs have been performed in Australia by AGAR since 2000.[9] Over this time the number of participating laboratories has varied from

Table 7. Methicillin-susceptible *Staphylococcus aureus:* number and proportion (%) non-susceptible

Drug	NSW/ACT		Qld/NT		SA		Vic/Tas		WA		Aus		Difference across region	
	n	%	n	%	n	%	n	%	n	%	n	%	X^2	*P*
Total isolates	689		511		263		688		352		2,503			
Penicillin	582	84.5	445	87.1	224	85.2	588	85.5	296	84.1	2,135	85.3	2.104	0.8346
Erythromycin	70	10.2	65	12.7	39	14.8	69	10.0	35	9.9	278	11.1	6.956	0.2239
Clindamycin*	7	1.0	3	0.6	0	0.0	7	1.0	4	1.1	21	0.8	3.512	0.6216
Tetracycline	29	4.2	15	2.9	5	1.9	27	3.9	13	3.7	89	3.6	3.822	0.5752
Trimethoprim-sulphamethoxazole	23	3.3	6	1.2	3	1.1	16	2.3	3	0.9	51	2.0	11.57	0.0412
Ciprofloxacin	17	2.5	5	1.0	3	1.1	15	2.2	3	0.9	43	1.7	6.899	0.2283
Gentamicin	9	1.3	5	1.0	1	0.4	7	1.0	1	0.3	23	0.9	3.625	0.6046
Fusidic acid	25	3.6	27	5.3	9	3.4	32	4.7	13	3.7	106	4.2	2.988	0.7018
Rifampicin	4	0.6	5	1.0	0	0.0	1	0.1	0	0.0	7	0.3	4.635	0.4620
Mupirocin	10	1.5	13	2.5	2	0.8	5	0.7	9	2.6	39	1.6	9.770	0.0820

X^2 – chi-square, *P* - probability

* Constitutive resistance

No resistance was detected to vancomycin, teicoplanin, quinupristin-dalfopristin or linezolid.

24 to 30 institutions with the percentage of *S. aureus* identified as MRSA increasing significantly from 10.3% in 2000[9] to 16% in 2006 ($P<0.0001$).

In the 2006 program the percentage of *S. aureus* identified as MRSA ranged from 11.3% in Western Australia to 23% in New South Wales/Australian Capital Territory. When compared with the 2004 program a significant decrease was observed in Queensland/Northern Territory (19.8% to 14.8%), which may be related to an increase in regional participation to 6 laboratories in 2006 with a resulting change in the study population. The increase in MRSA between 2000 and 2006 has primarily been due to the emergence of CA-MRSA clones in the Australian community. Of the 462 MRSA referred to the WA Gram-positive Bacteria Typing and Research Unit in 2006, 200 (6.7% of all *S. aureus*) and 262 (8.8%) were classified as HA-MRSA and CA-MRSA clones, respectively. When compared with the 2000 program the percentage of *S. aureus* characterised as HA-MRSA has not significantly increased (5.6% to 6.7%). However the percentage of *S. aureus* characterised as CA-MRSA has almost doubled increasing from 4.7% to 8.8% ($P<0.0001$). Consequently, the emergence of CA-MRSA has resulted in a significant increase in the burden of CO-MRSA disease in Australia.

Three HA-MRSA clones were identified in this survey: EA EMRSA (ST239-MRSA-III), EMRSA-15 (ST22-MRSA-IV) and New York/Japan MRSA (ST5-MRSA-II). EA-MRSA, initially reported in eastern Australia in the late 1970s[21, 22] and possibly the most successful international HA-MRSA clone, accounted for approximately 25% of CO-MRSA infections. Although EA-MRSA continues to be the most prevalent MRSA isolated in the Australian community, since the 2000 survey (when it accounted for almost half of CO-MRSA isolated[9]) it has decreased significantly in most regions of Australia; including New South Wales/Australian Capital Territory (50.4% to 29.3%), South Australia (40.0% to 2.8%) and Victoria/Tasmania (80.6% to 39.8%). However in Queensland/Northern Territory the percentage was similar ranging from 22.2% in 2000 to 18.6% in 2006, which once again may be due to a major change in the study population in the region in 2006. As in previous surveys, little or no EA-MRSA was isolated in Western Australia. This is due to the WA 'MRSA search and destroy' policy introduced in 1982, which has prevented EA-MRSA from becoming established in the state's hospitals and spilling over into the community.

