# Prevalence of MRSA among *Staphylococcus aureus* isolated from hospital inpatients, 2005: Report from the Australian Group for Antimicrobial Resistance

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

## Abstract

The Australian Group for Antimicrobial Resistance conducted a survey of the prevalence of antimicrobial resistance in unique clinical isolates of *Staphylococcus aureus* from patients admitted to hospital for more than 48 hours. Thirty-two laboratories from all states and territories collected 2,908 isolates from 1 May 2005, of which 31.9% were methicillin-resistant *Staphylococcus aureus* (MRSA). The regional prevalence of MRSA varied significantly (P<0.0001) from 22.5% in Western Australia to 43.4% in New South Wales/Australian Capital Territory. Prevalence of MRSA from individual laboratories varied even more from 4% to 58%. This variation was explained in part by distribution of age with the risk of MRSA significantly (P<0.0001) increasing with age. Other unmeasured factors including hospital activity and infection control practices in the individual institution may have also contributed. Further investigation is warranted as reductions in prevalence would reduce morbidity, mortality and healthcare costs. *Commun Dis Intell* 2007;31:288–296.

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

## Introduction

*Staphylococcus aureus* remains a major bacterial pathogen and is associated with considerable morbidity and mortality. Manifestations of *S. aureus* infection range from mild to moderate skin and soft tissue infections such as impetigo and furunculosis to invasive and often life threatening infections such as osteomyelitis, necrotising pneumonia and infective endocarditis. Bacteraemia is also common. In the pre-antibiotic era the mortality of staphylococcal bacteraemia was as high as 90%.[1] With antibiotic treatment, mortality has fallen but remains a major issue. With methicillin-sensitive *S. aureus* (MSSA) the median associated mortality is 25% (range 4%–52%) while with methicillin-resistant *S. aureus* (MRSA) the median is 35% (range 0%–83%).[2] In Australia, as in most of the world, antimicrobial resistance in *S. aureus* is a major impediment to effective treatment. Hospital strains are frequently resistant to methicillin (and all other beta-lactams) and multiple other antimicrobials.[3]

Methicillin-resistant *S. aureus* was first reported in Australia in 1968.[4] This archaic strain of MRSA was not usually resistant to other non-beta-lactam antimicrobials and was not resistant to gentamicin. The emergence of MRSA resistant to gentamicin and other classes of antimicrobials was first noted in eastern Australia in 1976. Outbreaks of hospital infection due to multi-resistant MRSA (mMRSA) occurred in the state of Victoria in the late 1970s and early 1980s.[5,6] mMRSA became endemic in hospitals in the eastern Australian states in the late 1980s and 1990s with some spread to hospitals in South Australia, the Northern Territory and Tasmania.[3,7] However, these strains did not become established in Western Australian hospitals due to active screening and infection control policies.[3,8] Eastern Australian MRSA has now been shown to be one clone by multi-locus sequence typing – ST239-MRSA-III.[9] This is one of the most successful MRSA clones and is now found extensively in Europe, Asia, and South America. More recently, MRSA clones of overseas origin have also been found in Australia. Most notably the United Kingdom strain, EMRSA-15, has spread widely in Australia to become a major endemic cause of hospital sepsis.[9]

Vancomycin has been the mainstay of treatment for serious infections due to MRSA. However, there is evidence that vancomycin is less effective in the treatment of methicillin-sensitive *S. aureus* than anti-staphylococcal beta-lactams.[10,11] Failure of vancomycin treatment of MRSA has been associated with the emergence of strains with MICs to vancomycin in the intermediate range (VISA).[12,13] These strains have been described in many parts of the world including Australia.[14] Isolation of VISA follows failure of prolonged treatment with vancomycin. One recent study has suggested that treatment failure is related to slightly higher vancomycin MICs (1.0–2.0 mg/L versus ≤0.5 mg/L) in pre-treatment isolates of MRSA.[15] Few treatment options remain for multi-resistant

MRSA and resistance to linezolid, one of the few new anti-staphylococcal agents of recent years, is already being reported.[16]

