Annual survey of methicillin-resistant *Staphylococcus aureus* (MRSA), 2012

Helen Heffernan and Sarah Bakker

Nosocomial Infections Laboratory, Institute of Environmental Science and Research Limited (ESR); July 2013

Introduction

ESR conducts annual surveys of methicillin-resistant *Staphylococcus aureus* (MRSA). Each year, all hospital and community microbiology laboratories in New Zealand are asked to refer all MRSA isolated during a one-month period to ESR. Laboratories provide epidemiological information with each isolate referred. At ESR, MRSA are typed to identify MRSA strains. The purpose of these annual surveys is to provide information on the epidemiology of MRSA in New Zealand and to monitor changes over time.

The results of the 2012 MRSA survey are presented in this report, along with the trends in MRSA prevalence.

Previous reports on the annual MRSA surveys are available at http://www.surv.esr.cri.nz/antimicrobial/mrsa_annual.php.

Methods

MRSA isolates and data collection

Hospital and community microbiology laboratories in New Zealand were asked to refer all MRSA isolated during August 2012 to ESR. The Microbiology Department, Hawke's Bay Hospital; Medlab Central, Palmerston North; and the Microbiology Laboratory, Nelson Hospital, referred isolates during a 31-day period between mid-August and mid-October 2012. All remaining laboratories, except the Microbiology Department, Middlemore Hospital, and Labtests, Auckland, referred MRSA during August 2012.

The Microbiology Department, Middlemore Hospital, and Labtests, Auckland, were requested to send MRSA for only a 14-day period due to the large number of MRSA these two laboratories isolate. Labtests referred isolates for a 14-day period in August 2012 and the Microbiology Department, Middlemore Hospital, referred MRSA for a 14-day period in September 2012. Unless otherwise stated, the analyses in this report have been adjusted for this shorter collection period from these two laboratories so that all data represents a 1-month period.

When referring MRSA isolates, laboratories were asked to supply some epidemiological data, including patient age, geographic location, hospitalisation status, MRSA isolation site, infection or colonisation status, and if the MRSA was obtained from a screen or a diagnostic specimen. Laboratories also provided information on the susceptibility of the MRSA isolates to non-β-lactam antibiotics. The two community laboratories in the greater Auckland area, Labtests and Diagnostic Medlab, receive specimens from multiple district health boards (DHBs), Waitemata, Auckland and Counties Manukau, therefore, for MRSA referred from these laboratories, NHI numbers were used to assign people with MRSA to a DHB.

People were classified as hospital patients or hospital staff if (i) they were inpatients or outpatients in a healthcare facility when MRSA was isolated, or had been in the previous three months; (ii) they were in a residential-care facility when MRSA was isolated, or had been in the previous three months; or (iii) they were employed in a healthcare or residential-care facility when MRSA was isolated. Patients or staff were classified as people in the community if (i) MRSA was isolated from patients while in the community and the patients had no history of being in a healthcare or residential-care facility in the previous three months; (ii) MRSA was isolated from healthcare or residential-care facility admission-screening of patients who had no history of being in such facilities in the previous three months; or (iii) MRSA was isolated from pre-employment swabs of healthcare staff with no employment history supplied.

All MRSA isolates received at ESR were assumed to be pure cultures of MRSA and methicillin/oxacillin resistance was not routinely confirmed.

spa *typing and based upon repeat pattern (BURP) analysis*

The polymorphic X region of the staphylococcal protein A gene (*spa*) was amplified as previously described.[1] PCR products were sequenced by the Sequencing Laboratory at ESR using an ABI 3130XL Sequencer. *spa* sequences were analysed using Ridom StaphType software version 2.2.1 (Ridom GmbH, Würzburg, Germany). Sequences were automatically assigned repeats and *spa* types using the software. Clustering of clonal complexes of related *spa* types (Spa-CCs) was performed using the based upon repeat pattern (BURP) algorithm of

the Ridom StaphType software and the default settings of the software which exclude *spa* types with less than five repeats and allow a maximum four costs to cluster *spa* types into the same Spa-CC.[2]

Pulsed-field gel electrophoresis (PFGE) and profile analysis

Where necessary to identify strains, PFGE of *Sma*I-digested genomic DNA was performed as previously described.[3] PFGE banding patterns were analysed using BioNumerics software version 6.6 (Applied Maths, St-Martens-Latem, Belgium), with the Dice coefficient and unweighted-pair group method with arithmetic averages, at settings of 0.5% optimisation and 1.5% position tolerance. PFGE banding patterns were interpreted using the criteria proposed by Tenover et al.[4]

Multilocus sequence typing (MLST) and sequence analysis

Where necessary to characterise strains, MLST was performed as previously described.[5] Sequences were analysed using BioNumerics software version 6.6 and sequence types (STs) were assigned using the *S. aureus* database accessible at http://www.mlst.net.

