Epidemiol. Infect. (2005), **133**, 899–904. © 2005 Cambridge University Press
doi:10.1017/S0950268805004024 Printed in the United Kingdom

A decade of community MRSA in New Zealand

J. M. B. SMITH* AND G. M. COOK

Department of Microbiology and Immunology, Otago School of Medical Sciences, University of Otago, Dunedin, New Zealand

(*Accepted 19 January 2005*)

SUMMARY

In 1992, isolates with a distinctive phage pattern were identified amongst the 186 MRSA recovered in New Zealand. These unusual isolates were recovered in the Auckland region from individuals who came from or had visited Western Samoa, and were called Western Samoan phage pattern (WSPP) MRSA. They were almost exclusively community based and were mainly responsible for the alarming 15-fold increase in MRSA seen in New Zealand over the next 6 years. Since 2000, the number of infections attributable to WSPP MRSA appears to be declining. WSPP isolates are clonal, possess a unique type IV SCC*mec* element, and a distinctive multilocus sequence allelic profile (ST30). WSPP isolates are invariably not multiresistant with methicillin MICs generally $\leqslant 32\,\mu$g/ml. Virulence of the WSPP clone appears to be related to its adhesive and consistent toxin- (e.g. Panton–Valentine leukocidin, α- and γ-haemolysins) producing capabilities. Isolates are most frequently associated with cutaneous lesions in younger age groups. Since 1998, MRSA isolates belonging to the UK-derived EMRSA-15 strain (also type IV SCC*mec*) have continued to increase in New Zealand, and together with WSPP, these strains now dominate MRSA isolations in New Zealand.

INTRODUCTION

As in many other countries, New Zealand has a National Reference Laboratory where all unusual microbes are sent for further investigation. Prior to 1999, all potentially methicillin-resistant *Staphylococcus aureus* (MRSA) recovered in microbiological laboratories (both hospital and community) from overt disease and from MRSA screening tests were submitted to the Reference Laboratory for further testing which included phage typing, methicillin/oxacillin minimum inhibitory concentration (MIC) confirmation, and general antimicrobial sensitivity testing. The national coverage and compliance with this scheme were excellent, which has allowed a detailed account of the changing epidemiological features of MRSA in New Zealand to be obtained. Results are published on a regular basis in local publications from the Communicable Disease Group, Institute of Environmental Science and Research (ESR) (e.g. *ESR LabLink*, *New Zealand Public Health Report*, *Weekly MRSA Report*) or in other local publications (e.g. *New Zealand Medical Journal*, *New Ethicals Journal*, *Proceedings of the University of Otago Medical School Research Society*). Unfortunately, access to these publications worldwide is limited and many of the unique features of New Zealand's MRSA saga have gone largely unnoticed by the rest of the world.

* Author for correspondence.
(Email: sandy.smith@stonebow.otago.ac.nz)

Historical aspects of MRSA in New Zealand

MRSA was first isolated in the Auckland region of New Zealand in 1975 [1]. Over the next 10 years,

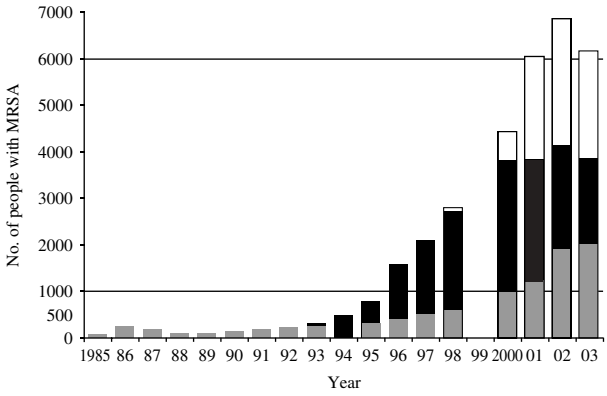

Fig. 1. Total numbers of MRSA belonging to WSPP (■), EMRSA-15 (□) or other clones (▩) isolated in New Zealand, 1985–2003. From 2000, numbers have been estimated from the results of a 1-month nationwide survey. No figures are available for 1999.