While it is well known that *S. aureus* is a major cause of severe sepsis, few population based estimates of its incidence or prevalence are available. A recent Australian survey of *S. aureus* bacteraemia from 1999 to 2002 documented 3,129 episodes.[2] Approximately 51% of bacteraemic episodes had their onset in hospitals. MRSA caused 40% of hospital-onset and 12% of community-onset episodes. The authors estimated that approximately 6,900 episodes of *S. aureus* bacteraemia occur in Australia annually. This equates to 35 episodes per 100,000 population. Meta-analysis of the outcomes of *S. aureus* bacteraemia has shown that the relative risk of death due to MRSA bacteraemia is approximately twice that due to MSSA.[17,18] It is widely acknowledged that nosocomial MRSA infection represents an additional burden of disease not just replacement of MSSA infection.[19] The cost of these additional infections is substantial for hospitals, patients and society. While costs vary from country to country, annual additional hospital costs due to MRSA in the United States of America are estimated at between US$1.5 billion and US$4.2 billion.[19] In Australia, the additional hospital costs associated with nosocomial *S. aureus* bacteraemia alone are estimated at approximately $150 million.[2] Effective infection control measures have been shown to reduce nosocomial infection significantly and to result in substantial savings.[19]

The objective of this study was to determine the prevalence of antimicrobial resistance in clinical isolates of *S. aureus* throughout Australia in hospital inpatients admitted for 48 hours or more.

## Methods

Thirty-two laboratories from all six states, the Australian Capital Territory and the Northern Territory participated in the *S. aureus* Australian Group for Antimicrobial Resistance (AGAR) survey. From 1 May 2005, each laboratory collected up to 100 consecutive significant clinical isolates from hospital inpatients (hospital stay >48 hours at the time of specimen collection). Only one isolate per patient was tested and no isolates from screening swabs were included. If *S. aureus* was isolated from more than one site, then the isolate from the most significant clinical site was tested. Specimens received for the purpose of gathering surveillance data were excluded.

### Species identification

*S. aureus* was identified by morphology and positive results of at least two of three tests: slide coagulase test, tube coagulase test, and demonstration of deoxyribonuclease production.[20] 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 CLSI breakpoints.[22,23] Penicillin susceptible strains were tested for ß-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 CDS methods.[21–23] The minimum inhibitory concentration (MIC) of mupirocin resistant isolates was determined by Etest® (AB Biodisk, Solna, Sweden). The macro Etest® method was used to determine hetero-resistance to vancomycin.

### 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 five age groups. All descriptive and inferential statistics were calculated using Epi Info version 6.0.4 (Centers for Disease Control and Prevention, Atlanta, Ga, USA) with the alpha level set at the 5% level for two-sided tests for significance.

## Results

Participating laboratories (27 public and 5 private) were located in New South Wales (8), the Australian Capital Territory (1), Queensland (6), Victoria (6), Tasmania (2), the Northern Territory (1), South Australia (4) and Western Australia (4). To ensure institutional anonymity data were combined for New South Wales and the Australian Capital Territory; Tasmania and Victoria; and Queensland and the Northern Territory (Table 1). There were 2,908 isolates included in the survey with the majority (76.1%) of isolates contributed by New South Wales/Australian Capital Territory (28.4%), Victoria/Tasmania (24.9%) and Queensland/Northern Territory (22.8%).

### Specimen source

The majority of *S. aureus* isolates (67.6%) were from skin and soft tissue infections (Table 2). Respiratory specimens were the second most common source (17.4%) followed by blood culture isolates, 6.7%, with significantly (P<0.0001) more isolates causing non-invasive (91.2%) than invasive (8.7%) infections.

### Susceptibility results

Nationally, 31.9% of *S. aureus* isolates were MRSA (Table 3) with the proportion varying significantly between states and territories (X2 = 110.54, P<0.0001). The proportion of MRSA in New South Wales/Australian Capital Territory hospitals (43.4%) was significantly higher (P<0.001) than the Australian average of 31.9%. There was no significant difference in the proportion of MRSA isolates that caused invasive infections (20.0% to 41.2% respectively, P=0.267) while the proportion of non-invasive infections ranged from 22.8% in Western Australia to 43.7% in New South Wales/Australian Capital Territory (P<0.0001). There was a wide range in the proportions of MRSA isolated by institutions with 31.0%–58.0% in New South Wales/Australian Capital Territory, 19.0%–36.0% in Queensland/Northern Territory, 15.0%–29.0% in South Australia, 4.0%–53.5% in Victoria/Tasmania and 14.5%–29.2% in Western Australia (Table 4).