Antibiotic susceptibility testing

Antibiotic susceptibility testing was performed where necessary to identify strains and to supplement the susceptibility information provided by referring laboratories. Disc susceptibility testing was performed according to the methods of the Clinical and Laboratory Standards Institute (CLSI).[6] Except for fusidic acid and mupirocin, zones of inhibition were interpreted according to CLSI criteria.[7] Fusidic acid zones of inhibition were determined with a 10 µg disc and interpreted as ≥21 mm susceptible, 20 mm intermediate and ≤19 mm resistant.[8] Mupirocin zones of inhibition were determined with a 5 µg disc and interpreted as ≥14 mm susceptible and ≤13 mm resistant.[9]

PCR for staphylococcal-specific 16S rRNA, nuc *and* mecA

Isolates that were not able to be *spa* typed were tested for the genes encoding staphylococcal-specific 16S rRNA, *S. aureus*-specific thermostable nuclease (*nuc*) and methicillin resistance (*mecA*) by triplex PCR as previously described.[10]

Assigning MRSA strains

Isolates were characterised primarily based upon *spa* types and antibiotic susceptibility patterns, with PFGE as a supplementary typing tool where *spa* typing was inconclusive. There were three situations in which *spa* typing was considered inconclusive: (i) when a *spa* type correlated to a known MRSA strain but the antibiotic susceptibility pattern did not; (ii) when an isolate had a novel *spa* type; and (iii) when an isolate had a *spa* type ESR had not yet correlated to an MRSA strain.

Epidemiological analyses

Epidemiological data and results were entered into ESR's laboratory information management system. Data and results were extracted and analysed using customised Microsoft Access queries. Point-prevalence rates were calculated based on the number of MRSA isolated per 100 000 population during the period of the survey. Mid-year New Zealand population estimates were used to calculate prevalence rates. 95% confidence intervals were calculated based on Poisson distribution. The statistical significance of time

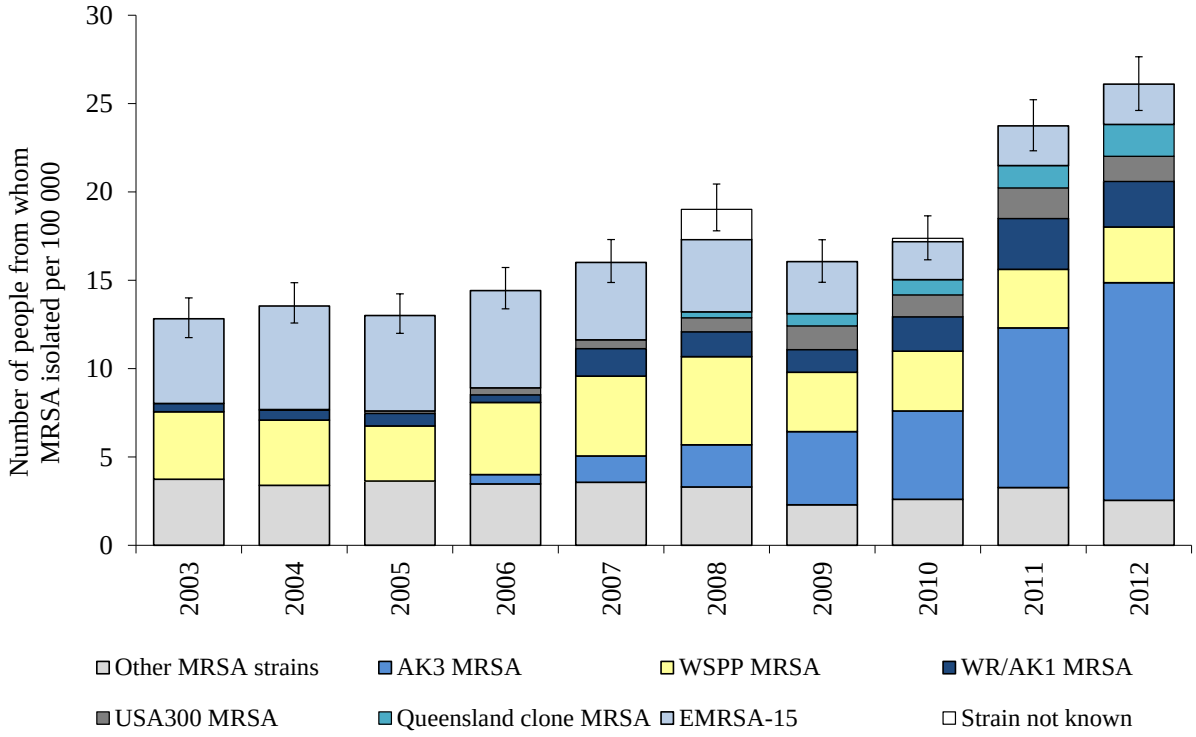
trends was calculated at a 95% confidence level using Poisson regression and the Mantel-Haenszel test for linear trend.

Results

National point-prevalence rates of MRSA, 2003-2012

During the period of the 2012 MRSA survey, MRSA were isolated from an estimated 1156 people, 1144 of whom were patients and 12 of whom were staff. There was a 10.1% increase in the MRSA point-prevalence rate between 2011 and 2012, from 23.7 to 26.1 people with MRSA per 100 000 population. Over the last 10 years, 2003 to 2012, the prevalence rate has more than doubled from 12.8 to 26.1 per 100 000 (Figure 1).

Figure 1. MRSA point-prevalence rates, 2003-2012



95% confidence intervals indicated by error bars. The category 'Strain not known' for 2008 and 2010 represents people identified with MRSA during the survey period but from whom no isolate was referred for strain identification.

