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Annual reports

Australian Meningococcal Surveillance Programme annual report, 2010

The Australian Meningococcal Surveillance Programme

Abstract

In 2010 there were 214 laboratory-confirmed cases of invasive meningococcal disease analysed by the National Neisseria Network, a nationwide network of reference laboratories. One hundred and twenty-four isolates of *Neisseria meningitidis* from invasive cases of meningococcal disease were available for which the phenotypes (serogroup, serotype and serosubtype) and/or genotype and antibiotic susceptibility were determined. An additional 90 cases were confirmed by non-culture based methods (77 by nucleic acid amplification testing and 13 by serology), and where possible, serotyping was determined. Nationally 167 (78%) laboratory-confirmed cases, where a serogroup was determined, were infected with serogroup B, 16 (7.5%) with serogroup C, 9 (4.2%) with serogroup W135 and 7 (3.3%) with serogroup Y meningococci. The national total of confirmed cases has decreased since 2004, but the number of cases may vary between jurisdictions each year. New South Wales had the highest number of recorded cases in 2010. Typical primary and secondary disease peaks were observed in those aged 4 years or less and in adolescents and young adults respectively. Serogroup B cases predominated in all age groups and jurisdictions. The common phenotype circulating in Australia continues to be B:15:P1.7, corresponding to the *porA* genotype P1.7,16-26. Serogroup C cases were again numerically low, as were serogroups W135 and Y. Eighty per cent of all isolates showed decreased susceptibility to the penicillin group of antibiotics (minimal inhibitory concentration (MIC) 0.06–0.5 mg/L). All isolates remained susceptible to ceftriaxone. One isolate had reduced susceptibility to ciprofloxacin, and none to rifampicin. *Commun Dis Intell* 2011;35(3):217–228.

Keywords: disease surveillance; meningococcal disease; *Neisseria meningitidis*

Introduction

The National Neisseria Network (NNN) is a long-term collaborative program for the laboratory surveillance of the pathogenic Neisseria species: *Neisseria meningitidis* and *N. gonorrhoeae*. Since 1994 the NNN has operated through a network of reference laboratories in each state and territory to provide a national laboratory-based program for the examination of *Neisseria meningitidis* from cases of invasive meningococcal disease (IMD).[1] The NNN supplies data on the phenotype and/or the genotype of invasive meningococci, and their antibiotic susceptibility, supplementing clinical notification data from the National Notifiable Diseases Surveillance System (NNDSS). The NNN receives samples for analysis from about 90% (range 85%–92% 2004–2009) of IMD cases notified to NNDSS.[2] The NNN annual reports are published in *Communicable Diseases Intelligence*.[3]

The characteristics of the meningococci responsible for IMD are important, both for individual patient management and to tailor the public health response for outbreaks or case clusters locally and nationally. The introduction of publicly funded conjugate serogroup C meningococcal vaccine onto the National Immunisation Program in 2003 (with a catch-up program for those aged 1–19 years that ran until May 2007) saw a significant and sustained reduction in the number of cases of IMD evident after 2004.[2] However, IMD remains an issue of public health concern in Australia. The success of any further vaccine initiatives in Australia is dependent upon detailed analysis of the *Neisseria meningitidis* isolates circulating locally. This report provides relevant details of cases of IMD confirmed by laboratory testing in Australia in 2010.

Methods

Isolate based invasive meningococcal disease cases

Case confirmation

Case confirmation was based upon isolation of, or positive nucleic acid amplification testing (NAAT) for, *N. meningitidis* from a normally sterile site; or by positive serology, and defined as IMD according to Public Health Laboratory Network criteria.[4] Information on the site of infection, the age and sex of the patient and the outcome (survived/died) of the infection was sought. The isolate-based subset of the program categorised cases on the basis of site of isolation of the organism. Where an isolate was

grown from both blood and cerebrospinal fluid (CSF) cultures in the same patient, the case was classified as one of meningitis. It is recognised that the total number of cases, and particularly the number of cases of meningitis, is underestimated because no lumbar puncture was performed, or was delayed and the culture sterile. However, the above approach has been used since the beginning of this program[1] and is continued for comparative purposes.