Resistance in MRSA to non-beta-lactam antimicrobials varied significantly between states with the exception of mupirocin (Table 5). Resistance with the widest range was identified for gentamicin (5.0% to 79.5%, P<0.0001), tetracycline (6.3% to 83.0%, P<0.0001), cotrimoxazole (7.5% to 80.8%, P<0.0001) and clindamycin (8.3% to 68.7%, P<0.0001). Resistance to ciprofloxacin was also common ranging from 42.5%–89.4% (P<0.0001). Resistance to fusidic acid across the states varied significantly (P=0.0023) with the highest proportion in South Australia (11.9%). There was no significant difference (P=0.713) in the low levels of mupirocin resistance. One isolate from Victoria/Tasmania had a quinupristin/dalfopristin MIC

**Table 1. Isolates by region**

| Region | Number of institutions | Total | % 95%CI |
|---|---|---|---|
| New South Wales/ Australian Capital Territory | 9 | 825 | 28.4 (26.7–30.0) |
| Queensland/ Northern Territory | 7 | 664 | 22.8 (21.3–24.4) |
| South Australia | 4 | 340 | 11.7 (10.5–12.9) |
| Victoria/Tasmania | 8 | 724 | 24.9 (23.3–26.5) |
| Western Australia | 4 | 355 | 12.2 (11.0–13.4) |
| Total | 32 | 2,908 | 100 |

**Table 2. Source of isolates**

| Specimen source | n | % |
|---|---|---|
| Skin and soft tissue | 1,967 | 67.6 |
| Respiratory | 506 | 17.4 |
| Blood | 194 | 6.7 |
| Urine | 92 | 3.2 |
| Eye | 62 | 2.1 |
| Sterile site | 50 | 1.7 |
| Ear | 13 | 0.4 |
| Cerebrospinal fluid | 8 | 0.3 |
| Other | 11 | 0.4 |
| Unknown | 5 | 0.2 |
| Total | 2,908 | |
| Invasive | 252 | 8.7 |
| Non-invasive | 2,651 | 91.2 |
| Not specified | 5 | 0.2 |

**Table 3. Proportion of methicillin-resistant *Staphylococcus aureus* for all isolates, invasive isolates and non-invasive isolates, by region**

| | All Isolates | | Invasive | | Non-invasive | |
|---|---|---|---|---|---|---|
| | n | % | n | % | n | % |
| NSW/ACT | 358/825 | 43.4 | 35/85 | 41.2 | 323/739 | 43.7 |
| Qld/NT | 177/664 | 26.7 | 13/36 | 36.1 | 164/628 | 26.1 |
| SA | 84/340 | 24.7 | 10/34 | 29.4 | 73/304 | 24.0 |
| Vic/Tas | 229/724 | 31.6 | 23/59 | 39.0 | 206/664 | 31.0 |
| WA | 80/355 | 22.5 | 6/30 | 20.0 | 74/325 | 22.8 |
| Aus | 928/2,908 | 31.9 | 87/244 | 35.7 | 840/2,660 | 31.6 |
| Difference across regions $\chi^2$ | 81.01 | | 5.20 | | 78.81 | |
| P value | <0.0001 | | 0.267 | | <0.0001 | |

of >2 mg/L by broth micro-dilution and an Etest MIC of 6 mg/L. In addition, one result for quinupristin/dalfospristin was missing. One isolate from New South Wales/Australian Capital Territory had Vitek MIC results of 4 mg/L for vancomycin and teicoplanin (non-susceptible). The broth dilution MIC of both agents was 2 mg/L and the isolate was confirmed as a hetero-vancomycin intermediate *S. aureus* (hVISA) by the macro Etest method.

MSSA were generally susceptible to most non-beta-lactam antimicrobials with no significant difference in proportion across all regions with the exception of the level of resistance in tetracycline (P=0.0005) with New South Wales/Australian Capital Territory having the highest level at 3.6%, and gentamicin (P=0.0047) with Victoria/Tasmania having the highest level at 3.2% (Table 6).

