*Original Report*

# Emergence of community-acquired methicillin-resistant *Staphylococcus aureus* (MRSA) infection in Queensland, Australia

Wendy J. Munckhof,[1] Jacqueline Schooneveldt,[2] Geoffrey W. Coombs,[3] Jane Hoare[4] and Graeme R. Nimmo[2]

**Objectives:** To investigate the incidence and epidemiology of non-multiresistant methicillin-resistant *Staphylococcus aureus* (nmMRSA) infection in south-east Queensland, Australia.

**Study design:** A retrospective survey was done of hospital records of all patients who had non-multiresistant MRSA isolated at Ipswich Hospital (a 250-bed general hospital, 40 km south-west of Brisbane, Queensland, Australia) between March 2000 and June 2001. Laboratory typing of these isolates was done with antibiogram, pulsed-field gel electrophoresis, bacteriophage typing and coagulase gene typing.

**Results:** There were 44 infections caused by nmMRSA. Seventeen infections (39%) occurred in patients from the south-west Pacific Islands (predominantly Samoa, Tonga and New Zealand). Laboratory typing showed that the isolates in Pacific Islanders were Pacific Island strains, and 16/17 of these infections were community acquired. Twenty-three infections (52%) occurred in Caucasians. Eleven of the isolates from Caucasians (48%) were a new predominantly community-acquired strain that we have termed the 'R' pulsotype, nine (39%) were Pacific Island strains, and three (13%) were health care institution-associated strains. Four infections occurred in patients who were not Caucasians or Pacific Islanders. Overall, 34 of all 44 infections (77%) were community acquired.

**Conclusions:** Non-multiresistant MRSA infection, relatively frequently observed in Pacific Islanders in south-east Queensland, is now a risk for Caucasians as well, and is usually community acquired. Clinicians should consider taking microbiological specimens for culture and antimicrobial susceptibility testing in patients with suspected staphylococcal infections who are not responding to empirical therapy with β-lactam antibiotics.

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## INTRODUCTION

Methicillin-resistant *Staphylococcus aureus* (MRSA) has been recognized since the 1970s as an important cause of nosocomial infection in Australian hospitals.[1] In the last 10 years, community-acquired strains of MRSA have arisen in many countries, including the USA,[2–4] Canada,[5] and New Zealand.[6,7] In the late 1980s, the first Australian cases of community-acquired MRSA infection were reported in the remote Kimberley region of Western Australia.[8,9] These strains were dubbed 'WA MRSA', and colonization rates in remote Aboriginal communities are high.[10,11] All strains of MRSA are resistant to all β-lactam antibiotics, and nosocomial strains of MRSA in most countries, including Australia, are usually resistant in vitro to many non-β-lactam antibiotics.[1,4,12] However, community-acquired MRSA strains in most countries, including Australia, are typically non-multiresistant.[1,3–5]

In the last 5 years, a new variety of non-multiresistant MRSA (nmMRSA) has emerged in Australia, particularly along the eastern seaboard.[13–15] Infections are usually community acquired, and occur predominantly in people from the south-western Pacific Islands, such as Samoa and Tonga. These strains have Western Samoan phage patterns (WSPP) on bacteriophage typing, and typing by pulsed-field gel electrophoresis (PFGE) shows a typical 'A' pulsotype.[14] Similar strains have caused community-acquired infections in Pacific Islanders in New Zealand.[6,7]

This study was performed because the incidence of infections due to community-acquired nmMRSA appeared to be increasing at Ipswich Hospital, particularly in Caucasians. If the incidence of this infection were high enough, it would alter the choice of empirical

[1]Infection Management Services, [2]Microbiology Department, and [3]Department of Medicine, Princess Alexandra Hospital, Brisbane, Queensland, Australia; [4]Department of Microbiology and Infectious Diseases, Royal Perth Hospital, Perth, Western Australia, Australia.

Address correspondence to: Dr Wendy J. Munckhof, Infection Management Services, Princess Alexandra Hospital, Ipswich Road, Woolloongabba, Brisbane, Queensland, Australia, 4102.

E-mail: wendy_munckhof@health.qld.gov.au

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therapy in severe community-acquired staphylococcal sepsis, for which β-lactam antibiotics represent the usual current therapy of choice. Therefore, the aim of the current study was to determine the relative incidence of nmMRSA in Polynesian, Caucasian and other ethnic groups, and to determine if the infections were likely to be nosocomially or community acquired. We also typed the strains of nmMRSA to determine if they were clonal and/or related to previously described strains of nmMRSA.

