AGAR community and hospital *Staphylococcus aureus* surveillance

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Point-prevalence antimicrobial surveillance programmes conducted by the Australian Group on Antimicrobial Resistance (AGAR) from 1986-1999 included consecutive clinical isolates of *Staphylococcus aureus* regardless of acquisition. Following a reported increase in community-acquired infections caused by methicillin-resistant *S. aureus* (MRSA) in the literature, AGAR performed the first survey of infections from outpatients, emergency department and general practitioner patients in 2000. Further community surveys were conducted in 2002, 2004 and 2006. In 2005 AGAR performed the first hospital-acquired infections survey (infections acquired more than 48 hours post admission) in part to track community MRSA clones emerging in the hospital setting. This article discusses the focus and main outcomes of the AGAR hospital and community surveys.

AGAR has conducted antimicrobial surveillance programmes of *S. aureus* infections in Australia since 1986. Initially, the medical microbiology laboratories in the major teaching hospitals were invited to participate in surveys; however, since 2000, the group has grown to include outer metropolitan and private laboratories. In 2008, 31 laboratories around Australia will contribute isolates and demographic data in AGAR surveys. Prior to 2005, susceptibility testing was performed by agar dilution against a range of commonly used antimicrobials. Since 2005, primary susceptibility testing has been determined by the Vitek2® automicrobial system using the AST-P545 susceptibility card. Additional disc diffusion and/or minimum inhibitory concentration (Etest®) testing is performed for cefoxitin, mupirocin and tigecycline. Since 2000, clone characterisation of all MRSA has been performed using a range of molecular methods.

AGAR has conducted community staphylococcal surveys in 2000, 2002, 2004 [1,2] and 2006 [unpublished data]. In these surveys each laboratory collected up to 100 consecutive clinical isolates from outpatient settings or from patients admitted to hospital less than 48 hours prior to specimen collection. Trend data shows that the proportion of *S. aureus* identified as MRSA in the outpatient population has increased in Australia (13% in 2000 to 16% in 2006, $p=0.0012$) particularly in New South Wales (21% to 25%, $p=0.0337$) and the Northern Territory (9% to 20%, $p=0.0264$). This increase has implications for the treatment of patients presenting at outpatients clinics, emergency departments and general practice.

The proportion of MRSA resistant to the non-ß-lactam antimicrobials (including erythromycin, clindamycin [constitutive resistance], tetracycline, gentamicin, fusidic acid, rifampicin and mupirocin), however, has generally decreased. This decrease is due to the clonal expansion of non-multi-resistant virulent community-associated MRSA in recent years which have replaced multi-resistant hospital-associated clones (AUS-2 and AUS-3 EMRSA, ST239-MRSA-III). For example, the Panton-Valentine leukocidin positive Queensland clone (ST93-MRSA-IV), which is generally susceptible to the non-ß-lactam antimicrobials, has increased from 5% to 19% of all MRSA strains.

Despite the declining numbers of ST239-MRSA-III, ciprofloxacin resistance has remained stable at approximately 50% due to the expansion of the ciprofloxacin-resistant EMRSA-15 (ST22-MRSA-IV) clone; from 10% of all MRSA in 2000 to 18% in 2006. In Australia, ciprofloxacin resistance remains a good indicator of epidemicity, with >90% of ciprofloxacin-resistant MRSA characterised as hospital-associated clones. Methicillin-susceptible *S. aureus* (MSSA) were generally susceptible to the non-ß-lactam antimicrobials (<5% resistance in all surveys), with the exception of erythromycin (11.1% in 2006). Resistance to the topical agent mupirocin remained low at 2% of all *S. aureus*, of which 50% had high level resistance (MIC >256mg/L). No resistance was detected to vancomycin, teicoplanin, linezolid or quinupristin/dalfopristin in MSSA or MRSA. Tigecycline was tested for the first time in 2006; resistance, as defined by EUCAST and FDA guidelines, was detected in 0.2% of isolates.

An AGAR hospital-based staphylococcal antimicrobial surveillance programme was conducted in 2005 [3]. Only isolates from clinical specimens collected at least 48 hours after hospital admission were included. This survey was the first national study of the resistance profiles of *S. aureus* causing infections in hospital inpatients. The proportion of isolates that were identified as MRSA varied considerably between institutions and was most likely a result of local infection control practices and the mean age of the patient population (increasing age was a demonstrated risk factor for MRSA). As expected, the proportion of *S. aureus*