isolates remained uncommon (<20 per year) and were predominantly hospital based [2]. These high-level methicillin-resistant *S. aureus* strains were classically multiresistant (mMRSA – apart from β-lactams, resistant to at least two other classes of antimicrobials), and in most cases seemed to have been imported from the eastern seaboard of Australia or the United Kingdom. During 1986–87 there was a 10-fold increase in isolates of MRSA as New Zealand hospitals experienced their first MRSA outbreaks [3, 4]. Following a moderate decline in 1988 and 1989, numbers of MRSA in New Zealand continued to increase until 2002 (Fig. 1), and currently (2003) stand at just under 165/100 000 population (New Zealand currently has a population of a little over 4 million). It was also evident during the early 1985–1990 period that a significant proportion (>50%) of MRSA displayed low-level resistance to methicillin (MICs 16–32 μg/ml). These isolates were invariably non-multiresistant and were often recovered from individuals with no evidence of recent overseas travel [4, 5]. During 1991–94, it also became apparent that while the majority (72%) of patients from whom MRSA was isolated were categorized as hospital patients, the proportion of patients who were community based (i.e. patients who were not reported to have been in hospital, long-term care facility or rest home in the previous 6 months) was increasing −35% in 1994 compared to 19% in 1991 [6]. In addition, while overseas travel in the previous 12 months was a feature of almost half of the individuals from whom MRSA was isolated in the 1985–1987 period, this was only noted in ∼10% of infected/colonized patients in the 1991–94 period.

In 1992, MRSA strains with a distinctive phage pattern were recovered from individuals in the Auckland region who came from or had visited Western Samoa. These strains comprised 3% of the 186 MRSA isolates received by the National Reference Laboratory, ESR, that year. In 1993, 23% of the 306 MRSA isolates referred to ESR were of this Western Samoan lineage, and consisted of two distinct phage types designated Western Samoan phage patterns (WSPP) 1 and 2 – (WSPP1: 29/52/52A/80/55/54/77/84/95/96/+; WSPP2: 29/81/54/77/84/+). Unlike the earlier mMRSA strains, these WSPP strains were seldom multiresistant and seemed to be community rather than hospital acquired. At least in New Zealand, the pending significance of community-associated MRSA as a cause of both cutaneous and more systemic infection was clearly obvious in 1993. Over the next 5 years, most of the alarming 10-fold increase in MRSA in New Zealand was attributable to these WSPP strains, which reached a level of just over 76% of all MRSA isolated in 1998 (see Fig. 1). It was also evident during this time that while up to 30% of the WSPP isolates were initially recovered from patients in hospitals, most of these so-called hospital-acquired isolates in fact represented infections originating in community patients who required admission to hospital for subsequent investigation and treatment of cutaneous or more serious lesions. The terms 'hospital-acquired' and 'community-acquired' are rather misleading in this situation. Clearly isolates first recovered in hospital laboratories from patients admitted for investigation of a community-acquired infection, do not represent true hospital-acquired isolates.

Because of the increasing number of WSPP isolates encountered, the national surveillance of MRSA was rationalized in October 1998 to regularly include only mMRSA. From this time, regular annual short-term (1-month) surveys of all MRSA have been conducted, although most mMRSA isolates are still routinely forwarded to ESR. From surveys across New Zealand undertaken in July or August 2000–3, it was apparent that while the total number of MRSA continued to increase, the proportion which were WSPP MRSA decreased from ∼62% in 2000 to 30% in 2003 (see Fig. 1). This decrease has been attributed to the increasing proportion of MRSA that were of the UK-derived EMRSA-15 strain. These have increased from 2·5% of all MRSA isolates in 1998, to almost

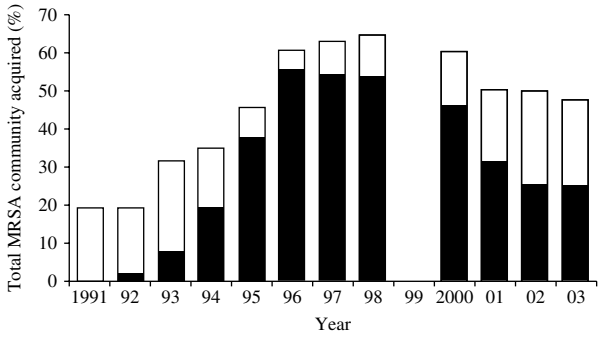

Fig. 2. Percentage of total yearly MRSA isolates (1991–2003) which were listed as community acquired. From 2000, numbers have been estimated from the results of a 1-month nationwide survey. WSPP MRSA (■); other MRSA (□).