## METHODS

### Patients and setting

Ipswich Hospital is a general hospital of 250 beds, 40 km south-west of Brisbane in south-east Queensland, Australia. It services a population of approximately 125 000 people, 3% of whom of were born in the south-western Pacific Islands (predominantly Tonga, Samoa, Fiji, and the Cook Islands) or New Zealand.[16]

We performed a retrospective medical record review of all patients who had nmMRSA isolated from clinical specimens sent to the Ipswich Hospital microbiology laboratory during a 16-month period between 1 March 2000 and 30 June 2001. MRSA isolates were classified as non-multiresistant if they tested resistant to two or fewer classes of non-β-lactam antibiotics (gentamicin, erythromycin, clindamycin, tetracycline, cotrimoxazole, ciprofloxacin, rifampicin, fusidic acid, vancomycin) in addition to β-lactams.[1]

### Data collection

The medical records of all patients who had nmMRSA isolated from clinical specimens during the study period were available and were reviewed. The following characteristics were identified in the medical record review: demographics, including place of birth and ethnicity, date of treatment, coexisting morbidities, risk factors for acquisition of MRSA in the previous 12 months, clinical manifestations of infection, and outcome (duration of hospital admission, need for surgery, whether discharged to usual residence, whether outcome was fatal, treatment with antibiotics with and without in vitro activity against MRSA).

Classification of infections as community acquired or nosocomial (hospital, hospital-based unit, nursing home or other long-term care institution) was in accordance with Centers for Disease Control definitions.[17] Infections were classified as:

1. Community-acquired if *S. aureus* was isolated from a specimen taken within 48 h of hospital presentation, and
2. With no risk factors if the patient had not attended a hospital or hospital-based unit, or resided in a nursing home or other long-term care institution, within the 12 months before presentation, and did not have a predisposing chronic disease (including diabetes, liver disease, renal failure, malignancy, HIV infection, intravenous drug use or corticosteroid therapy).

### Laboratory analysis and typing of isolates

*S. aureus* was identified by the Slidex Staph-Kit latex agglutination test (bioMerieux, Lyon, France) and by detection of the *nuc* gene, and oxacillin (methicillin) resistance was confirmed by detection of the *mecA* gene. The multiplex PCR procedure used was based on a modification of the method of Unal et al,[18] the *mecA* primers were as described by Murakami and Minamide,[19] and the *nuc* primers were as described by Brakstad et al.[20] The 25-μL reaction mixture consisted of 10 μL of lysate, 100 μM (each) deoxynucleoside triphosphate, 0.2 μM (each) primer, and 0.5 U of AmpliTaq DNA polymerase (Perkin Elmer, Roche Molecular Systems Inc., NJ, USA) in 10× PCR buffer (1× is 10 mM Tris-HCl (pH 8.3), 50 mM KCl, 1.5 mM $MgCl_2$, 0.001% (w/v) gelatin). DNA amplification consisted of an initial cycle of 94°C for 5 min, 55°C for 30 s, and 72°C for 2 min, followed by 29 cycles of 94°C for 30 s, 55°C for 30 s, and 72°C for 2 min. PCR products were visualized on 2% agarose gels stained with ethidium bromide. Antimicrobial susceptibility testing was performed on the isolates using the VITEK GPS-IX card (bioMerieux-Vitek, Hazelwood, MO, USA), and was confirmed using standard National Committee for Clinical Laboratory Standards disk methodology.[21,22]

Typing was performed on isolates from all patients of non-Pacific Island ethnicity. Seven of the 17 patients of Pacific Island ethnicity had isolates typed. These seven isolates from Pacific Islanders were taken randomly throughout the study, and yielded identical typing results to those previously published for this ethnic group.[14] For this reason, and because typing is very expensive and time-consuming, not every patient in this ethnic group had an isolate tested. Isolates were typed with the following four methods: antibiotyping using the antimicrobial susceptibility testing pattern, PFGE, bacteriophage typing, and coagulase gene typing by restriction analysis of PCR products.

#### *Fingerprinting by pulsed-field gel electrophoresis*

PFGE of chromosomal DNA was performed using the enzyme *Sma*I. DNA was separated on a GenePath system (Bio-Rad, Hercules, CA, USA), using the GenePath group 1 reagent kit (Bio-Rad) with initial pulse times of 5.3 and 34.9 s at the end of the 20-h run. Gels were stained with ethidium bromide and photographed under UV illumination. The patterns were confirmed visually using the criteria of Tenover et al,[23] and were analyzed with GelCompar software (Applied Maths, Kortrijk, Belgium). Results were analyzed using the

unweighted pair group method for arithmetic averages and the Dice coefficient with 1.2% band tolerance.[24]

*Bacteriophage typing*

Phage typing was performed using the method of Blair and Williams.[25] The following phage-typing sets were used: the Basic International Set of Typing Phages (23 phages), the International Set of Experimental Phages for MRSA (10 phages),[26] and the Australian Set of Experimental Phages for MRSA (nine phages).[27] All phages were used at 100× routine test dilution.