Phenotyping and genotyping

Phenotyping of invasive isolates of meningococci by serotyping and serosubtyping was based on the detection of outer membrane protein (porin) antigens using a standard set of monoclonal antibodies obtained from The Netherlands National Institute for Public Health. Increasingly, sequencing of products derived from amplification of the porin genes *porA* and *porB* and *FetA* (genotyping) is used to supplement and supplant meningococcal serotyping analyses based on the use of monoclonal antibodies.

Antibiotic susceptibility

Antibiotic susceptibility was assessed by determining the minimal inhibitory concentration (MIC) to antibiotics used for therapeutic and prophylactic purposes. This program uses the following parameters to define the various levels of penicillin susceptibility/resistance when determined by a standardised agar plate dilution technique.[5]

sensitive: MIC $\leq$ 0.03 mg/L

less sensitive: MIC 0.06–0.5 mg/L

relatively resistant: MIC $\geq$ 1 mg/L

Strains with MIC values that place them in the category of 'sensitive' or 'less sensitive' would be considered to be amenable to penicillin therapy when used in currently recommended doses. However precise the MIC, outcome correlations are difficult to obtain because of the nature of IMD.

Non-culture-based laboratory-confirmed cases

Additional laboratory confirmation of suspected cases of IMD was obtained by means of non-culture based methods, primarily by NAAT, and occasionally by serological techniques. NAAT testing is essentially by polymerase chain reaction (PCR) techniques[6] that demonstrate the presence of meningococcal-specific nucleic acid in appropriate samples and has been progressively introduced and updated in the different jurisdictions. Data from the results of these investigations were included for the first time in the 1999 report. The serological results are based on results of tests performed using the methods and test criteria of the Manchester Health Protection Agency Reference Laboratory, United Kingdom as assessed for Australian conditions.[7–10] Where age, sex and outcome data for patients with non-culture-based diagnoses are available, these were also recorded. The site of a sample of a positive NAAT is also used to define the clinical syndrome.

Results

Aggregated data on cases confirmed by culture-based and non-culture-based methods

Number of laboratory confirmed cases

There were 214 isolates of IMD tested in Australia in 2010 (Table 1) representing 93% of invasive meningococcal notifications to NNDSS.[2] In 124 cases (58%), a positive culture was obtained

Table 1: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 2010, by serogroup and state or territory

State or territory	Serogroup						Total
	B	C	Y	W135	NG	ND	
ACT	2	0	0	0	0	0	2
NSW	51	8	2	3	0	12	76
NT	3	0	0	1	0	0	4
Qld	39	5	0	1	0	2	47
SA	19	0	1	0	0	0	20
Tas	5	1	0	0	0	1	7
Vic	31	1	3	3	0	0	38
WA	17	1	1	1	0	0	20
Australia	167	16	7	9	0	15	214

NG Non-groupable

ND Non-determined, samples were examined by nucleic acid amplification test and serological methods.

with or without a positive non-culture-based test and 90 cases (42%)were confirmed by a non-culture-based method alone. The highest number of laboratory confirmed cases was from New South Wales (76 cases), which has decreased from 82 cases in 2009. The total number of all laboratory confirmed cases in Queensland was 47, a decrease from 60 in 2009; 83 in 2008 and 75 in 2007. There were 38 laboratory confirmed cases in Victoria, 20 in Western Australia and in South Australia, seven in Tasmania, four in the Northern Territory and two in the Australian Capital Territory.

Small or no numerical differences from 2009 were noted in these jurisdictions.

Seasonality

Forty-four cases occurred between 1 January and 31 March, 52 between 1 April and 30 June, 64 between 1 July and 30 September and 54 between 1 October and 31 December. A winter peak of meningococcal disease is usual and the above pattern was also present in 2007, 2008 and 2009.