MRSA infection status, strain prevalence, and strain association with healthcare facilities versus the community and with patient age

In 2012, of the 1144 patients with MRSA, 57.9% were categorised as community patients and 42.1% as hospital patients. MRSA was reported as causing infection in 75.8% of the 1007 patients for whom this information was provided.

Six MRSA strains (AK3 MRSA, WSPP MRSA, WR/AK1 MRSA, EMRSA-15, Queensland clone MRSA and USA300 MRSA) were predominant in 2012 and collectively represented 90.2% of all MRSA isolations (Table 1). The dominance of AK3 MRSA increased further in 2012 with this strain accounting for 47.2% of all MRSA included in the survey. The point-prevalence rates for the three most prevalent strains, AK3, WSPP and WR/AK1 were 12.3, 3.2 and 2.6 per 100 000 population, respectively (Figure 1).

Table 1. MRSA strain prevalence, association with healthcare facilities versus the community and association with patient age, 2012

Strain	Proportion (%) of all MRSA isolations[a]	Proportion (%) of each strain isolated from:		
		hospital patients or staff	people in the community	patients ≥60 years of age[b]
AK3 MRSA [ST5, SCC*mec* type IV][c]	47.2	40.5	59.5	16.7
WSPP MRSA [ST30, SCC*mec* type IV]	12.1	34.3	65.7	14.9
WR/AK1 MRSA [ST1, SCC*mec* type IV]	9.9	38.6	61.4	37.1
EMRSA-15 MRSA [ST22, SCC*mec* type IV]	8.7	62.4	36.6	78.3
Queensland clone MRSA [ST93, SCC*mec* type IV]	6.9	26.3	72.5	14.8
USA300 MRSA [ST8, SCC*mec* type IV]	5.4	44.4	55.6	34.6

a Other strains accounted for the remaining 9.8% of MRSA.

b Age distribution for patients only, staff not included. Data not adjusted for the shorter collection period from the Microbiology Department, Middlemore Hospital, and Labtests, Auckland.

c ST, multilocus sequence type; SCC*mec*, staphylococcal cassette chromosome *mec*.

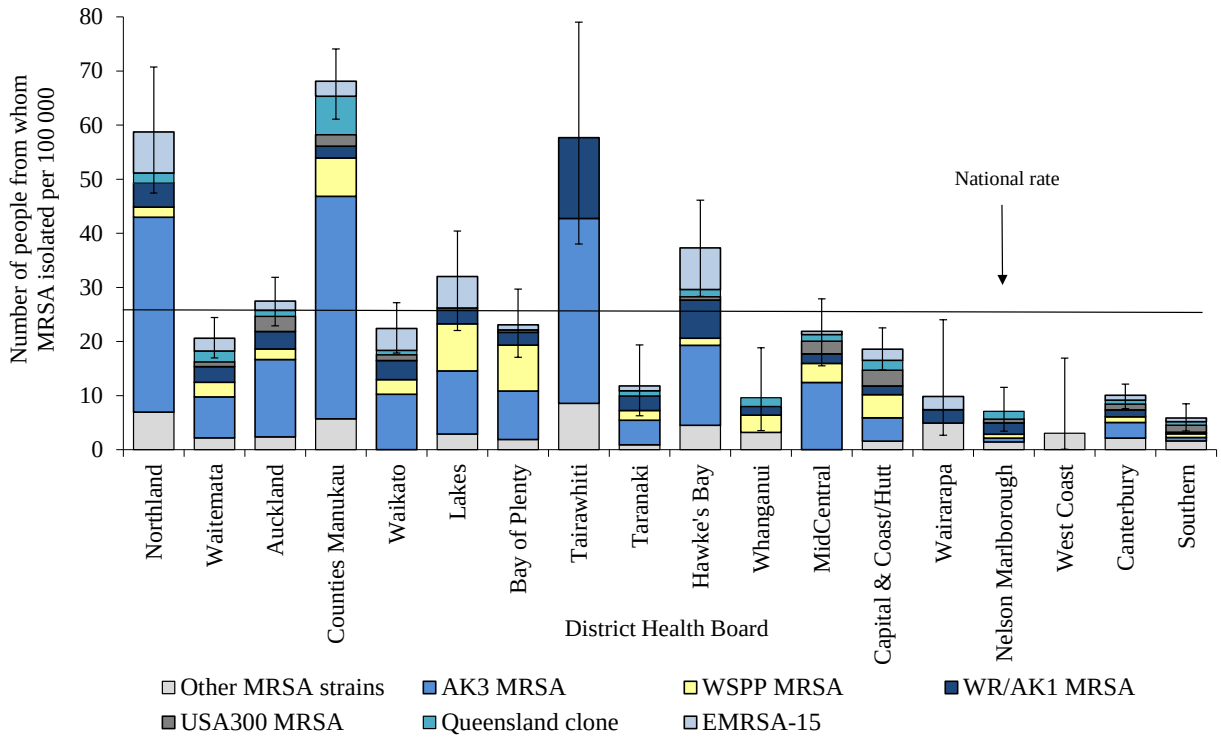
Geographic distribution of MRSA

There were significant geographical differences in the point-prevalence rates of MRSA isolations in 2012. Rates exceeded the national rate of 26.1 people with MRSA per 100 000 population in six DHBs: Counties Manukau (68.1 per 100 000), Northland (58.7), Tairawhiti (57.7), Hawke's Bay (37.3), Lakes (32.0), and Auckland (27.5) (Figure 2).