*Coagulase gene typing by restriction analysis of PCR products*

Molecular typing on the basis of coagulase gene polymorphisms was performed by a modification of the method of Goh et al.[28]

### Relative incidence of methicillin and gentamicin resistance in *Staphylococcus aureus*

We used the laboratory information system database to locate all patient isolates of *S. aureus* at the Ipswich Hospital microbiology laboratory between 1 March 2000 and 30 June 2001. Because in vitro resistance to gentamicin is a good surrogate marker of nosocomial acquisition of MRSA, and conversely community-acquired strains of MRSA are usually gentamicin susceptible in vitro,[1,13–15,29] we used gentamicin susceptibility as a surrogate marker of community acquisition of strains.

## RESULTS

Forty-four cases of infection caused by nmMRSA were identified from clinical specimens sent to the Ipswich Hospital microbiology laboratory.

### Patient characteristics and clinical manifestations of infection

The medical records of all 44 patients were reviewed. Patient characteristics are shown in Table 1. Most patients were born in Australia, New Zealand or the Pacific Islands. Thirty-two of the 44 patients (73%) were male, and this sex bias was particularly noted in the Pacific Islander group. Of note is the youth of many patients, with 41% of Pacific Islanders and 31% of Caucasians aged less than 20 years. Seventy percent of all patients presented with superficial abscesses or boils, and there were four serious life-threatening infections. Thirty-four of the 44 patients were hospitalized, with nine patients being hospitalized for more than 1 week. Figure 1 shows the incidence of infections for the various ethnic groups over the study period.