Age distribution

The age distribution of IMD cases in Australia in 2010 is shown in Table 2. Nationally, the peak incidence of meningococcal disease was again in those aged 4 years and under. Those aged less than 1 year or in the 1–4 year age group together accounted for 72 cases (34% of the total) in 2010, similar to the proportion reported in this age group in 2007–2009 (33%–36%). A secondary disease peak is also usual in the adolescent/young adult age group (15–24 years). The total of 31 confirmed cases (14%) in those aged 15–19 years in 2010 was less than the range reported in this age group in 2007 to 2009 (19%–20%). The 15–24 year age group accounted for 48 cases (22%), compared with 27%–31% reported in this age group in 2007 to 2009. In 2010, 11% of cases were in the 25–44 years age group, which was lower than the 15% in 2009. Thirteen per cent of cases were in the 45–64 years age, which was higher than the 6% in 2009.

Serogroup data

The serogroup was determined in 199 of the 214 laboratory confirmed cases of IMD. Of these, 167 (84%) were serogroup B and 16 (8%) were serogroup C. This distribution was little changed from the range reported over the period 2007–2009, where 85%–88% were serogroup B and 6%–7% were serogroup C. In 2010, there were 9 cases (4.5%) of serogroup W135 and 7 cases (3.5%) of serogroup Y. With the continuing low numbers of serogroup C infections, serogroup B meningococci predominated in all age groups and jurisdictional differences in serogroup distribution were not evident.

The 16 serogroup C cases of IMD were distributed in 5 jurisdictions: New South Wales (8); Queensland (5); Western Australia; Victoria and Tasmania (1 each). Ten of the 16 cases of serogroup C disease in 2010 were aged 25 years or more; 3 cases were in the 5–14 age group, two were reported in those aged 4 years or less and there was a single case in those aged 15–19 years and none in those aged 20–24 years.

Table 3 shows a national comparison of the number and proportion of serogroup B and C cases by age from 2004 to 2010. In those aged 14 years or less, there was a continued decrease in the total case numbers of serogroup B cases in 2010. Serogroup C case numbers were also low in these age groups across this period. In those aged 15–24 years, the number of serogroup B cases decreased to 44 in 2010 from 52 in 2009, but the proportion of serogroup B cases showed an increase to 92% in 2010 from 84% in 2009. Serogroup C cases continued to decline in number and proportion in the 15–24 years age group. The relative proportion of serogroup B and C IMD cases was unaltered in 2010 from that observed in 2007 to 2009. In older (25 years or more) age groups in 2010 there was a decrease in the number and proportion of serogroup B cases and an increase in serogroup C cases when compared with 2009.

Phenotypes of invasive meningococcal isolates

Serogroup B meningococci are typically of heterogeneous phenotypes. In 2010 the phenotypes of invasive isolates, based on a determination of their serogroup, serotype and serosubtype, were analysed for New South Wales, the Australian Capital Territory, South Australia, Queensland and the Northern Territory (Darwin). The serogroup B and C serotypes and serosubtypes are shown in Table 4. Serogroup B meningococci are in general more difficult to characterise by serological methods and a number could not be phenotyped. A total of 75 isolates were serotyped. Sixty-one of these were serogroup B, where 16 belonged to serotype 15 and 12 of these were serosubtype P1.7, which has been circulating in Australia for many years; six were serotype 4, three (all from Queensland) of which were serosubtype P1.4, which has been circulating in New Zealand at high rates for many years. Twenty were non-typeable.

Seven serogroup C strains were phenotyped and four (all from New South Wales) were serotype 2a. This phenotype has predominated in serogroup C meningococci in Australia for many years. Of the 4 serotype 2a isolates, two were phenotyped as C:2a:P1.4, one was phenotype C:2a:P1.5 and one C:2a strain was non-subtypeable. Three serogroup C strains were non-typeable and non-subtypeable. There is continuing interest in the

Table 2: All laboratory confirmed cases of invasive meningococcal disease, Australia, 2010, by age, state or territory and B and C serogroups