Similar geographical differences were evident in the point-prevalence rates of MRSA isolated only from infection, with five of the same six DHBs having rates above the national point-prevalence rate of 17.2 people with an MRSA infection per 100 000 population: Northland (47.4 per 100 000), Counties Manukau (41.5), Tairawhiti (34.2), Hawke's Bay (22.5), and Auckland (18.8) (Figure 3).

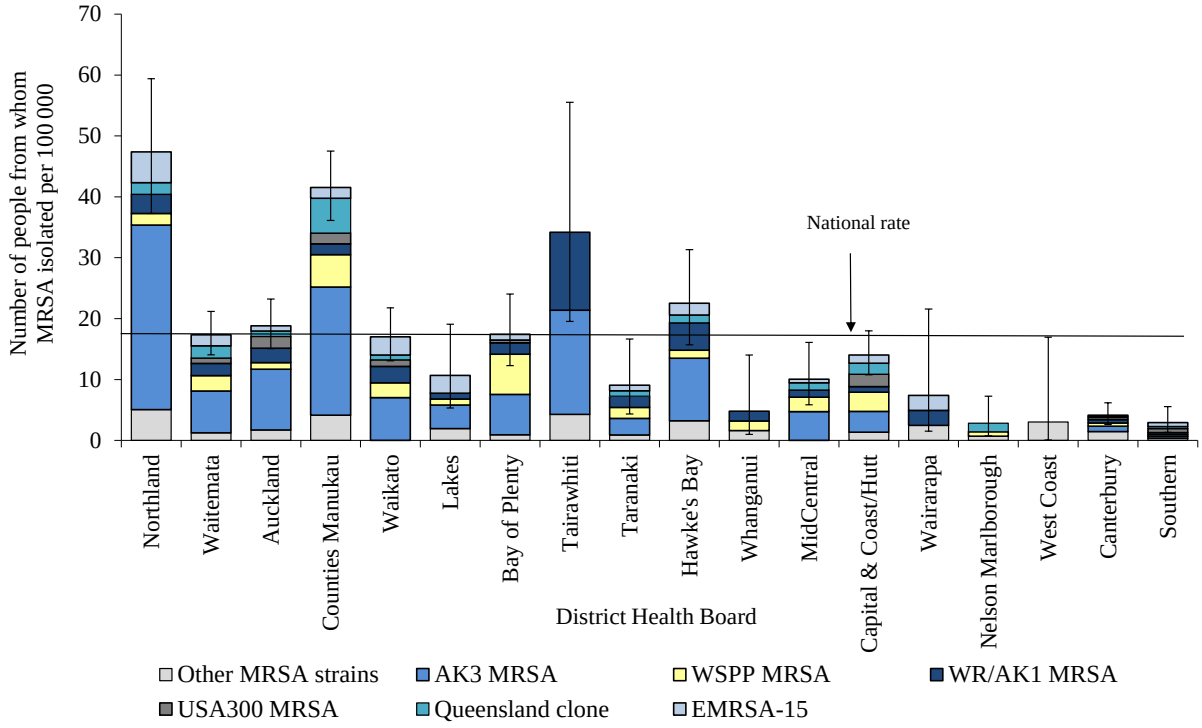
AK3 MRSA was the most prevalent MRSA strain in all North Island DHBs except, Whanganui, Capital & Coast/Hutt, and Wairarapa.

Figure 2. MRSA point-prevalence rates by district health board, 2012



95% confidence intervals indicated by error bars. Data for the Capital & Coast and Hutt DHBs are combined as 'Capital & Coast/Hutt', and data for the Canterbury and South Canterbury DHBs are combined as 'Canterbury'.

Figure 3. MRSA infection point-prevalence rates by district health board, 2012



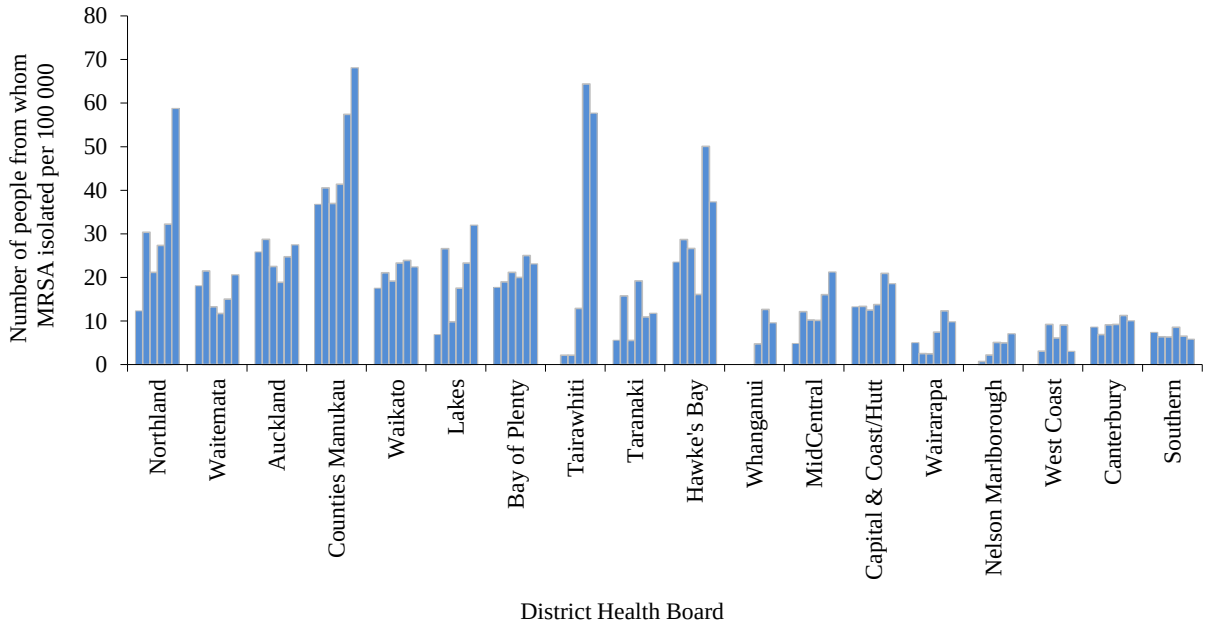
95% confidence intervals indicated by error bars. Data for the Capital & Coast and Hutt DHBs are combined as 'Capital & Coast/Hutt', and data for the Canterbury and South Canterbury DHBs are combined as 'Canterbury'.