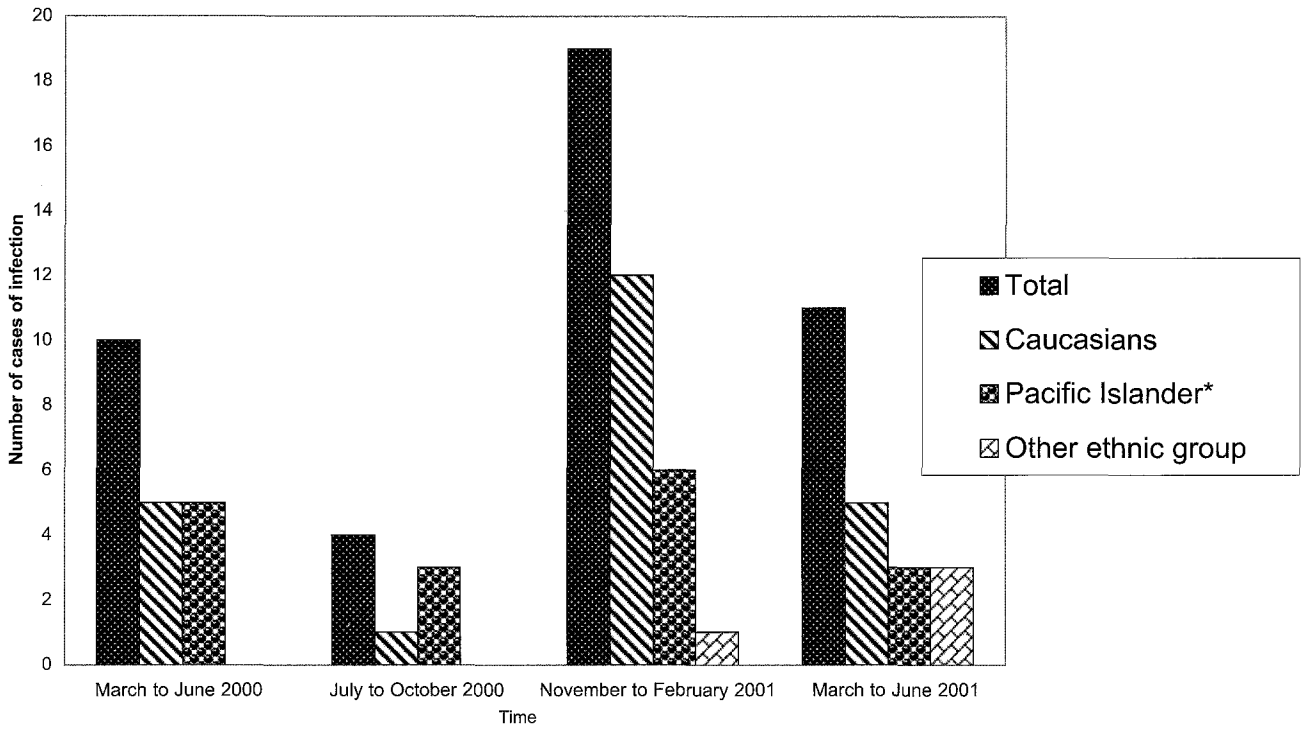
### Antibiotic resistance

Forty-one of the 44 strains of nmMRSA were susceptible in vitro to all of the non-β-lactam antibiotics tested. In vitro non-β-lactam antibiotic resistance was found in three strains that were resistant to erythromycin, and one of these strains was also resistant to clindamycin. We have previously described erythromycin and inducible clindamycin resistance in two Pacific Islanders from

**Table 1.** Characteristics of 44 patients infected with non-multiresistant MRSA at Ipswich Hospital, by ethnic group

| | *Caucasian* | *South-west Pacific Islander*[a] | *Other ethnicity* | *Total* |
|---|---|---|---|---|
| Demographic characteristics | | | | |
| Number of cases of infection | 23 | 17 | 4 | 44 |
| Male | 15/23 (65%) | 15/17 (88%) | 2/4 (50%) | 32/44 (73%) |
| Median age in years (range) | 31.5 | 34.5 | 23.3 | 33 |
| Place of birth | | | | |
| Australia | 20 | 5 | 3 | 28 |
| New Zealand | 2 | 4 | 0 | 6 |
| South-west Pacific Islands[a] | 0 | 8 | 0 | 8 |
| Other country | 1 | 0 | 1 | 2 |
| Nature of infection | | | | |
| Boil/superficial abscess | 14 | 14 | 3 | 31 |
| Cellulitis | 5 | 2 | 1 | 8 |
| Surgical wound infection | 1 | 0 | 0 | 1 |
| Septic arthritis | 0 | 1 | 0 | 1 |
| Diabetic foot osteomyelitis | 1 | 0 | 0 | 1 |
| Septicemia | 1 | 0 | 0 | 1 |
| Septicemia and osteomyelitis | 1 | 0 | 0 | 1 |
| Pulsotype by pulsed-field gel electrophoresis | | | | |
| A pulsotype (Pacific Island type) | 9 | 7[b] | 2 | 18 |
| R pulsotype (new strain) | 11 | 0 | 2 | 13 |
| Other strains | 3 | 0 | 0 | 3 |

[a]South-west Pacific Islander denotes person of south-west Pacific Island ethnicity, predominantly Tongan or Samoan.
[b]Typing by PFGE was performed on seven of the 17 patients of Pacific Island ethnicity.




*Pacific Islander denotes person of south-western Pacific Island ethnicity, predominantly Tonga or Western Samoa.

**Figure 1.** Frequency of cases of non-multiresistant MRSA infection at Ipswich Hospital, by ethnic group.

Ipswich with community-acquired MRSA infection,[30] but there were no further isolates of this strain.

## Typing results and risk factors for acquisition of MRSA

### *Pacific Islanders*

There were 17 isolates of nmMRSA obtained from Pacific Islanders, and all were resistant to β-lactam antibiotics only. All seven isolates that were typed were pulsotype A pattern on PFGE (Figure 2), typical of the Pacific Island strain.[14] Sixteen of the 17 Pacific Islanders had community-acquired infections. All three patients with community-acquired infections who had risk factors for acquisition of MRSA had diabetes mellitus.

### *Caucasians*

There were 23 isolates of nmMRSA in Caucasians (Table 1), which fell into three distinct groups on typing by PFGE:

#### GROUP 1: 'PACIFIC ISLAND PATTERN'

Nine strains in Caucasians were of the pulsotype A Pacific Island pattern on PFGE (Figure 2). These isolates were resistant only to β-lactam antibiotics. Six of these strains were community acquired in patients without risk factors for acquisition of MRSA, and the other three strains were hospital associated.

#### GROUP 2: NEW STRAIN 'R PULSOTYPE'

Typing results for 11 isolates from Caucasians showed a clonal pattern on PFGE, which we have termed the 'R' pulsotype[21] (Figure 2). This PFGE pattern is new to Queensland,[31] and was not seen in a 1997–98 Brisbane study of MRSA.[14] These isolates were also non-typable on phage typing. All isolates were resistant to β-lactam antibiotics only, with the exception of one early erythromycin- and clindamycin-resistant isolate. Seven strains were community acquired in patients with no risk factors for acquisition of MRSA, one community-acquired strain occurred in an intravenous drug user, and the other three strains were hospital associated. All three hospital-acquired infections occurred within the same month in patients admitted to the surgical ward at the hospital. The index case of this cluster was a long-term psychiatric hospital inmate. All R pulsotype strains were isolated in the last 9 months of the 16-month study.