40% in the 2002–3 period. EMRSA-15 are not overly multiresistant (β-lactams, ciprofloxacin and usually erythromycin), and contain a type IV staphylococcal chromosome cassette *mec* (SCC*mec*) element – features characteristic of community MRSA. As previously found, the majority (~73–78%) of WSPP isolates during 2000–3 were obtained from people in the community. However, as discussed above, a more accurate figure of all WSPP isolates which are community occurring is probably >90%.

In addition, it is noticeable that the percentage of all MRSA in New Zealand which are community based may be slowly decreasing from a peak of just over 63% in 1998 to ~50% in the 2001–2003 period (Fig. 2). While most of the community MRSA isolates encountered between 1995 and 1998 were WSPP MRSA (76·0% in 1995, 88·2% in 1996, 82·9% in 1997, 82·8% in 1998), this is clearly changing. In 2003, just under 50% of community MRSA isolates were this clone with most of the remainder being mMRSA, especially EMRSA-15 (Fig. 2). Since 2000, WSPP MRSA has represented a decreasing proportion of the MRSA isolations, and since 2001 the actual number of WSPP isolations has also decreased. Associated with this decline has been a concomitant rise in isolations of the EMRSA-15 strain and multiresistant strains such as AKh4 and WR/AK1. The Auckland Health District continues to be the 'hot bed' of MRSA in New Zealand, with the number of people from whom MRSA was isolated in 2002 being just under 450/100 000 population. This was around 2·5 times greater than the overall national rate of 182·7/100 000 in that year. Reasons for this are unknown, although socio-economic factors (e.g. overcrowding) and environmental factors (e.g. humid conditions) are potentially implicated. Other health districts with consistent MRSA problems over the last 3 years have been Hutt, Wellington and Hawkes Bay.

Epidemiological features of WSPP MRSA

Several studies in New Zealand have shed light on the epidemiological characteristics and significance of community-occurring WSPP MRSA. By 1996, it was obvious in New Zealand that most of the WSPP infections were community derived and that isolates invariably had methicillin MICs $<32\,\mu$g/ml [7]. In 1998, it was shown that the prevalence of MRSA bacteraemias in Middlemore Hospital, Auckland, was disproportionately high in Pacific Island patients compared to other ethnic groups, and that the recovery of MRSA from community patients in the Auckland region was highest in areas densely populated with Pacific Islanders [8]. The apparent affinity of WSPP for Pacific Island ethnic groups was finally confirmed following an analysis of 448 cases of colonization or infection encountered over a 30-month period (September 1995 to March 1998) at Middlemore Hospital [9]. Phage typing of 421 of isolates revealed 348 (82·7%) to be WSPP strains with these accounting for 91·5% (215/235) MRSA isolates from Pacific Islanders (mainly Samoan), 79·2% (38/48) from Maori and 55·8% (43/77) from Europeans. Of the 448 documented cases, 45% were Samoan, a group making up only 11% of admissions, i.e. WSPP MRSA was over four times more common in Samoans [10]. Most (>95%) of the infections were deemed to be community acquired.

Because of the disproportionate numbers of Samoan patients with MRSA in Middlemore Hospital, a survey of the nasal carriage of *S. aureus* in adults passing through the front door of a hospital in Western Samoa, and attending Middlemore Hospital in Auckland, was undertaken [10]. The study revealed that 29% (32/110) in Samoa were colonized with *S. aureus* including 2·7% (3/110) with WSPP MRSA. In Auckland, the colonization rate with *S. aureus* was lower −22% of 296 individuals including 60 Samoans. Only one MRSA was recovered. MRSA nasal carriage in South Auckland individuals was thus lower than would be expected from the frequency with which WSPP MRSA were being encountered in clinical specimens at that time.