Point-prevalence rates of MRSA by DHB, 2007-2012

Over the six-year period 2007 and 2012, there were statistically significant increases in MRSA point-prevalence rates in 9 of the 18 DHB/DHB combinations analysed. These DHBs were, ordered from the DHB with the highest increase to that with the smallest increase: Tairawhiti, Whanganui, Nelson-Marlborough, Northland, MidCentral, Lakes, Counties Manukau, Hawkes's Bay, and Capital and Coast (Figure 4).

In this report, time-trend data is analysed and presented for the first time for each of the three DHBs in the Auckland region (ie, Waitemata, Auckland and Counties Manukau) as data from 2007 onwards is now available for the individual DHBs. Notably, this separate analysis shows that rates of MRSA have only increased in the Counties Manukau DHB, and rates have actually decreased in Waitemata and Auckland DHBs, although these decreases were not significant.

Figure 4. MRSA point-prevalence rates by district health board, 2007-2012



The series of bars for each DHB represent the individual years 2007 to 2012 from left to right. Data for the Capital & Coast and Hutt DHBs are combined as 'Capital & Coast/Hutt', and data for the Canterbury and South Canterbury DHBs are combined as 'Canterbury'.

MRSA strain association with spa *types*

In 2012, the AK3 MRSA strain was most commonly associated with *spa* type t002, WSPP MRSA with t019, WR/AK1 MRSA with t127, EMRSA-15 with t032, the Queensland clone MRSA with t3949, and the USA300 MRSA with t008 (Table 2). AK3 MRSA was associated with the greatest variety of *spa* types.

Table 2. *spa* types of the most six most prevalent MRSA strains in 2012[a]

Strain	Number of isolates of the strain	*spa* clonal cluster	*spa* type[b]	Number of isolates of the *spa* type
AK3 MRSA [ST5, SCC*mec* type IV][c]	396[d]	Spa-CC002	t002	334
			t045	11
			t088	6
			t548	6
			t067	4
			t105	3
			t306	3
			t062	2
			t179	2
			t242	2
			t311	2
			t5677	2
			t6787	2
WSPP MRSA [ST30, SCC*mec* type IV]	114	Spa-CC019	t019	105
			t2208	2
Alternative names: Southwest Pacific clone and Oceania clone				
WR/AK1 MRSA [ST1, SCC*mec* type IV]	99	Spa-CC127	t127	82
			t267	4
			t591	3
Alternative name: Western Australia (WA) MRSA-1		Spa-CC008	t701	5
		Singleton[e]	t10753	2
EMRSA-15 [ST22, SCC*mec* type IV]	95[f]	Spa-CC032	t032	66
			t852	6
			t1401	3
			t005	2
			t022	2
			t379	2
			t2818	2
Queensland clone MRSA [ST93, SCC*mec* type IV]	54	Spa-CC202	t3949	39
			t202	11
USA300 MRSA [ST8, SCC*mec* type IV]	53	Spa-CC008	t008	40
			t024	9

Footnotes: see next page.

Table 2 footnotes:

a The data in this table is based on isolates received for the survey, that is, the data is not adjusted for the shorter collection period from the Microbiology Department, Middlemore Hospital, and Labtests, Auckland.

b The *spa* types are only listed in the table if there were ≥2 isolates of the type. In addition to the *spa* types listed in the table:

among the AK3 MRSA isolates there was also 1 isolate of each of the following *spa* types: t010, t071, t214, t575, t653, t1062, t1107, t1781, t2051, t2069, t2225, t3469, t3979, t4865, t5181, t5213 and t11036;

among the WSPP MRSA isolates there was also 1 isolate of each of the following *spa* types: t018, t138, t1133, t3593, t6653, t11174 and t11417;

among the WR/AK1 MRSA isolates there was also 1 isolate of each of the following *spa* types: t304, t359 and t11784;

among the EMRSA-15 MRSA isolates there was also 1 isolate of each of the following *spa* types: t906, t1214, t1222, t1378, t1467, t4573, t5830, t7105, t9639, t11331, t11434 and t11783;

among the Queensland clone MRSA isolates there was also 1 isolate of each of the following *spa* types: t1811, t4699, t11037 and t11609; and

among the USA300 MRSA isolates there was also 1 isolate of each of the following *spa* types: t068, t211, t967 and t1767.

c ST, multilocus sequence type; SCC*mec*, staphylococcal cassette chromosome *mec*.

d The total number of AK3 MRSA isolates was 397, but the *spa* type of 1 isolate could not be determined and therefore this isolate was identified solely by PFGE typing.

e A 'singleton' *spa* type does not cluster by BURP analysis with any other *spa* types.

f The total number of EMRSA-15 isolates was 96, but the *spa* type of 1 isolate could not be determined and therefore this isolate were identified solely by PFGE typing.