#### GROUP 3: MIXED HEALTH CARE INSTITUTION-ASSOCIATED STRAINS

The last three isolates in Caucasians had three different patterns on PFGE and had additional in vitro

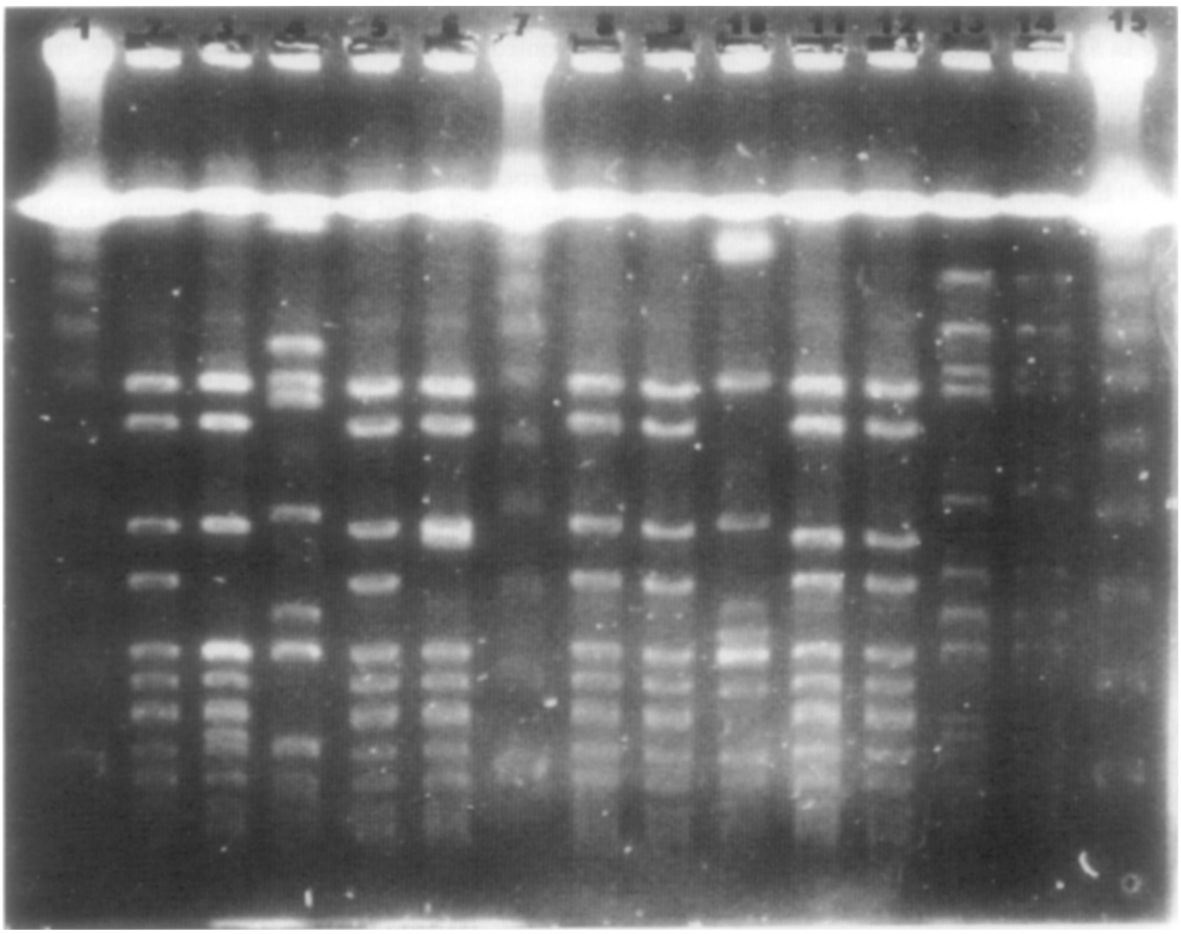


**Figure 2.** Pulsed-field gel electrophoresis (PFGE) of strains of MRSA isolated in south-east Queensland, Australia. Lanes 1, 7 and 15: size markers. Lanes 2 and 3: A pulsotype community-acquired strains isolated from Pacific Islanders at Princess Alexandra Hospital, Brisbane, Queensland, Australia. Lane 4: nosocomially acquired strain from a Caucasian isolated at Princess Alexandra Hospital, Brisbane. Lanes 5, 6, 11 and 12: A pulsotype community-acquired Ipswich strains isolated from Pacific Islanders. Lanes 8 and 9: A pulsotype community-acquired Ipswich strains isolated from Caucasians. Lane 10: new R pulsotype community-acquired Ipswich strains isolated from a Caucasian. Lanes 13 and 14: nosocomially acquired Ipswich strains isolated in Caucasians.

non-β-lactam antibiotic resistances (erythromycin, clindamycin). All three strains were hospital associated.