In the southern region of New Zealand, MRSA is not as common as in the northern Auckland region, with the incidence being ~10-fold less. However,

https://doi.org/10.1017/S0950268805004024 Published online by Cambridge University Press

a survey conducted in Dunedin Hospital between 1992 and mid-1998 revealed striking differences in the demographics of individuals from whom WSPP MRSA or a local mMRSA strain, designated DNDH1, were isolated [11]. DNDH1 seemed to be imported from eastern Australia (a closely related mMRSA strain, AKh4, is presently circulating in the Auckland region). Most (90%) of the 53 patients from whom WSPP MRSA was recovered had clinical staphylococcal disease on admission to hospital (i.e. community-derived infections). In comparison, this was a feature of only 15% of the 33 individuals from whom the DNDH1 strain of MRSA was isolated. While most (70%) of the WSPP patients had overt skin disease, this was a feature found in only 6% of patients from whom the DNDH1 strain was isolated. The other striking difference concerned the age of the patient involved with 70% of those in the WSPP group being <40 years of age, and 77% of the DNDH1 group being >39 years of age. Children <10 years of age were frequently infected with WSPP MRSA (19% isolates came from this age group).

Molecular aspects of WSPP MRSA

Of special interest with regard to community MRSA, is the regular occurrence of the shorter (20–24 kb) type IV SCC*mec* elements which appear to have arisen by combination between two different SCC*mec* types [12–15]. Examination of New Zealand WSPP isolates revealed that they represent a distinct genetic clone identical to WSPP isolates from Western Samoa and the eastern coast of Australia [16, 17]. Regardless of phage pattern, isolates examined were identical with regard to the pulsed-field gel electrophoresis (PFGE) pattern of *Sma*I-digested DNA, coagulase gene restriction fragment length polymorphism pattern, localization of *mecA* to a 194-kb *Sma*I digestion fragment, accessory gene regulatory allele (*agr* type 3), *ccr* complex (type 2), class B *mec* region, and multilocus sequence type (MLST) (ST30, allelic profile 2-2-2-2-6-3-2). Isolates of this clone appear to have improved attachment properties and superior general fitness to mMRSA (possibly associated with the shorter SCC*mec*). They are also consistently egg-yolk opacity factor-negative, which had earlier been associated with increased virulence in *S. aureus* [18]. Genes coding for a variety of toxins – e.g. Panton–Valentine leukocidin (the bacteriophage associated PVL locus – *lukF*, *lukS*), another leukocidin (the related *lukE, lukD*), α-haemolysin (*hla*), γ-haemolysin (*hlg*), a possible variant β-haemolysin, and enterotoxins (enterotoxin gene cluster *egc*) – are uniformly present [16, 17, 19]. In our New Zealand study [16], all 10 WSPP isolates studied were positive by PCR for the *hla* gene, but negative for the *hlb* gene (β-haemolysin). However, by employing Southern hybridization, all isolates appeared to contain *hlb* suggesting that the primers used to amplify the *hlb* gene were not sufficiently similar to the *hlb* gene of the WSPP clone [16]. Other workers [17] have also failed to detect the *hlb* gene in WSPP isolates using PCR. The PVL locus has been found in all 16 WSPP isolates studied so far [17, 19]. Compared to most other *S. aureus* clonal complexes (CCs), it has been shown that isolates belonging to CC30 (to which ST30 and related MLST belong) are characterized by increased positivity with respect to a number of potential virulence genes, including *tst* (TSST-1 exotoxin), *sea* (enterotoxin A) and *bbp* (adhesin for bone sialoprotein) [20].

WSPP strains also appear to possess a unique SCC*mec* DNA region. While similar to the type IV cassette shared by other community MRSA, there seems to be considerable diversity in the left-hand integration region of the WSPP SCC*mec* element. Clearly the WSPP clone has a distinct genetic background associated with its southwest Pacific origin, and is distinct from other community MRSA and from the local hospital-associated MRSA. Although it is logical to assume that the WSPP clone arose from a dominant human methicillin-susceptible *S. aureus* (MSSA) the possibility that it is somehow related to an animal MSSA cannot be ignored.