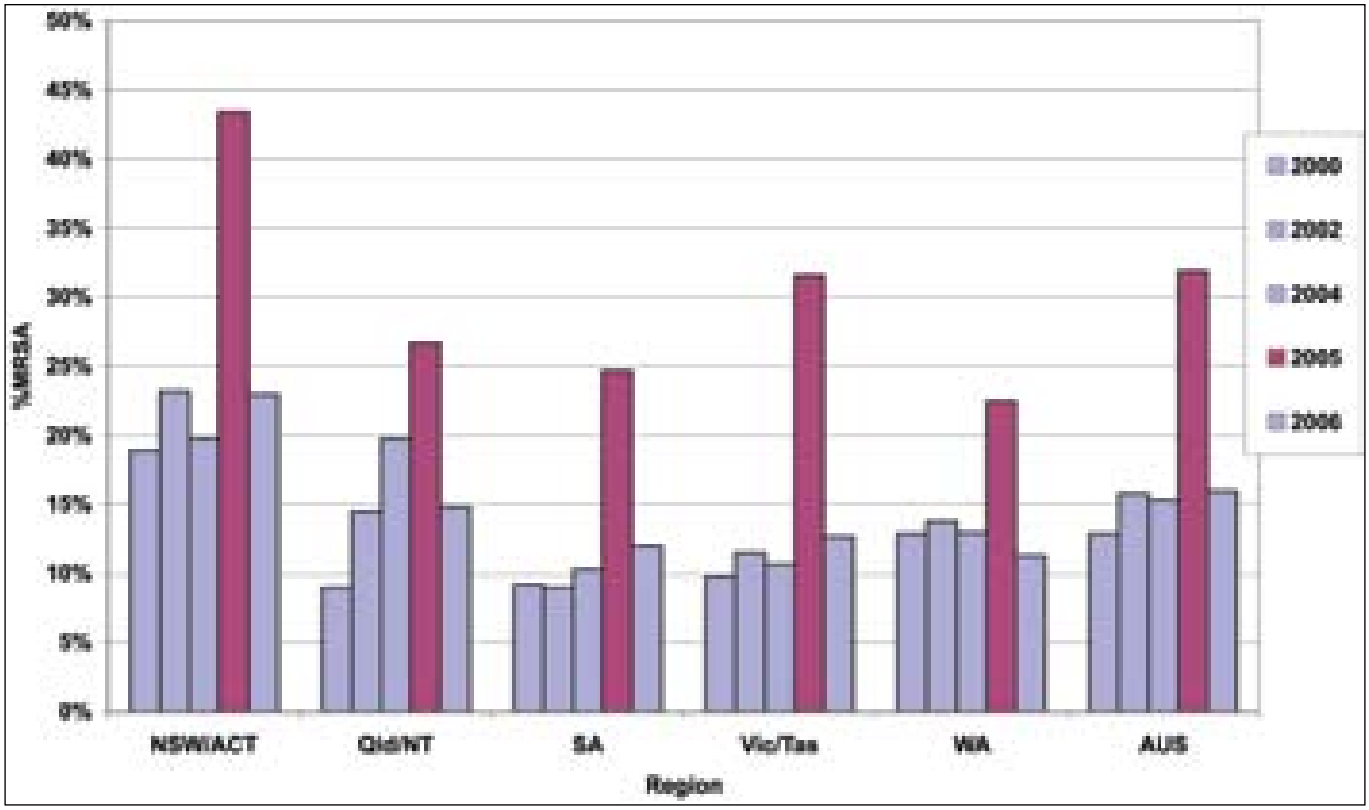

Figure 1. The proportion of *S. aureus* that are MRSA by region for the community surveys (2000, 2002, 2004 and 2006) and the hospital inpatients survey (2005).

that were MRSA was considerably higher in the inpatient population compared with the outpatient population; 32% in the 2005 hospital survey compared with 16% in the 2006 community survey (Figure 1).

In MRSA, resistance to the non-ß-lactam antimicrobials was common (approx 60% for tetracycline, co-trimoxazole and gentamicin and approximately 80% for erythromycin and ciprofloxacin). However, considerable regional variability was reported, primarily due to the differences in distribution of ST293-MRSA-III which was the predominant clone (63% of all MRSA). ST22-MRSA-IV was the second most common clone which accounted for 16% of all MRSA. More than 20% of MRSA in hospital inpatients were characterised as community MRSA clones. Resistance in MSSA was not significantly different from the rates seen in the community surveys. No resistance was detected to vancomycin, teicoplanin or linezolid in MSSA or MRSA. One MRSA was resistant to quinupristin/dalfopristin.

Annual susceptibility testing of large numbers of clinical *S. aureus* such as those conducted by AGAR provide a longitudinal record of the changing susceptibility profiles of *S. aureus* at a national and regional level. By performing alternate community and hospital surveys, AGAR provides important clinical information to hospital clinicians, general practitioners, infection control specialists and government departments. Of particular importance is the documented increase in MRSA in the community, particularly in patients without recent hospital contact. By performing molecular typing, AGAR records the epidemiology of MRSA and documents the emergence and spread of epidemic and virulent MRSA community strains isolated in Australia. Detailed AGAR reports can be accessed via the AGAR website [4].

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