In contrast to EA-MRSA, the percentage of MRSA identified as EMRSA-15 has increased in most regions of Australia. EMRSA-15 is an international HA-MRSA associated with hospital infection and was first documented in Australia in Perth in 1997, where it was detected during the pre-employment screening of healthcare workers coming from the United Kingdom (UK) and Ireland.[23] This clone is now well established throughout Australia increasing from 11.7% of CO-MRSA infections in 2000 to 18.4% in 2006.[9] During this time, significant increases have been reported in New South Wales/Australian Capital Territory (18.4% to 25.8%),

Table 8. Methicillin-resistant *Staphylococcus aureus* clones: Panton-Valentine leukocidin polymerase chain reaction results

Clone	MLST/SCCmec	Number of isolates	PVL PCR positive	%
EA-EMRSA	ST239/III	114	0	0
Qld MRSA	ST93/IV	87	87	100
EMRSA-15	ST22/IV	85	1	1
WA MRSA-1	ST1/IV	62	5	8
SWP MRSA	ST30/IV	31	30	97
WA MRSA-3	ST5/IV	20	0	0
WA MRSA-2	ST78/IV	13	0	0
Victorian MRSA	ST45/IV	9	0	0
NT MRSA	ST75/IV	7	0	0
USA300	ST8/IV	6	6	100
WA MRSA-4	ST45/V	5	0	0
WA MRSA-5	ST8/IV	3	0	0
European MRSA	ST80/IV	2	2	100
Taiwan MRSA	ST59/V_T	2	2	100
---------	ST5/V	2	0	0
New York/Japan	ST5/II	1	0	0
WA MRSA-13	ST584/IV	1	0	0
WA MRSA-15	ST59/IV	1	0	0
WA MRSA-35	ST5/V	1	0	0
WA MRSA-69	ST12/IV	1	0	0
SWP variant	ST30/IV	1	1	100
---------	ST1/V	1	0	0
---------	ST20/V	1	0	0
---------	ST30slv*/IV	1	1	100
---------	ST338/V	1	1	100
---------	ST361/IV	1	0	0
---------	ST72/V	1	0	0
---------	ST7/V	1	0	0
---------	ST8/IV	1	0	0
Total MRSA		462	136	29

MLST multi-locus sequence type
SCCmec staphylococcal chromosomal cassette mec
PVL Panton-Valentine leukocidin
PCR polymerase chain reaction
* slv – single locus variant

South Australia (12% to 22.2%), Victoria/Tasmania (0% to 17.4%) and Western Australia (6.8% to 11.4%). The percentage has not significantly altered in Queensland/Northern Territory (3.7% to 4.7%).

In Western Australia EMRSA-15 has become a successful coloniser in many of the state's long term care facility residents, who with healthcare workers from the UK and Ireland have become the major source of EMRSA-15. The mean ages of EMRSA-15 and EA-MRSA patients in this survey were significantly higher than that seen with other MRSA clones, which is consistent with their known association with healthcare related infection and with long term care facilities.

A single isolate of New York/Japan MRSA (ST5-MRSA-II) was isolated in 2006. Although this clone is a major HA-MRSA in the United States of America (where it is also known as USA100) in recent years it has become a major cause of CO-MRSA infections.[24] In Australia, a single strain outbreak of

New York/Japan was recently reported in regional Western Australia.[25] The outbreak which involved several hospitals and long term care facilities was linked to an Australian healthcare worker who had previously worked and received surgery in a New York city hospital. Although the outbreak was able to be contained, the strain continues to be recovered from residents living in the region's long term care facilities. From the Western Australia experience it has been demonstrated that New York/Japan MRSA is highly transmissible and therefore may become a major MRSA strain in both the hospital and community setting if the opportunity arises.

As has been reported overseas, CA-MRSA in Australia is polyclonal. In this survey 25 different clones were characterised by MLST/SCC*mec* typing. Using BURST analysis these clones can be grouped into 13 clonal clusters and 2 singletons. Within some clonal clusters more than 1 clone was identified. In addition, both SCC*mec* IV and V were described. This suggests that CA-MRSA clones have evolved on multiple occasions in Australia with the emergence of new clones due to the horizontal and vertical transfer of SCC*mec* into *S. aureus* with diverse genetic backgrounds. Although multiple CA-MRSA clones were identified, as in previous surveys over 85% of CA-MRSA can be classified into 6 clones; Qld MRSA (ST93-MRSA-IV) (18.8% of MRSA), WA MRSA-1 (ST1-MRSA-IV) (13.4%) SWP MRSA (ST30-MRSA-IV) (6.9%), WA MRSA-3 (ST5-MRSA-IV) (4.3%), WA MRSA-2 (ST78-MRSA-IV) (2.8%) and the Victorian MRSA (ST45-MRSA-IV) (1.9%) In contrast to previous surveys, ST93-MRSA-IV (a PVL positive clone), which is now found throughout Australia, has become the predominant CA-MRSA clone in Australia.

The predominance of the Qld MRSA clone has resulted in a significant change in the percentage of CA-MRSA in Australia that are PVL positive. Initially, CO-MRSA infections in Australia were dominated by PVL negative strains including WA MRSA-1, WA MRSA-2, WA MRSA-3, NT MRSA (ST75-MRSA-IV) and the Victorian MRSA (ST45-MRSA-IV). However with the emergence of the Qld MRSA and the introduction of several international PVL-positive community-associated MRSA strains, including SWP (ST30-MRSA-IV), USA300 (ST8-MRSA-IV), USA400 (ST1-MRSA-IV), European MRSA (ST80-MRSA-IV) and the Taiwan MRSA (ST59-MRSA-V_T), over 50% of CA-MRSA in Australia are now PVL positive. Although the mean age of patients with MRSA was significantly older compared with MSSA (52 vs 45 years; $P<0.001$) 70% of PVL-positive MRSA infections occurred in patients 10–19 years of age.