In addition to the six most prevalent MRSA strains listed in Table 2, isolates of several other recognized MRSA strains were identified. These included six isolates of the Bengal Bay MRSA clone (ST772, SCC*mec* type V), four isolates of the AKh4 MRSA strain (ST239, SCC*mec* type III), one isolate of the WA MRSA-2 strain (ST78, SCC*mec* type IV), and one isolate of the CC398 MRSA clone (CC398, SCC*mec* type V).

The Bengal Bay MRSA clone is a multiresistant MRSA, typically resistant to ciprofloxacin, erythromycin and gentamicin. This strain carries the genes for several virulence factors including the Panton Valentine leukocidin (PVL) genes and the enterotoxin gene cluster.

The AKh4 MRSA is a healthcare-associated MRSA (HA-MRSA) strain that is multiresistant to ciprofloxacin, clindamycin, co-trimoxazole, erythromycin, gentamicin and tetracycline. This strain is a common cause of HA-MRSA infections in many parts of the world including the east coast states of Australia. Its prevalence in New Zealand has decreased in recent years, but it still occasionally causes small outbreaks in healthcare facilities.

CC398 MRSA is a livestock-associated MRSA, which was first identified in New Zealand during the 2011 MRSA survey. The isolate identified in the 2012 survey was a repeat isolate from patient from whom this strain was first isolated in 2011.

There were 82 isolates that were not associated with a recognized MRSA strain, and the most common *spa* types among these isolates were t1853 (10 isolates) and t976 (6 isolates). There were less than five isolates of any other *spa* type not associated with a known MRSA strain.

Discussion

Based on data from these annual national surveys, the prevalence of MRSA among the population in New Zealand has approximately doubled over the last 10 years (2003-2012), with the largest single-year increase (37.0%) occurring between 2010 and 2011. The 10.1% increase between 2011 and 2012 was somewhat smaller and more similar to the average yearly increase over the last 10 years.

These increases in the prevalence of MRSA most likely reflect the increasing incidence of *S. aureus* infections in New Zealand rather than increases in the proportion of *S. aureus* that are methicillin resistant. National data collated by ESR and based on routine susceptibility testing undertaken in diagnostic laboratories, indicates that the proportion of *S. aureus* that tested as methicillin resistant only increased from 7.5% to 10.4% between 2003 and 2011.[11] Whereas a recently published study of *S. aureus* skin and soft tissue infections among children in New Zealand reported that the incidence of these infections, which were predominantly due to methicillin-susceptible *S. aureus,* almost doubled in just the 4 years 2007 to 2010.[12]

The large geographical differences in MRSA prevalence noted for several years were again evident in 2012, with rates generally highest in DHBs in the upper half of the North Island. Concomitant with the national trend of increasing MRSA prevalence, there have also been significant increases in many DHBs in recent years. With one exception (Nelson Marlborough), all DHBs in which there have been significant increases are North Island DHBs. By far the largest increase has been in the Tairawhiti DHB, which, until 2010, had a relatively low prevalence of MRSA.

The MRSA strains associated with community-acquired infections often belong to lineages distinct from MRSA associated with healthcare-acquired infections,[13] although this distinction is blurring with some community-associated MRSA (CA-MRSA) strains now also causing healthcare-associated infections.[14,15] In 2012, six MRSA strains, AK3 MRSA, WSPP MRSA, WR/AK1 MRSA, EMRSA-15, Queensland clone MRSA and USA300 MRSA were collectively responsible for 90.2% of MRSA isolations in New Zealand. Five of these six most common strains - AK3 MRSA, WSPP MRSA, WR/AK1 MRSA, Queensland clone MRSA and USA300 MRSA - are usually considered CA-MRSA. The EMRSA-15 strain was the only healthcare-associated MRSA (HCA-MRSA) strain represented among the six most common strains in 2012 and accounted for just 8.7% of MRSA.

The current predominance of CA-MRSA strains indicates that once again, as in the 1990s, MRSA is more commonly transmitted and acquired in the community in New Zealand than in our healthcare facilities.[16] The most notable change in MRSA strains in recent years has been the emergence in 2005 and subsequent spread of the AK3 MRSA. The prevalence of this strain increased again in 2012 and accounted for almost half (47.2%) of MRSA isolations – up from 38.0% in 2011. AK3 MRSA was almost 4-times as prevalent as any other MRSA strain. It was the most prevalent strain in most DHBs in the upper and central North Island, and was particularly dominant in Counties Manukau, Northland and Tairawhiti DHBs.