Using MLST, the genetic background of WSPP MRSA isolates (ST30) appears identical to a 1962 non-typable MSSA strain (E1410) isolated in Denmark [21], and 17 MSSA isolates (approximately half of which were community based) included in the United Kingdom (Oxford area, England) study of Enright and colleagues [22]. In an expanded study by the UK group [23], MSSA belonging to ST30 was the most frequent clone (52 isolates) among 306 MSSA isolates from the Oxford region. ST30 isolates also appear to be closely related in genotype to EMRSA-16 (ST36) isolates, where the allelic profile differs at only one locus (*pta*) [21, 22]. Other MSSA with similar allelic profiles to WSPP MRSA were also found in the UK studies.

Elsewhere in the world (e.g. United States, Australia) community MRSA of differing PFGE patterns, MLST and allelic profiles have been described [19, 24–26, 28]. Like the WSPP clone, these

usually possess a type 2 *ccr* complex and class B *mec* complex; however, other unrelated and/or presently undescribed *ccr* complexes and *mec* classes obviously exist within the community MRSA group. They have clearly been present in indigenous communities living in Western Australia since at least 1986 [24].

Since the early Auckland epidemiological studies, it has become apparent that the MIC of methicillin for Auckland WSPP strains has steadily increased from the $\leqslant 32\,\mu$g/ml level found almost uniformly prior to 1994. By 1997 $\sim 36\%$ of Auckland isolates revealed MICs of $\geqslant 64\,\mu$g/ml [10]. However, an ESR survey in 2002 revealed that 96·7% of 182 WSPP isolates still had methicillin MICs of $\leqslant 32\,\mu$g/ml with only six (3·3%) isolates with MICs of $\geqslant 64\,\mu$g/ml. When methicillin was replaced with oxacillin in tests, 157 (78·6%) and 25 (13·7%) had MICs of $\leqslant 32\,\mu$g/ml and $\geqslant 64\,\mu$g/ml respectively (H. Heffernan, personal communication). Reasons for the initially low and now increasing Auckland MICs are unknown, although it is not unreasonable to assume that it is in some way associated with the evolution of the WSPP MRSA clone. However, despite the rising methicillin MIC in some localities, the incidence of multiresistance in WSPP MRSA has continued to remain constant at $<2\%$ of all isolates.

Current MRSA situation in New Zealand

What then is the present situation regarding community MRSA in New Zealand? Since at least 1991, an increasing reservoir of MRSA has occurred in the New Zealand community. This has been predominantly attributable to the non-multiresistant WSPP clone which although possessing an affinity for Polynesian populations has spread widely to other ethnic groups. The MLST of this clone (ST30) is identical to that of a common MSSA found in the United Kingdom, suggesting an evolutionary role for MSSA in the development of this community WSPP MRSA clone. While there has always been a low number of other MRSA strains in the community, it seems that the dominance of the WSPP clone in New Zealand is diminishing as mMRSA strains such as EMRSA-15 and WR/AK1 as well as non-multiresistant clones other than WSPP become more prevalent – with a consequent reduction in the proportion of community-occurring MRSA which are of the WSPP clone. Macrorestriction typing of *Sma*I-digested DNA from EMRSA-15 isolates reveals a trend of increasing diversity within this strain in New Zealand [27]. In addition, $\sim 33\%$ of current (2003) EMSRA-15 isolates are not multiresistant being ciprofloxacin resistant but erythromycin susceptible. The level of resistance to erythromycin in this strain appears to be decreasing. Resistance to clindamycin, tetracycline, fusidic acid and rifampicin is only occasionally encountered. EMRSA-15 isolates carry a type IV SCC*mec* region, are often not classically multiresistant and belong to CC22 (ST22 – allelic profile 7615886), one of the major lineages of MRSA [21, 26]. They currently present something of an enigma and clearly display fitness and virulence traits characteristic of community MRSA.