State or territory	Serogroup	Age group										
		<1	1–4	5–9	10–14	15–19	20–24	25–44	45–64	65+	NS	Total
ACT	B	1	0	0	1	0	0	0	0	0	0	2
	C	0	0	0	0	0	0	0	0	0	0	0
	Total	1	0	0	1	0	0	0	0	0	0	2
NSW	B	7	11	4	2	9	5	3	5	2	3	51
	C	1	0	0	1	1	0	1	1	3	0	8
	Total	9	16	5	3	10	7	7	10	6	3	76
NT	B	0	1	0	0	0	0	1	1	0	0	3
	C	0	0	0	0	0	0	0	0	0	0	0
	Total	0	2	0	0	0	0	1	1	0	0	4
Qld	B	4	8	2	1	11	1	5	6	1	0	39
	C	0	0	1	1	0	0	1	1	1	0	5
	Total	4	8	5	2	11	1	6	7	3	0	47
SA	B	0	6	0	3	4	2	3	1	0	0	19
	C	0	0	0	0	0	0	0	0	0	0	0
	Total	0	6	0	3	4	2	3	2	0	0	20
Tas	B	3	0	1	0	1	0	0	0	0	0	5
	C	0	0	0	0	0	0	0	1	0	0	1
	Total	4	0	1	0	1	0	0	1	0	0	7
Vic	B	8	4	3	1	3	3	3	5	1	0	31
	C	1	0	0	0	0	0	0	0	0	0	1
	Total	9	5	3	1	3	3	4	6	4	0	38
WA	B	2	6	0	1	1	4	1	1	0	1	17
	C	0	0	0	0	0	0	0	0	1	0	1
	Total	2	6	0	1	2	4	2	1	1	1	20
Australia	B	25	36	10	9	29	15	16	19	4	4	167
	C	2	0	1	2	1	0	2	3	5	0	16
	Total B+C	27	36	11	11	30	15	18	22	9	4	183
	other	2	7	3	0	1	2	5	6	5	0	31
	Total	29	43	14	11	31	17	23	28	14	4	214
	% of all	14	20	6	5	15	8	11	13	6	2	

NS Age not stated.

Totals include cases due to other serogroups (16) and cases where the serogroup was not determined (15).

presence of any serogroup B or serogroup C meningococci of serotypes that indicate the possibility of genetic recombination events. Among serogroup C strains, phenotype C:2a:P1.4 had been of particular interest where it figured prominently in Victorian data in previous years. In 2003 there were 29 isolates of this serogroup C serotype/serosubtype detected nationally, with 21 in 2004 and eight in 2005. However, other than the two C:2a:P1.4 meningococcal isolates reported in New South Wales in 2010, no isolates with this phenotype or its equivalent genotype were seen other jurisdictions in 2009 or 2010.

Genotyping data of invasive meningococcal samples (culture or NAAT products)

Sequencing products derived from amplification of the variable region *porA* and *porB* and *FetA* genes is used in an increasing number of jurisdictions in place of serotyping using monoclonal antibodies. Since 2009 some jurisdictions have moved to the use of genotyping (Victoria, Queensland, Western Australia and Tasmania and a number of isolates from New South Wales). There was a heterogeneity of typing data across jurisdictions with predominance of a few phenotypes or genotypes as shown in Table 4 and Figure 1. Figure 2 shows

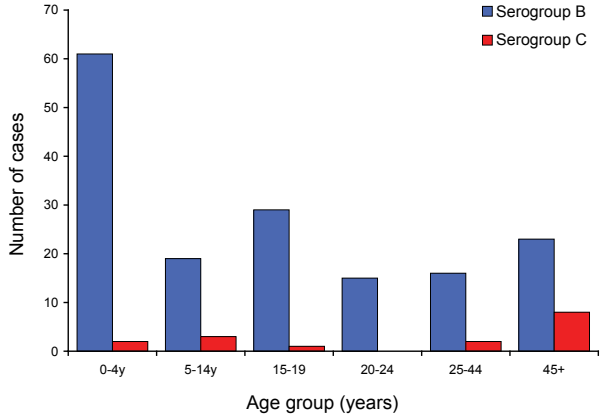
Table 3: A comparison of the number and proportion of serogroup B and serogroup C laboratory-confirmed cases, 2004 to 2010, by known age