AK3 MRSA is considered primarily a CA-MRSA strain in New Zealand, with the majority (59.5% in 2012) of patients from whom it is isolated being categorised as 'community' patients by our criteria. In addition, the relatively young age profile of the patients from whom AK3 MRSA is isolated is characteristic of CA-MRSA. Like many CA-MRSA, the

AK3 MRSA strain has type IV SCC*mec* element and it is not multiresistant – being most commonly resistant to only fusidic acid in addition to β-lactams. However, atypically for a CA-MRSA strain, it does not produce Panton-Valentine leukocidin (PVL). AK3 MRSA is multilocus sequence type 5 (ST5). Based on its MLST and SCC*mec* type, AK3 MRSA appears to belong to the globally widespread 'Paediatric Clone'. This clone has achieved pandemic spread and is a major cause of MRSA infections.[17]

The Bengal Bay clone is an unusually resistant and virulent CA-MRSA strain, which appears to have first emerged in Bangladesh and India, and, when isolated elsewhere, there is usually a link by travel or ethnicity to the Bengal Bay area. Retrospective analysis shows that this strain has been isolated in New Zealand since at least 2008. A recently published enhanced analysis of the results of the seven annual MRSA surveys conducted between 2005 and 2011, which included data on the ethnicity of patients, found that 10 of 14 patients identified with the Bengal Bay MRSA strain were of Indian ethnicity.[18] Fortunately, this strain appears to be only infrequently isolated in New Zealand, with just six people identified with it in the 2012 survey.

The livestock-associated CC398 MRSA strain was first identified among pigs and veal calves in the Netherlands.[19] Initial isolations of CC398 MRSA from humans were from people who had contact with pig farms. The strain quickly spread to other countries in Europe, North America and Asia, and also to other animal species. While the transmissibility of this strain among humans may be lower than that of other widespread MRSA strains, CC398 MRSA clearly has the ability to become widespread among herds of animals, which creates a substantial reservoir and therefore risk for human MRSA colonisation and infection.[20]

CC398 MRSA was first identified in New Zealand from three people during the 2011 survey. Between the 2011 and 2012 surveys this strain was identified from a further two people. All five people resided in the South Island and none were identified as having the risk factors for this strain, in particular, contact with livestock, especially pigs, or travel to Europe. However, one patient had travelled to Cambodia where this clone has been identified.[21] The one isolate of CC398 MRSA identified among the 2012 survey isolates was a repeat isolate from one of the five initial people with CC398 MRSA. Therefore this strain appears to still be uncommon, at least among human hosts, in New Zealand. Little is known about the prevalence and types of MRSA among pigs and other food-producing animals in New Zealand.

These annual MRSA surveys have several limitations. First, as MRSA from both diagnostic specimens and screening specimens are included, any apparent differences in MRSA rates, for example, over time and between DHBs, could be partly due to changes and differences in screening policies.

Second, for the 2012 survey, MRSA isolates were collected for only a 14-day period from two laboratories in the Auckland area since these laboratories serve high-prevalence areas. While the data presented in the analyses in this report was adjusted to allow for the 14-day collection period, this shortened collection period could potentially have reduced the representativeness of the data if, for example, the 14-day collection period did not reflect the epidemiology of MRSA over the full one-month period. However, there is no information to suggest the 14-day period was not representative. In addition, the numbers of isolates collected from these two laboratories were still several magnitudes greater that the numbers collected from low-prevalence areas.

Third, only limited demographic data is routinely collected about the people from whom MRSA are isolated. In particular, no data is collected on ethnicity or socioeconomic status. In addition, the categorisation of patients as 'hospital' or 'community' patients may not always be accurate. The recent hospitalisation history of a person in the community when their MRSA was isolated may not be reported to ESR, which would result in people who have been hospitalised in the previous 3 months being incorrectly categorised as community patients. Conversely however, people in a healthcare facility when their MRSA was isolated are categorised as hospital patients, but may have acquired MRSA prior to their admission.

Recently the data from each annual survey between 2005 and 2011 was further analysed and reported.[18] The methods used in this retrospective, multi-year analysis addressed several of the above limitations. To minimise any impact of differences in screening policies, the analysis was restricted to patients who had a clinical MRSA isolate. Patient demographic information was enhanced by obtaining data on patient ethnicity, socioeconomic status and hospitalisation history from the Ministry of Health's National Minimum Dataset (NMDS). The hospitalisation history obtained from the NMDS enabled more accurate categorisation of MRSA as either community associated or healthcare associated than the categorisation of patients as 'hospital' or 'community' patients standardly applied to survey isolates.

This enhanced analysis confirmed trends and associations evident from the standard analyses of the surveys, including that the increase in MRSA prevalence in recent years has been driven almost entirely by increases in community-associated or community-onset MRSA rather than healthcare-onset cases. Importantly, the enhanced analysis showed that MRSA is significantly ($P \leq 0.05$) more prevalent in Māori and Pacific Peoples than other ethnic groups. Moreover, compared to patients with HCA-MRSA, patients with CA-MRSA were significantly younger, more likely to be Māori or Pacific Peoples, and more likely to live in the most deprived neighborhoods (ie, NZDep score 8-10 areas).