A recent survey involving MRSA colonization in patients >60 years admitted to Auckland Hospital from either residential care facilities (nursing homes) or the community, not surprisingly revealed that patients from the former were significantly more likely to be colonized – 9% *vs.* 3% [27]. However, the disturbing feature was that of the 25 isolates obtained, 24 were EMRSA-15. While only 12 (63%) of the 19 colonized residential-care patients had been in hospital within the previous 12 months, all six colonized community patients had been hospitalized in the previous year. While EMRSA-15 strains have been predominantly hospital associated, they are increasingly being recovered from patients in health-care facilities other than public hospitals and from the community. Presently, $\sim 25\%$ of isolates are obtained from people in the community, although this may be an overestimate as some community laboratories do not accurately record previous hospitalization history. It seems that strains like EMRSA-15 are increasing in fitness and possibly in their ability to survive outside health-care facilities.

As MRSA strains become increasingly common in a variety of settings (e.g. hospitals, other long-term health-care facilities, army barracks and other extended congregations of healthy individuals, the general community), somewhat restrictive terms like community- and hospital-acquired MRSA become less definable as spread between all populations obviously occurs. In New Zealand, it is now apparent that type IV SCC*mec* strains (e.g. WSPP, EMRSA-15) dominate MRSA isolations, and have done so since 1995. In each year since then, they have comprised on average almost three-quarters of all isolates (range 61–80%, with a mean of 74%). It seems probable that this situation will also occur worldwide. The potential to recover an increasing array of

SCC*mec* types, especially from community-occurring MRSA strains, clearly exists [19, 28].

ACKNOWLEDGEMENTS

The help and advice of Helen Heffernan in the preparation of this manuscript is gratefully acknowledged.