		Age (years)									
		< 4		5–14		15–19		20–24		25+	
Year	Serogroup	n	%	n	%	n	%	n	%	n	%
2010	B	61	85.0	19	76.0	29	94.0	15	88.0	39	60.0
	C	2	3.0	3	12.0	1	3.0	0	0.0	10	15.0
	All*	72		25		31		17		65	
2009	B	72	94.0	21	75.0	38	83.0	14	88.0	41	76.0
	C	2	2.6	3	11.0	1	2.2	1	6.3	4	7.0
	All*	77		28		46		16		55	
2008	B	82	89.0	23	96.0	42	91.3	15	83.0	57	85.0
	C	4	4.4	0	0.0	1	2.2	2	11.1	8	11.0
	All*	92		24		46		18		67	
2007	B	83	90.0	19	83.0	48	91.0	24	80.0	49	75.0
	C	4	4.0	0	0.0	2	4.0	3	10.0	8	12.0
	All	92		23		53		30		65	
2006	B	93	93.0	21	84.0	40	82.0	21	70.0	38	61.0
	C	2	2.0	3	12.0	4	8.2	7	23.0	10	16.0
	All	100		25		49		30		62	
2005	B	99	90.0	38	75.0	39	81.0	22	67.0	51	50.0
	C	6	5.5	5	10.0	4	8.0	8	24.0	27	27.0
	All	110		51		48		33		101	
2004	B	97	88.0	27	77.0	40	65.0	20	57.0	59	50.0
	C	6	5.5	5	14.0	17	28.0	11	31.0	32	27.0
	All	110		35		61		35		117	

* All cases where a serogroup was determined and patient's age was supplied.

the collation of the national genotyping data of *porA* genotypes by number and serogroup in confirmed cases of invasive meningococcal disease for 2010. The predominant *porA* genotypes, all belonging to serogroup B meningococcus, include P1.7-2,4 (20 isolates), P1.7,16–26 (15 isolates) and P1.22,14–6 (10 isolates).

Figure 1: Number of serogroup B and C cases of invasive meningococcal disease confirmed by all methods, Australia, 2010, by age



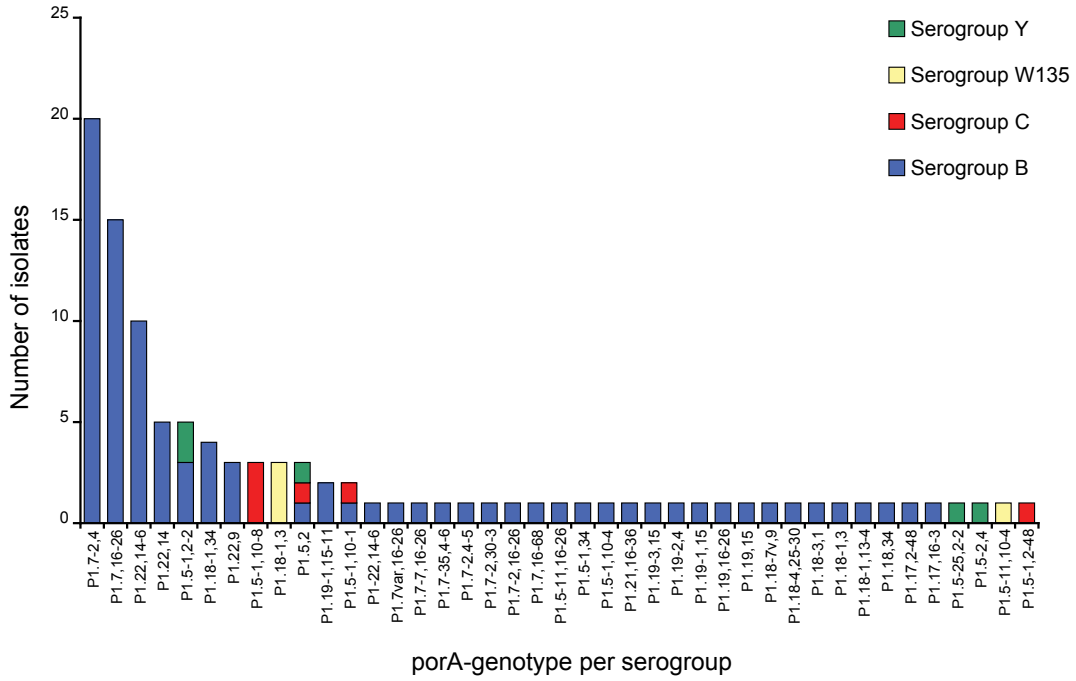
Outcome data for invasive meningococcal disease for laboratory confirmed cases