In conclusion, the prevalence of MRSA is continuing to increase in many DHBs in New Zealand, although there are still large variations in prevalence between DHBs. CA-MRSA strains are predominant, with AK3 MRSA now the most common strain and its prevalence increasing further in 2012. As the recently published enhanced analysis of data from the 2005 to 2011 MRSA surveys showed, there are clearly opportunities to improve the epidemiological value of these surveys by sourcing more accurate and complete patient demographic data from the NMDS rather than directly from laboratories.

References

1. Strommenger B, Braulke C, Heuck D, Schmidt C, Pasemann B, Nübel U, et al. *spa* typing of *Staphylococcus aureus* as a frontline tool in epidemiological typing. J Clin Microbiol 2008; 46: 574-81.

2. Mellmann A, Weniger T, Berssenbrugge C, Rothganger J, Sammeth M, Stoye J, et al. Based upon repeat pattern (BURP): an algorithm to characterize the long-term evolution of *Staphylococcus aureus* populations based on spa polymorphisms. BMC Microbiol 2007; 7: 98.

3. Goering RV. Pulsed-field gel electrophoresis. In: Persing DH, Tenover FC, Versalovic J, Tang YW, Unger ER, Relman DA, White TJ, editors. Molecular microbiology: diagnostic principles and practice. Washington: ASM Press; 2004. p. 185-96.

4. Tenover FC, Arbeit RD, Goering RV, Mickelsen PA, Murray BE, Persing DH, et al. Interpreting chromosomal DNA restriction patterns produced by pulsed- field gel electrophoresis: criteria for bacterial strain typing. J Clin Microbiol 1995; 33: 2233-9.

5. 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: 1008-15.

6. Clinical and Laboratory Standards Institute. Performance standards for antimicrobial disk susceptibility tests; approved standard – eleventh edition. Wayne (PA): CLSI; 2012. CLSI document M2-A11.

7. Clinical and Laboratory Standards Institute. Performance standards for antimicrobial susceptibility testing; twenty-second informational supplement. Wayne (PA): CLSI; 2012. CLSI document M100-S22.

8. Toma E, Barriault D. Antimicrobial activity of fusidic acid and disk diffusion susceptibility testing criteria for gram-positive cocci. J Clin Microbiol 1995; 33: 1712-5.

9. Finlay JE, Miller LA, Poupard JA. Interpretive criteria for testing susceptibility of staphylococci to mupirocin. Antimicrob Agents and Chemother 1997; 41: 1137-9.

10. Maes N, Magdalena J, Rottiers S, De Gheldre Y, Struelens MJ. Evaluation of a triplex PCR assay to discriminate *Staphylococcus aureus* from coagulase-negative Staphylococci and determine methicillin resistance from blood cultures. J Clin Microbiol 2002; 40: 1514-7.

11. Institute of Environmental Science and Research Ltd. General antimicrobial susceptibility data. Available at http://www.surv.esr.cri.nz/antimicrobial/ general_antimicrobial_susceptibility.php.

12. Williamson DA, Ritchie SR, Lennon D, Roberts SA, Stewart J, Thomas, MG, Baker MG. Increasing incidence and sociodemographic variation in community-onset *Staphylococcus aureus* skin and soft tissue infections in New Zealand children. J Ped Infect Dis 2013; doi: 10.1097/INF.0b013e3182905f3d.

13. DeLeo FR, Otto M, Kreiswirth BN, Chambers HF. Community-associated meticillin-resistant *Staphylococcus aureus*. Lancet 2010; 375: 1557-68.

14. Tenover FC. Community-associated methicillin-resistant *Staphylococcus aureus*: it's not just in communities anymore. Clin Microbiol Newslett 2006; 28: 33-6.

15. Skov RL, Jensen KS. Community-associated methicillin-resistant *Staphylococcus aureus* as a cause of hospital-acquired infections. J Hosp Infect 2009; 73: 364-70.

16. Smith JMB, Cook GM. A decade of community MRSA in New Zealand. Epidemiol Infect 2005; 133: 899-904.

17. Monecke S, Coombs G, Shore AC, Coleman DC, Akpaka P, Borg M, et al. A field guide to pandemic, epidemic and sporadic clones of methicillin-resistant *Staphylococcus aureus.* PLoS ONE 2011; 6: e17936.

18. Williamson DA, Roberts SA, Ritchie SR, Coombs GW, Fraser JD, Heffernan H**.** Clinical and molecular epidemiology of methicillin-resistant *Staphylococcus aureus* in New Zealand: rapid emergence of Sequence Type 5 (ST5)-SCC*mec*-IV as the dominant community-associated MRSA clone. PLoS ONE 2013; 8: e62020. Available at: http://dx.plos.org/10.1371/journal.pone.0062020.

19. Price LB, Stegger M, Hasman H, Aziz M, Larsen J, Andersen PS, et al. *Staphylococcus aureus* CC398: host adaptation and emergence of methicillin resistance in livestock. mBio 2012; 3: e00305-11.

20. Wulf M, Voss A. MRSA in livestock animals – an epidemic waiting to happen? Clin Microbiol Infect 2008; 14: 519-21.

21. Williamson DA, Coombs GW, Roberts SA, Tan HL, Bakker S, Heffernan H**.** Emergence and molecular characterization of clonal complex 398 (CC398) methicillin-resistant *Staphylococcus aureus* (MRSA) in New Zealand. Australian Society for Antimicrobials Annual Conference; 2013 Feb 21-3; Sydney.