*Other ethnic groups*

There were two isolates of the new 'R' pulsotype obtained from an Aboriginal and a Torres Strait Islander. These infections were community acquired in patients with no risk factors for acquisition of MRSA and occurred towards the end of the study, after the first isolates of this strain in Caucasians. There were also two community-acquired infections in Africans, one of whom had diabetes mellitus, which were of pulsotype A (Pacific Island pattern) on typing with PFGE.

## Outcomes

There were no deaths, and all patients were discharged to their usual residences. Thirty of 44 patients underwent surgical treatment, with 25 of the 31 patients with superficial abscesses or boils being treated with incision and drainage. All 44 patients were treated with antibiotics. Twenty-five of the 44 patients were treated only with β-lactam antibiotics, which have no activity against MRSA (penicillin, flucloxacillin, dicloxacillin, ampicillin, ampicillin–clavulanic acid, cephalexin, and ceftriaxone). The other 19 patients mostly received a β-lactam antibiotic until MRSA was cultured, and were then changed to antibiotics with in vitro activity against MRSA. These antibiotics included intravenous vancomycin (seven patients), intravenous vancomycin followed by oral clindamycin (two patients), intravenous vancomycin followed by oral clindamycin followed by oral rifampicin and fusidic acid (one patient), intravenous vancomycin with concurrent oral rifampicin (one patient), intravenous vancomycin followed by oral erythromycin (one patient), oral erythromycin (five patients), and oral clindamycin (two patients).

### Relative incidence of methicillin and gentamicin resistance in *Staphylococcus aureus*

By using the laboratory information system database, we located all patient isolates of *S. aureus* at the Ipswich Hospital microbiology laboratory between 1 March 2000 and 30 June 2001 (Figure 3). We noted that the total monthly isolation rate of *S. aureus* did not change significantly, and that methicillin-susceptible strains comprised between 73% and 92% of all *S. aureus* strains isolated, depending on the month. Gentamicin-resistant MRSA, predominantly nosocomially acquired in patients transferred to Ipswich Hospital from Brisbane tertiary hospitals, comprised 3–19% of all *S. aureus* strains isolated. Gentamicin-susceptible strains of MRSA, a surrogate marker of community acquisition of MRSA, comprised 4–16% of all isolates of *S. aureus,* with some variability from month to month, but concluding at a rate of 10% of *S. aureus* at the end of the study.

## DISCUSSION

MRSA was traditionally considered to be a nosocomial pathogen until the emergence of community-acquired MRSA in Australia, initially in the 1980s from Western Australia.[1,8] The classification of acquisition status in the study of community-acquired MRSA infection is controversial. Contact with a health care institution in the 12 months prior to presentation has been identified as the most important risk factor for MRSA carriage,[32] but other factors, such as the presence of preexisting chronic disease, are also important.[33] For this reason, we have subdivided apparently community-acquired cases into those with and without risk factors for MRSA acquisition. Only five of the 34 apparently community-acquired cases in our study occurred in patients with chronic diseases. Hence, we consider that most cases of nmMRSA in our study were truly community acquired.

Community-acquired MRSA infections have previously been identified as a significant problem for the Pacific Islander community in eastern Australia,[1,13–15,29,30] and in Australia there is an increasing awareness of these infections in this ethnic group. Fourteen patients in our study (32%) were born in the south-western Pacific Islands or New Zealand, compared to 3% of the Ipswich population.[16] Hence, there is a roughly 10-fold increased risk of nmMRSA infection in Pacific Islanders or New Zealanders in the Ipswich area compared to the general population. A similar rate of increased risk in Pacific Islanders and New Zealanders was noted in a study from Sydney, Australia.[15]