Outcome data (survived or died) were available for 51 (24%) of the 214 laboratory confirmed cases as shown in Table 5. Of these, 4 deaths were recorded (1.9%), all attributable to septicaemia; three with serogroup B infection and one with W135 infection. Outcome data were available for 42 of 167 cases with serogroup B infection and four of the 9 serogroup W135 infections. No deaths were recorded for the infections caused by other serogroups.

Anatomical source of samples for laboratory confirmed cases

Table 6 shows the source of clinical samples by which laboratory confirmation of IMD was obtained. Those diagnoses shown as culture positive may have

Figure 2: Number of *porA* genotypes per serogroup in cases of invasive meningococcal disease, Australia, 2010



Only includes cases where genotype data were available.

Table 4: Phenotypes (serotype, serosubtype) and genotypes: *porB* variable region type), *porA* variable region type, and *FetA* type of isolates or DNA extracts from cases of invasive meningococcal disease, Australia, 2010, by state or territory

State or territory	Serogroup	Phenotype				Genotype					
		Serotype	n	Sero-subtype	n	*porB*	n	*porA*	n	*Fet A*	n
ACT	B	15	1	P1.7							
		NT	1	P1.16							
NSW	B	15*	8	P1.7	6	A,A,A,Ba	2	P1.7,16-26	2	F3-3	2
				P1.7,16	1						
				P1.15	1						
		1	4	P1.14	1						
				P1.7	1						
				NST	2						
		4	3	P1.12	1						
				P1.15	1						
				P1.74	1						
		18-1	1	P1.3	1						
		2a	1	NST	1						
		NT	15	P1.14	2						
				P1.4	4						
				P1.5,2	1						
				P1.7	3						
				P1.7,15	1						
				NST	4						
		ND	1	ND	1						

Table 4 *continued*: Phenotypes (serotype, serosubtype) and genotypes: *porB* variable region type), *porA* variable region type, and *FetA* type of isolates or DNA extracts from cases of invasive meningococcal disease, Australia, 2010, by state or territory

State or territory	Serogroup	Phenotype				Genotype					
		Serotype	n	Sero-subtype	n	*porB*	n	*porA*	n	*Fet A*	n
NSW, *cont'd*	C	2a	4	P1.4	2						
				P1.5	1						
				NST	1						
		NT	1	P1.14	1						
	W135	NT	3	P1.16	1						
				P1.63	1						
				NST	1						
	Y	NT	1	NST	1						
NT	B	ND	2	P1.22,14-6	1			P1.22,14-6	1	F1-5	1
				P1.18-3,1	1						
	W135	ND	1	ND	1						
Qld	B	15	1	NST	1			P1.17,16-3	1	F5-5	1
		15	1	P1.15	1			P1.17,2-48	1	F1-19	1
		ND	1	NST	1			P1.18-1,13-4	1	F5-12	1
		1	1	P1.6	1			P1.18-4,25-30	1	F1-5	1
								P1.19,15	1	F3-3	1
								P1.19,16-26	1	F3-3	1
								P1.19-1,15	1	F5-1	1
		ND	1	NST	1			P1.19-1,15-11	1	F5-1	1
								P1.22,9	1	F5-12	1
		15	1	P1.7	1			P1.5-1,2-2	1	F3-3	1
								P1.5-11,16-26	1	F3-6	1
		15	2	P1.7	2			P1.7,16-26	8	F3-3	6
				NST	1						
										F5-5	2
		15	1	P1.7	1			P1.7,16-68	1	F3-3	1
								P1.7-2,16-26	1	F3-3	1
		4	3	P1.4	3			P1.7-2,4	7	F1-5	6
		1	1	P1.4	2						
										F5-9	1
				NST	1			P1.7-35,4-6	1	F1-5	1
								P1.7-7,16-26	1	F3-3	1
								ND	9	F1-5	2
		15	1	P1.7	1					F3-3	3
		15	1	P1.7	1					ND	3
		1	1	NST	1					F5-1	1
	C	ND	1	P1.5	1			P1.5-1,10-1	1	F3-6	1
		15	1	NST	1			P1.5-1,10-8	3	F3-3	1
										F3-6	2
								P1.5-1,2-48	1	F3-6	1
	W135			NST	1			ND		F4-1	1
SA	B			P1.7-2,4	7			P1.7-2,4	7		
				P1.7-2.4-5	1			P1.7-2.4-5	1		
	Y			P1.5,2	1			P1.5,2	1		