**Table 4. Proportion of methicillin-resistant *Staphylococcus aureus*, by institution**

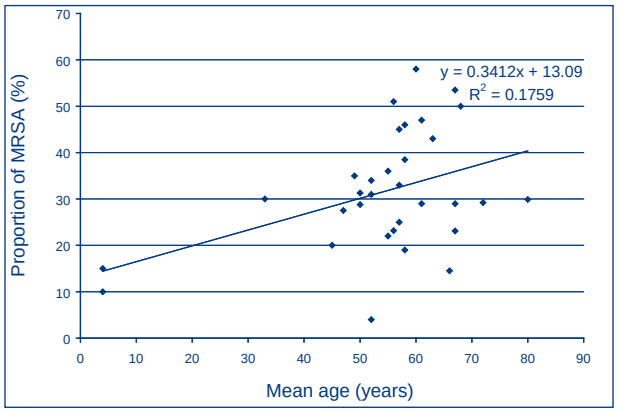
| Region | Laboratory code | % MRSA |
|---|---|---|
| NSW/ACT | 1 | 31.0 |
| | 2 | 50.0 |
| | 3 | 31.3 |
| | 4 | 47.0 |
| | 5 | 58.0 |
| | 6 | 51.0 |
| | 7 | 38.5 |
| | 8 | 46.0 |
| | 9 | 34.0 |
| Qld/NT | 10 | 30.0 |
| | 11 | 19.0 |
| | 12 | 20.0 |
| | 13 | 29.9 |
| | 28 | 23.2 |
| | 29 | 28.8 |
| | 30 | 36.0 |
| SA | 14 | 29.0 |
| | 15 | 29.0 |
| | 16 | 15.0 |
| | 17 | 27.5 |
| Vic/Tas | 18 | 4.0 |
| | 19 | 45.0 |
| | 20 | 23.1 |
| | 21 | 10.0 |
| | 22 | 43.0 |
| | 23 | 53.5 |
| | 31 | 35.0 |
| | 32 | 33.0 |
| WA | 24 | 14.5 |
| | 25 | 25.0 |
| | 26 | 22.0 |
| | 27 | 29.2 |
| Australia | | 31.9 |

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

Patients with MRSA ranged in age from less than one year to 100 years, with a mean of 54.3 years. The distribution of age was skewed towards the elderly with the 25th percentile at 35 years, the 50th at 61 years and the 75th at 77 years. MSSA was significantly (P<0.0001) more common than MRSA in all five age groups; neonatal (<1–1 year), paediatric (2–16 years), adult (17–40 years), middle-age (41–61 years) and the older (62–100 years) (Table 7).

When the relationship between mean age and proportion of MRSA in institutions was examined, a significant (P two tailed = 0.02), but weak linear trend (r = 0.4195), was identified (Figure 1). The sample sizes contributed by the member hospitals were small with a wide dispersion of the mean age (Figure 2) across the 32 facilities. However, when age was categorised into five ranges for the aggregated data from all hospitals and odds ratio of MRSA cases for each age group was examined against the youngest, MRSA was significantly more likely to occur in patients in successively older age groups compared with MSSA (Table 8). Advancing age is a strongly significant risk factor for acquisition with patients aged between 62 years and 100 years being 10.33 (P<0.0001) times more likely to have MRSA (not MSSA) compared with babies.

**Figure 1. Relationship of mean age and proportion of methicillin-resistant *Staphylococcus aureus* for 32 institutions**



## Table 5. Number and proportion non-susceptible methicillin-resistant *Staphylococcus aureus* isolates, by region

| Region | Em | | Cm* | | Tc | | Tmp-SXT | | Cf | | Gm | | Fa | | Mp | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | n | % | n | % | n | % | n | % | n | % | n | % | n | % | n | % |
| NSW/ACT | 309/357 | 86.6 | 193/281 | 68.7 | 247/358 | 69.0 | 251/358 | 70.1 | 320/358 | 89.4 | 250/358 | 69.8 | 13/358 | 3.6 | 12/358 | 3.4 |
| Qld/NT | 129/177 | 72.9 | 74/177 | 41.8 | 79/177 | 44.6 | 91/177 | 51.4 | 111/177 | 62.7 | 98/177 | 55.4 | 10/177 | 5.6 | 4/177 | 2.3 |
| SA | 51/84 | 60.7 | 7/84 | 8.3 | 30/84 | 35.7 | 27/84 | 32.1 | 46/84 | 54.8 | 28/84 | 33.3 | 10/84 | 11.9 | 1/84 | 1.2 |
| Vic/Tas | 207/229 | 90.4 | 94/228 | 41.2 | 190/229 | 83.0 | 185/229 | 80.8 | 202/229 | 88.2 | 182/229 | 79.5 | 4/229 | 1.7 | 6/229 | 2.6 |
| WA | 46/80 | 57.5 | 8/80 | 10.0 | 5/80 | 6.3 | 6/80 | 7.5 | 34/80 | 42.5 | 4/80 | 5.0 | 3/80 | 3.8 | 1/80 | 1.3 |
| Aus | 742/927 | 80.0 | 376/850 | 44.2 | 551/928 | 59.4 | 560/928 | 60.3 | 713/928 | 76.8 | 562/928 | 60.6 | 40/928 | 4.3 | 24/928 | 2.6 |
| Difference across regions $\chi^2$ | 75.61 | | 151.25 | | 201.42 | | 181.44 | | 144.13 | | 178.66 | | 16.63 | | 2.13 | |
| P value | <0.0001 | | <0.0001 | | <0.0001 | | <0.0001 | | <0.0001 | | <0.0001 | | 0.0023 | | 0.713 | |