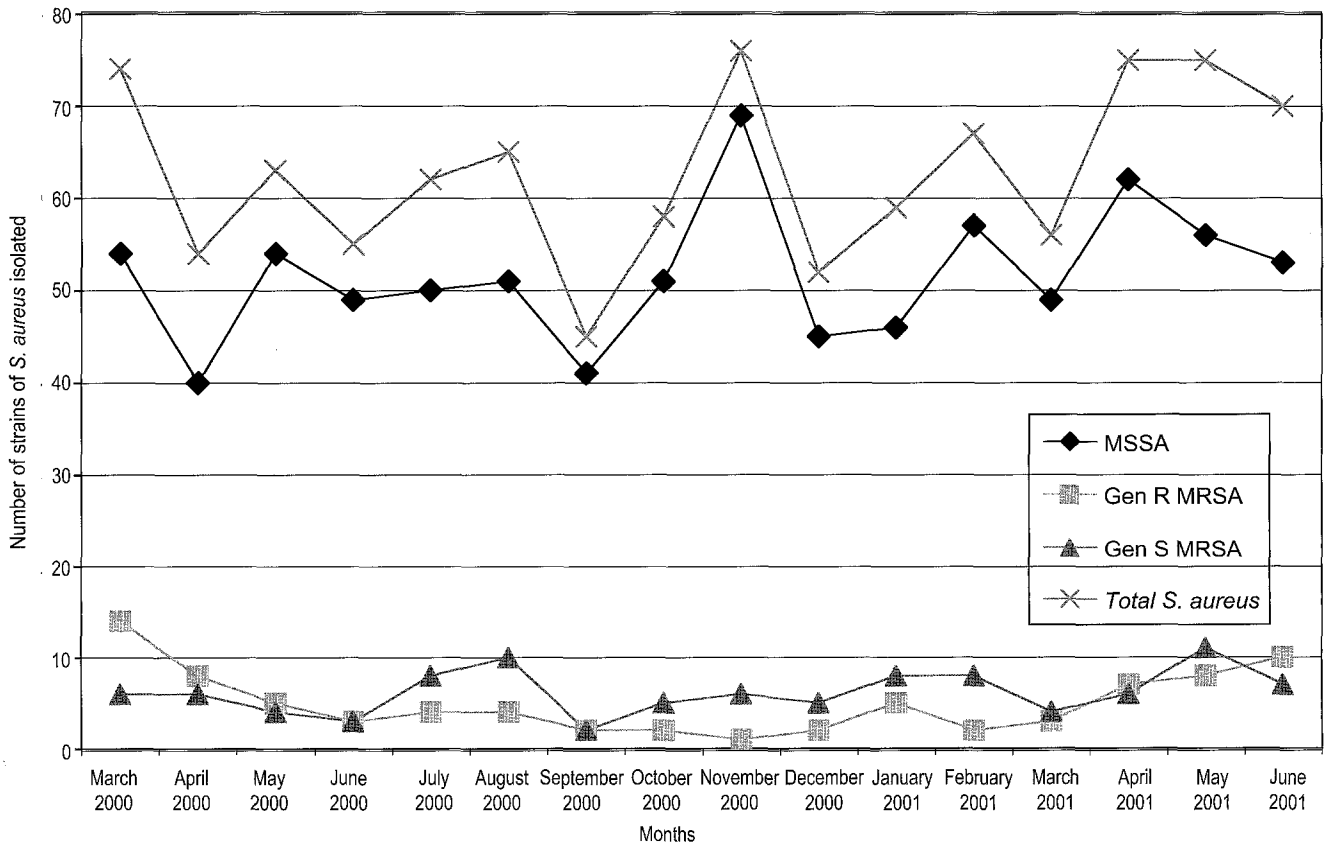



**Figure 3.** Incidence and types of *S. aureus* isolated at Ipswich Hospital microbiology laboratory. MSSA, methicillin-susceptible *S. aureus*; Gen R MRSA, gentamicin-resistant MRSA (predominantly nosocomially acquired strains); Gen S MRSA, gentamicin-susceptible MRSA (predominantly community-acquired strains).

Community-acquired MRSA infections have only relatively recently emerged in the Caucasian population in Australia.[11,14,15] In our study, roughly half of the nmMRSA infections in Caucasians were due to the Pacific Island strain, and half were due to the emergence of the new strain that we have termed the R pulsotype. We note, however, that the proportion of nmMRSA in the Caucasian population rose from 45% in the first 6 months of the study, to 80% in the last 6 months of the study. This was primarily due to the emergence of the R pulsotype strain, which has not previously been reported from Queensland,[14,31] and which was predominantly community acquired. Unfortunately, we are unable to provide accurate figures on the incidence of nmMRSA prior to the year 2000, as these files are no longer available on our laboratory information system database.