REFERENCES

1. **Humble MW.** Imported methicillin-resistant *Staphylococcus aureus* infection: a case report. N Z Med J 1976; **84**: 476–478.
2. **Heffernan H, Stehr-Green J, Davies H, Brett M, Bowers S.** Methicillin resistant *Staphylococcus aureus* (MRSA) in New Zealand 1988–90. N Z Med J 1993; **106**: 72–74.
3. **Jones MR, Martin DR.** Outbreak of methicillin-resistant *Staphylococcus aureus* infection in a New Zealand hospital. N Z Med J 1987; **100**: 369–373.
4. **Martin DR, Heffernan HM, Davies HG.** Methicillin-resistant *Staphylococcus aureus*: an increasing threat in New Zealand hospitals. N Z Med J 1989; **102**: 367–369.
5. **Heffernan H, Davies H, Brett M.** MRSA increasing in New Zealand. N Z Pub Health Rep 1995; **2**: 97–99.
6. **Heffernan H, Davies H, Brett M.** Current epidemiology of MRSA in New Zealand. Lab Link 1997; **4**: 25–26.
7. **Mitchell JM, MacCulloch D, Morris AJ.** MRSA in the community. N Z Med J 1996; **109**: 411.
8. **Riley D, MacCulloch D, Morris AJ.** Methicillin-resistant *S. aureus* in the suburbs. N Z Med J 1998; **111**: 59.
9. **Rings T, Findlay R, Lang S.** Ethnicity and methicillin-resistant *S. aureus* in South Auckland. N Z Med J 1998; **111**: 151.
10. **Lang S, Taylor S, Morris A.** Community-acquired methicillin-resistant *Staphylococcus aureus*. Antibiotics Chemother 2001; **5**: 12–13.
11. **Smith JMB, Al-Salami SF, van der Linden AJ.** Western Samoan phage pattern (WSPP) strains of methicillin-resistant *Staphylococcus aureus* in Dunedin Hospital. Proc Univ Otago Med School 1999; **77**: 28–29.
12. **Hiramatsu K, Cui L, Kuroda M, Ito T.** The emergence and evolution of methicillin-resistant *Staphylococcus aureus*. Trends Microbiol 2001; **9**: 486–493.
13. **Fluit Ad C, Schmitz F-J.** Population structure of MRSA. In: Fluit Ad C, Schmitz F-J, eds. MRSA current perspectives. Wymondham, Norfolk, UK: Caister Academic Press; 2003: 159–186.
14. **Rohrer S, Bischoff M, Rossi J, Berger-Bachi B.** Mechanisms of methicillin resistance. In: Fluit Ad C, Schmitz F-J, eds. MRSA current perspectives. Wymondham, Norfolk, UK: Caister Academic Press, 2003: 31–53.
15. **Fitzgerald JR, Musser JM.** The molecular evolution of methicillin-resistant *Staphylococcus aureus*. In: Fluit Ad C, Schmitz F-J, eds. MRSA current perspectives. Wymondham, Norfolk, UK: Caister Academic Press, 2003, 137–157.
16. **Adhikari RP, Cook GM, Lamont I, Heffernan H, Smith JMB.** Phenotypic and molecular characterization of community occurring, Western Samoan phage pattern methicillin-resistant *Staphylococcus aureus*. J Antimicrob Chemother 2002; **50**: 825–831.
17. **Vandenesch F, Naimi T, Enright MC, et al.** Community-acquired methicillin-resistant *Staphylococcus aureus* carrying Panton-Valentine leukocidin genes: worldwide emergence. Emerg Infect Dis 2003; **9**: 978–984.
18. **Jessen O, Faber V, Rosendal K, Eriksen KR.** Some properties of *Staphylococcus aureus*, possibly related to pathogenicity. Part 2: in-vitro properties and origin of the infecting strains correlated to mortality in 190 patients with *Staphylococcus aureus* bacteremia. Acta Path Microbiol Scand 1959; **47**: 327–335.
19. **O'Brien FG, Lim TT, Chong FN, et al.** Diversity among community isolates of methicillin-resistant *Staphylococcus aureus* in Australia. J Clin Microbiol 2004; **42**: 3185–3190.
20. **Peacock SJ, Moore CE, Justice A, et al.** Virulent combinations of adhesin and toxin genes in natural populations of *Staphylococcus aureus*. Infect Immun 2002; **70**: 4987–4996.
21. **Crisostomo MI, Westh H, Tomasz A, Chung M, Oliveira DC, de Lencastre H.** The evolution of methicillin resistance in *Staphylococcus aureus*: similarity of genetic backgrounds in historically early methicillin-susceptible and-resistant isolates and contemporary epidemic clones. Proc Nat Acad Sci USA 2001; **98**: 9865–9870.
22. **Enright MC, Day NP, Davies CE, Peacock SJ, Spratt BG.** Multilocus sequence typing for characterization of methicillin-resistant and methicillin-susceptible clones of *Staphylococcus aureus*. J Clin Microbiol 2000; **38**: 1008–1015.
23. **Feil EJ, Cooper JE, Grundmann H, et al.** How clonal is *Staphylococcus aureus*? J Bacteriol 2003; **185**: 3307–3316.
24. **Lim TT, Chong FN, O'Brien FG, Grubb WB.** Are all community methicillin-resistant *Staphylococcus aureus* related? A comparison of their *mec* regions. Pathology 2003; **35**: 1–8.
25. **McDougal LK, Steward CD, Killgore GE, Chaitram JM, McAllister SK, Tenover FC.** Pulsed-field gel electrophoresis typing of oxacillin-resistant *Staphylococcus aureus* isolates from the United States: establishing a national database. J Clin Microbiol 2003; **41**: 5113–5120.
26. **Robinson DA, Enright MC.** Evolutionary models of the emergence of methicillin-resistant *Staphylococcus aureus*. Antimicrob Agents Chemother 2003; **47**: 3926–3934.
27. **Briggs S, McGuiness C, Foster M, Roberts S.** A reservoir of methicillin-resistant *Staphylococcus aureus* in the Auckland community? N Z Med J 2002; **115**: 1–7.
28. **Coombs GW, Nimmo GR, Bell JM, et al.** Genetic diversity among community methicillin-resistant *Staphylococcus aureus* strains causing outpatient infections in Australia. J Clin Microbiol 2004; **42**: 4735–4743.