Em: erythromycin, Cm: clindamycin, Tc: tetracycline, Tmp-SXT: trimethoprim/sulphamethoxazole, Cf: ciprofloxacin, Gm: gentamicin, Fa: fusidic acid, Mp: mupirocin

* Constitutive resistance.

## Table 6. Number and proportion non-susceptible methicillin sensitive *Staphylococcus aureus* isolates, by region

| Region | Pc | | Em | | Cm | | Tc | | Tmp-SXT | | Cf | | Gm | | Fa | | Mp | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | n | % | n | % | n | % | n | % | n | % | n | % | n | % | n | % | n | % |
| NSW/ACT | 405/467 | 86.7 | 60/467 | 12.8 | 8/448 | 1.8 | 17/467 | 3.6 | 12/467 | 2.6 | 18/466 | 3.9 | 5/467 | 1.1 | 13/467 | 2.8 | 4/467 | 0.9 |
| Qld/NT | 416/487 | 85.4 | 63/487 | 12.9 | 2/487 | 0.4 | 8/487 | 1.6 | 3/487 | 0.6 | 8/487 | 1.6 | 5/487 | 1.0 | 18/487 | 3.7 | 5/487 | 1.0 |
| SA | 219/256 | 85.5 | 22/256 | 8.6 | 3/256 | 1.2 | 7/256 | 2.7 | 3/256 | 1.2 | 6/256 | 2.3 | 2/256 | 0.8 | 7/256 | 2.7 | 2/256 | 0.8 |
| Vic/Tas | 406/495 | 82.0 | 66/495 | 13.3 | 8/495 | 1.6 | 25/495 | 5.1 | 8/495 | 1.6 | 10/495 | 2.0 | 16/495 | 3.2 | 18/495 | 3.6 | 6/495 | 1.2 |
| WA | 241/275 | 87.6 | 21/275 | 7.6 | 4/275 | 1.5 | 0/275 | 0.0 | 2/275 | 0.7 | 6/275 | 2.2 | 1/275 | 0.4 | 15/275 | 5.5 | 3/275 | 1.1 |
| Aus | 1,687/1,980 | 85.2 | 232/1,980 | 11.7 | 25/1,961 | 1.3 | 57/1,980 | 2.9 | 28/1,980 | 1.4 | 48/1,979 | 2.4 | 29/1,980 | 1.5 | 71/1,980 | 3.6 | 20/1,980 | 1.0 |
| Difference across regions $\chi^2$ | 6.17 | | 9.37 | | 4.37 | | 20.15 | | 7.88 | | 5.75 | | 15.01 | | 4.20 | | 0.47 | |
| P value | 0.187 | | 0.052 | | 0.358 | | 0.0005 | | 0.096 | | 0.219 | | 0.0047 | | 0.379 | | 0.977 | |

Em: erythromycin, Cm: clindamycin, Tc: tetracycline, Tmp-SXT: trimethoprim/sulphamethoxazole, Cf: ciprofloxacin, Gm: gentamicin, Fa: fusidic acid, Mp: mupirocin

* Constitutive resistance.