Although transmission of PVL-positive CA-MRSA in the community has been reported, outbreaks of these strains within the hospital environment have not. In this survey a PVL-positive EMRSA-15 strain was identified. EMRSA-15 is a highly transmissible HA-MRSA frequently isolated in many Australian hospitals and long term care facilities.

In conclusion, the AGAR 2006 *S. aureus* surveillance program has shown that the proportion of community-onset *S. aureus* infections due to MRSA is increasing throughout Australia, and that this increase is due to the spread of the PVL-positive Qld MRSA clone as well as the introduction of several international PVL-positive clones including USA300 (ST8-MRSA-IV). This shift in the molecular epidemiology of MRSA clones in the Australian community will potentially increase the number of SSTI in young Australians. As SSTI caused by PVL-positive *S. aureus* frequently results in hospitalisation the emergence of these strains in the community as well as the detection of a PVL-positive healthcare-associated MRSA strain (EMRSA-15) is a major health concern.

A full detailed report of this study may be found on the Australian Group on Antimicrobial Resistance website: http://www.antimicrobial-resistance.com/ under 'AMR surveillance'.

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The AGAR participants were:

Australian Capital Territory

Peter Collignon, Susan Bradbury, The Canberra Hospital

New South Wales

John Ferguson, Jo Anderson, Hunter Area Pathology Service
Tom Gottlieb, Glenn Funnell, Concord Repatriation General Hospital
George Kotsiou, Clarence Fernandes, Royal North Shore Hospital

Richard Benn, Barbara Yan, Royal Prince Alfred Hospital
Iain Gosbell, Helen Ziochos, South Western Area Pathology Service
David Mitchell, Lee Thomas, Westmead Hospital
James Branley, Donna Barbaro, Nepean Hospital
Miriam Paul, Richard Jones, Douglass Hanly Moir Pathology

Northern Territory

Gary Lum, Paul Southwell, Royal Darwin Hospital

Queensland

Graeme Nimmo, Narelle George, Pathology Queensland Central Laboratory
Joan Faoagali, Gweneth Lye, Pathology Queensland, Princess Alexandra Hospital
Chris Coulter, Sonali Gribble, Pathology Queensland, Prince Charles Hospital
Dale Thorley, Pathology Queensland, Gold Coast Hospital
Jenny Robson, Sullivan Nicolaides Pathology

South Australia

John Turnidge, Jan Bell, Women's and Children's Hospital
Ivan Bastian, Rachael Pratt, Institute of Medical & Veterinary Science
David Gordon, Hendrik Pruul, Flinders Medical Centre

Tasmania

Alistair McGregor, Rob Peterson, Royal Hobart Hospital, TAS; Erika Cox, Kathy Wilcox, Launceston General Hospital

Victoria

Denis Spelman, Clare Franklin, Alfred Hospital
Suzanne Garland, Gena Gonis, Royal Children's and Women's Hospitals
Mary Jo Waters, Linda Joyce, St Vincent's Hospital
Barrie Mayall, Peter Ward, Austin Health
John Andrew, Di Olden, Gribbles Pathology (VIC) Pty Ltd
Tony Korman, Despina Kotsanas, Monash Medical Centre

Western Australia

Clay Gollege, Barbara Henderson, PathWest QEII
Keryn Christiansen, Geoff Coombs, PathWest Royal Perth Hospital

David McGechie, Graham Francis, PathWest Fremantle Hospital
Susan Benson, Janine Fenton, St John of God Pathology

Author details

Geoffrey W Coombs[1]
Graeme R Nimmo[2]
Julie C Pearson[3]
Keryn J Christiansen[4]
Jan M Bell[5]
Peter J Collignon[6]
Mary-Louise McLaws[7]

1. Principal Scientist, Department of Microbiology and Infectious Diseases, PathWest Laboratory Medicine WA, Royal Perth Hospital, Perth, Western Australia
2. Director, Division of Microbiology, Pathology Queensland Central Laboratory, Herston, Queensland and Associate Professor, Faculty of Health Sciences, University of Queensland
3. Scientific Officer for the Australian Group on Antimicrobial Resistance, Department of Microbiology and Infectious Diseases, PathWest Laboratory Medicine WA, Royal Perth Hospital, Western Australia
4. Director, Department of Microbiology and Infectious Diseases, PathWest Laboratory Medicine WA, Royal Perth Hospital, Perth, Western Australia
5. Senior Scientist, Department of Microbiology and Infectious Diseases, Women's and Children's Hospital, North Adelaide, South Australia
6. Director, Infectious Diseases Unit and Microbiology Department, The Canberra Hospital, Garran, Australian Capital Territory
7. Director, Hospital Infection Epidemiology and Surveillance Unit, University of New South Wales, Sydney, New South Wales

Corresponding author: Assoc. Professor GR Nimmo, Division of Microbiology, Pathology Queensland Central Laboratory, Block 7, Herston Hospitals Complex, HERSTON QLD 4029. Telephone: +61 7 3636 8050. Facsimile: +61 7 3636 1336. Email: Graeme_Nimmo@health.qld.gov.au

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