The rising incidence of community-acquired MRSA infections raises the question of whether to isolate or cohort these patients on admission to hospital, particularly if they have open skin lesions. Isolation or cohorting is a common practice in some Australian states, to prevent cross-transmission of nosocomial multiresistant strains of MRSA, and the question of whether to do the same for community-acquired strains has not been well addressed in the current literature. A nosocomial outbreak of infection due to cross-transmission of the community-acquired WA-MRSA strain has been reported,[11] but reports of nosocomial cross-transmission of community-acquired MRSA strains are much less frequent than those of the well-documented cross-transmission of nosocomial strains. In our study, there was no evidence of cross-transmission within the hospital of the Pacific Island strain, but there was a small cluster of apparent nosocomial cross-transmission of the R pulsotype strain. Since that time, it has been our practice to admit patients with open skin infections due to community MRSA into single rooms wherever possible, and we have not noted any further episodes of cross-transmission.

In our study, 70% of patients with nmMRSA infection had superficial abscesses or boils. These infections were the predominant manifestation of community MRSA in other Australian studies.[14,15] Because most infections occurred in a community setting and because the incidence of community MRSA in Australia is low, most patients were empirically treated with β-lactam antibiotics. Twenty-five of the 44 patients in our study received only β-lactam antibiotics, which are not active against MRSA. Most of these patients were seen in the casualty department and were not reviewed after MRSA was cultured. Seventeen of the 25 patients who received non-active antibiotics had superficial abscesses or boils that were surgically incised and drained. It is likely that these patients were cured with surgery alone. The other eight patients who received non-active antibiotics had minor infections that did not require admission, such as cellulitis.

The 19 patients in our study who received antibiotics that are active in vitro against MRSA mostly received parenteral vancomycin, or oral macrolides (clindamycin or erythromycin). The limited data available on the treatment of nmMRSA infections are difficult to interpret, because many infections respond to drainage alone, as illustrated by many of our cases. Most publications either extrapolate from non-β-lactam therapy of methicillin-susceptible *S. aureus* infection or consist of anecdotal reports.[34] In Australia, treatment of nosocomial multiresistant strains of MRSA is with parenteral vancomycin or combination oral therapy, usually with rifampicin and fusidic acid (if strains are susceptible in vitro to these agents).[34] Monotherapy with oral antibiotics for nosocomial multiresistant MRSA strains has been associated with the rapid emergence of resistance, with, for example, up to 70% of Australian strains of nosocomial MRSA now resistant to ciprofloxacin.[1] At present, there are few clinical trials to show whether non-multiresistant strains of MRSA can be treated with antibiotics that test sensitive in vitro, but that have not traditionally been used to treat MRSA infections, such as clindamycin. There are anecdotal reports of the efficacy of clindamycin, a bacteriostatic agent, for the treatment of mild-to-moderate infections caused by community-acquired MRSA,[34,35] and it is an effective therapeutic agent in methicillin-susceptible *S. aureus* infections.[36] In general, we would not recommend the use of clindamycin in erythromycin-resistant strains, as these strains may have inducible $MLS_B$ resistance to other macrolides such as clindamycin,[29,37] and failure of clindamycin therapy has been reported for such strains.[37] We would also generally avoid bacteriostatic agents such as clindamycin for serious infections, such as endocarditis or septicemia.

There are also limited data available on the treatment of serious life-threatening nmMRSA infections.[34] All four patients in our study with serious life-threatening infections (Table 1) were cured with parenteral vancomycin, which is generally considered the gold standard therapeutic agent for MRSA infection. We caution, however, that the widespread empirical use of vancomycin may hasten the emergence of vancomycin resistance in staphylococci and other Gram-positive bacteria, such as enterococci.[38] Hence, the empirical use of vancomycin for community-acquired staphylococcal infections should be avoided, unless the prevalence of community-acquired MRSA exceeds an acceptable threshold. We consider that the current rate of approximately 10% of all *S. aureus* strains isolated at the Ipswich Hospital is too low to lead to a recommendation of a change from empirical β-lactam therapy of suspected staphylococcal infection to other non-β-lactam therapies, but we plan to periodically review these rates and antibiotic policies in the future. It should also be noted that preliminary data suggest that current rates of community MRSA in other parts of Australia are less than this figure,[31] and, based

on our knowledge of these data, we would not advocate a change to the empirical therapy of staphylococcal infection in other parts of Australia at present.

Thus, there is now evidence that community-acquired MRSA infection, relatively frequently observed in the Pacific Islander community of south-east Queensland, is a risk to the Caucasian community as well. Clinicians should be aware of the presence of this organism, and should consider taking microbiological specimens from patients with suspected staphylococcal infections who are not responding to empirical therapy with β-lactam antibiotics.

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