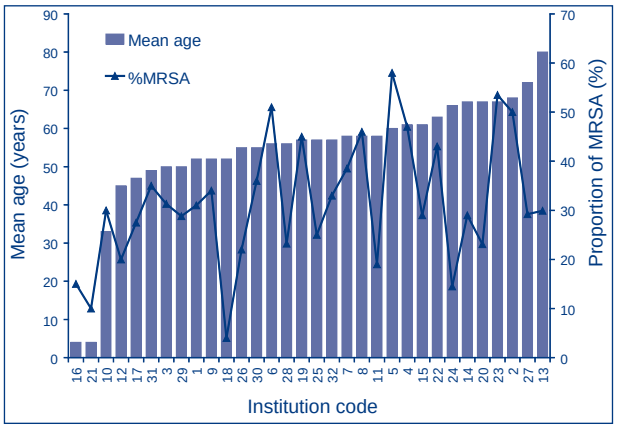
**Table 7. Age by methicillin susceptibility of *Staphylococcus aureus***

| Age | Total | | | MRSA | | | MSSA | | | Difference in isolates by age category (row) | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | n | % | 95% CI | n | Row % | Column % | n | Row % | Column % | $\chi^2$ | P |
| 0–1 | 264 | 9.1 | 8.1–10.2 | 17 | 6.4 | 1.8 | 247 | 93.6 | 12.5 | 400.76 | <0.0001 |
| 2–16 | 132 | 4.5 | 3.8–5.4 | 29 | 22.0 | 3.1 | 103 | 78.0 | 5.2 | 82.97 | <0.0001 |
| 17–40 | 426 | 14.7 | 13.4–16.0 | 113 | 26.5 | 12.2 | 313 | 73.5 | 15.8 | 187.79 | <0.0001 |
| 41–61 | 642 | 22.1 | 20.6–23.6 | 207 | 32.2 | 22.3 | 435 | 67.8 | 22.0 | 161.94 | <0.0001 |
| 62–100 | 1,443 | 49.6 | 47.8–51.5 | 562 | 38.9 | 60.6 | 881 | 61.1 | 44.5 | 1142.81 | <0.0001 |
| Total | 2,907 | 100 | – | 928 | 31.9 | 100 | 1,979 | 68.1 | 100 | 103.96 | <0.0001 |

**Table 8. Risk of methicillin-resistant *Staphylococcus aureus*, by age groups**

| Age | Unadjusted Odds Ratio | 95% CI | P | Adjusted Odds Ratio* | 95%CI | P |
|---|---|---|---|---|---|---|
| 0–1 | 1 (referent group) | – | – | 1 (referent group) | – | – |
| 2–16 | 4.09 | 2.06 – 8.16 | <0.0001 | 4.25 | 2.22 – 8.11 | <0.0001 |
| 17–40 | 5.25 | 2.99 – 9.32 | <0.0001 | 5.72 | 3.22 – 9.85 | <0.0001 |
| 41–61 | 6.91 | 4.02 – 12.04 | <0.0001 | 7.37 | 4.36 – 12.46 | <0.0001 |
| 62–100 | 9.27 | 5.49 – 15.86 | <0.0001 | 10.33 | 6.21 – 17.10 | <0.0001 |
| | P<0.0001, $\chi^2$ for linearity = 119.729 | | | * Adjusted for state and territories | | |

**Figure 2. Mean age compared with proportion of methicillin-resistant *Staphylococcus aureus* in participating institutions**




## Discussion

Surveys conducted by AGAR from 1986 to 1999 included all consecutive clinical isolates of *S. aureus* during the survey period regardless of acquisition.[3,7,24] Participating laboratories did not need to acquire any additional information to distinguish between inpatients and outpatients and so an overall MRSA prevalence was derived. Compliance with methodology was a potential issue particularly in the early days of the surveys but this simple data collection was reliably achieved. It also allowed for comparison of results over a prolonged period. The advent of community strains of MRSA during the 1990s[25,26] however, led to interest in studying the prevalence of MRSA in outpatient infections alone. AGAR responded by conducting biennial outpatient surveys from 2000 onwards.[9,27] Since then evidence has emerged that strains that initially were acquired almost exclusively in the community were now being acquired in the health care setting with increasing frequency.[28] Therefore, in 2005 a survey of hospital-acquired *S. aureus* infection was undertaken. The results provide us with the first accurate estimates at a national level of the proportion of hospital-acquired *S. aureus* infection that are due to MRSA.

In this survey 2,908 isolates were collected in 32 laboratories covering all states and territories. Overall, 31.9% of isolates were MRSA. While there was a significant difference in the proportion of MRSA between regions (from 22.5% in Western Australia to 43.4% in New South Wales), this may have been due in part to different age distributions. The overall proportion of MRSA in invasive (mainly bacteraemia) isolates was similar to that of non-invasive isolates (35.7% and 31.6% respectively, P=0.195. The high proportion of MRSA in invasive isolates is of concern as MRSA bacteraemia is associated with increased mortality compared with MSSA.[17,18,31] Direct comparison with prevalence in other countries is difficult due to methodological differences. For example, the

European surveillance system reports the proportion of MRSA in bacteraemia isolates in both inpatients and outpatients in 23 countries.[32] Even so, the overall proportion in Europe in 2005 varied from 1.7% in Denmark to 55% in Malta. The Netherlands and the Scandinavian countries have been consistently able to keep MRSA at very low levels in their hospitals over long periods.

Resistance to non-beta-lactams in MRSA was common for erythromycin, clindamycin, tetracycline, cotrimoxazole, ciprofloxacin and gentamicin and varied considerably from region to region. This regional variability is due to the differential distribution of MRSA clones in the major cities. For example, ST239-MRSA-III (AUS-2 and AUS-3 strains), which is resistant to multiple non-beta-lactams including gentamicin, erythromycin and tetracycline, is endemic in the eastern states but is less common in Western Australia and South Australia. ST22-MRSA-IV (UK EMRSA-15), which is resistant to ciprofloxacin and often erythromycin but susceptible to all other non-beta-lactams, is more common in Western Australia as are other non-multi-resistant strains.[9,27] Resistance of MSSA to non-beta-lactam antimicrobials was uncommon except for erythromycin. There was little variability between regions in the low levels of resistance to other agents, with the exception of tetracycline and gentamicin. Once again this may be due to regional variations in the prevalence of strains of MSSA carrying different combinations of resistance genes.

The prevalence of MRSA isolates varied from 4.0% to 58.0% between institutions. The high levels in some institutions are a cause for concern given the increased mortality, morbidity and cost associated with MRSA infection.[19,33] While it is generally accepted that the prevalence of MRSA in an institution reflects the effectiveness of infection control practice,[34] it is also true that age is a risk factor or proxy for MRSA infection.[35] Analysis of the 2005 survey data confirmed that risk of MRSA did increase significantly with age ($P<0.0001$). There was also a weak association between mean age and proportion of MRSA in institutions. The weakness of the association was due in part to the low sample size resulting in variability in the mean age. Equally, other factors such as variability in activity, acuity and infection control practice may also have contributed. Given the marked variability in prevalence between institutions it seems unlikely that mean age alone could explain the difference. Until other risk factors have been accurately identified, the elderly should be considered to be at highest risk when developing strategies for the control of MRSA. The possibility of controlling MRSA in the health care setting was demonstrated quite early in Australia.[8] There is now ample and consistent evidence that infection control strategies based on screening, isolation and decolonisation are successful and highly cost effective.[19] The reasons for significant variability between regional and institutional prevalence of MRSA is worthy of further study. Reduction of MRSA infection in high prevalence institutions is likely to result in lower levels of morbidity and mortality and in lower health care costs.

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

## Acknowledgements

This study was fully supported by a grant from the Australian Government Department of Health and Ageing.

The AGAR participants were:

Joan Faoagali, Narelle George, QHPS, Royal Brisbane Hospital, Qld; Graeme Nimmo, Jacqueline Schooneveldt, QHPS, Princess Alexandra Hospital, Qld; Chris Coulter, Sonali Gribble, QHPS, Prince Charles Hospital, Qld; Dale Thorley, QHPS, Gold Coast Hospital, Qld; Enzo Binotto, Bronwyn Thomsett, QHPS, Cairns Hospital, Qld; Jenny Robson, Renee Bell, Sullivan Nicolaides Pathology, Qld; Peter Collignon, Susan Bradbury, The Canberra Hospital, ACT; John Ferguson, Jo Anderson, Hunter Area Pathology Service, NSW; Tom Gottlieb, Glenn Funnell, Concord Repatriation General Hospital, NSW; George Kotsiou, Clarence Fernandes, Royal North Shore Hospital, NSW; Richard Benn, Barbara Yan, Royal Prince Alfred Hospital, NSW; Iain Gosbell, Helen Ziochos, South Western Area Pathology Service, NSW; David Mitchell, Lee Thomas, Westmead Hospital, NSW; Samantha Ryder, James Branley, Nepean Hospital, NSW; Miriam Paul, Richard Jones, Douglass Hanly Moir Pathology, NSW; Denis Spelman, Clare Franklin, Alfred Hospital, Vic; Suzanne Garland, Gena Gonis, Royal Children's and Women's Hospitals, Vic; Mary Jo Waters, Linda Joyce, St Vincent's Hospital, Vic; Barrie Mayall, Peter Ward, Austin Health, Vic; John Andrew, Di Olden, Gribbles Pathology (Vic) Pty Ltd, Vic; Tony Korman, Despina Kotsanas, Monash Medical Centre, Vic; Alistair McGregor, Rob Peterson, Royal Hobart Hospital, Tas; Erika Cox, Kathy Wilcox, Launceston General Hospital, Tas; John Turnidge, Jan Bell, Women's and Children's Hospital, SA; Ivan Bastian, Rachael Pratt, Institute of Medical & Veterinary Science, SA; David Gordon, Hendrik Pruul, Flinders Medical Centre, SA; PC Lee, Barbara Koldej, Gribbles Pathology (SA), SA; Clay Gollege, Barbara Henderson, PathCentre, WA; Keryn Christiansen, Geoff Coombs, Julie Pearson, Royal Perth Hospital, WA; David McGechie, Graham Francis, Fremantle Hospital, WA; Sue Benson, Janine Fenton, St John of God Pathology, WA; Gary Lum, Paul Southwell, Royal Darwin Hospital, NT.

## Author details

Graeme R Nimmo, Director [1]

Julie C Pearson, Scientific Officer for the Australian Group on Antimicrobial Resistance[2]

Peter J Collignon, Director[3]

Keryn J Christiansen, Director [2]

Geoffrey W Coombs, Principal Scientist [2]

Jan M Bell, Senior Scientist [4]

Mary-Louise McLaws, Director [5]

1. Division of Microbiology, Herston, Queensland
2. Department of Microbiology and Infectious Diseases, PathWest Laboratory Medicine WA, Royal Perth Hospital, Western Australia
3. Infectious Diseases Unit and Microbiology Department, The Canberra Hospital, Garran, Australian Capital Territory
4. Department of Microbiology and Infectious Diseases, Women's and Children's Hospital, North Adelaide, South Australia
5. Hospital Infection Epidemiology and Surveillance Unit, University of New South Wales, Sydney, New South Wales

Corresponding author: Assoc. Professor GR Nimmo, Division of Microbiology, Queensland Health Pathology Service – 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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# Pneumonia cluster in a boarding school – implications for influenza control

Patrick Cashman, Peter Massey, David Durrheim, Fakhrul Islam, Tony Merritt, Keith Eastwood

## Abstract

*Streptococcus pneumoniae* is a common cause of community acquired pneumonia (CAP). Influenza infection increases susceptibility to *S. pneumoniae* infection in adults but this link is less well described in children. We report on an outbreak of CAP affecting 25 previously well adolescents in a New South Wales boarding school. *S. pneumoniae* 1 was confirmed in two cases. During this period, the school also experienced an influenza outbreak with an influenza-like illness attack rate peaking at 27% in Year 8 students. A planned school closure may have contributed to controlling the outbreak. Boarding schools are vulnerable to outbreaks of respiratory illness and strategies for limiting this risk are required. *Commun Dis Intell* 2007;31:296–298.

Keywords: *Streptococcus pneumoniae*, influenza, boarding school, school closure

## Introduction

*Streptococcus pneumoniae* is the most common cause of community acquired pneumonia (CAP).[1] Institutionalisation is a risk factor for pneumococcal clusters but these have generally been described in the elderly.[2] Serotype 1 has been associated with severe pneumonia in otherwise healthy children, has a propensity for invasive disease and has caused outbreaks in institutions.[3] This serotype remains highly susceptible to antibiotic therapy.[4]

Influenza infection frequently precedes pneumococcal pneumonia in adults but this relationship is less well documented in children.[3] Influenza virus may increase susceptibility to invasive pneumococcal disease through destroying the physical respiratory barrier, increasing virus adherence, decreasing mucociliary activity and disrupting immune system responses.[5]