Table 4 *continued*: Phenotypes (serotype, serosubtype) and genotypes: *porB* variable region type), *porA* variable region type, and *FetA* type of isolates or DNA extracts from cases of invasive meningococcal disease, Australia, 2010, by state or territory

State or territory	Serogroup	Phenotype				Genotype					
		Serotype	n	Sero-subtype	n	*porB*	n	*porA*	n	*Fet A*	n
Tas	B					D,Ea,2b,C	1	P1.18-7v,9	1	F1-5	1
						19,Ac,7a,1	1	P1.19-3,15	1	F3-6	1
						A,A,A,Ba	1	P1.7var,16-26	1	F3-3	1
Vic	B					19,A,10,Aa	1	P1.22,14-6	1	F1-5	1
						19,Ac,7a,1	5	P1.18-1,34	2	F1-5	2
								P1.18,34	1	F1-5	1
								P1.22,14	1	F5-5	1
								P1.7-2,4	1	F1-5	1
						19,aC,7var,1	1	P1.18-1,34	1	F1-5	1
						19,Db,7c,14	1	P1.5-1,10-1	1	F1-5	1
						19,Dvar,7b,Bvar	1	P1.19-2,4	1	F3-6	1
						4,D,7,14a	3	P1.18-1,3	1	ND	1
								P1.5-1,10-4	1	ND	1
								P1.7-2,4	1	F1-5	1
						A,A,A,Ba	4	P1.7,16-26	4	F3-3	4
						B,C,7,146	1	P1.5-1,34	1	F5-1	1
						B,C,7,14b	2	P1.5-1,2-2	2	F5-1	2
						new,Dvar,7b,Bvar	2	P1.22,9	2	F5-12	1
										ND	1
						ND	10	ND	3	ND	3
								P1.18-1,34	1	ND	1
								P1.21,16-36	1	F5-8	1
								P1.22,14	3	F1-5	1
										F5-5	1
										ND	1
								P1.5,2	1	F5-8	1
								P1.7-2,4	1	F1-5	1
	C					C,Eb,2a,C	1	P1.7-2,4	1	F5-2	1
	W135					D,Ed,new,Db	2	P1.18-1,3	1	F4-1	1
								P1.18-1,3	1	ND	1
						D,Ed,new,Ca(var)	1	P1.18-1,3	1	F4-1	1
	Y					C,E var, new,Db	1	P1.5-1, 2-2	1	ND	1
						C,Evar,Zvar,Db	1	P1.5-1,2-2	1	F5-1	1
						19,Db,7c,14var	1	P1.5-2,4	1	F4-1	1
WA	B							P1.19-1,15-11	1	F5-1	1
								P1.22,14	1	F5-5	1
								P1.22,14-6	2	F1-5	1
										F4-24	1
								P1.7,16-26	1	F3-3	1
								P1.7-2,30-3	1	F5-1	1
								P1.7-2,4	2	F1-5	2
								P1-22,14-6	1	F1-5	1
	C							P1.5,2	1	F3-6	1
	W135							P1.5-11,10-4	1	F3-4	1
	Y							P1.5-25,2-2	1	F5-8	1

had positive NAAT and/or serology; those shown as NAAT positive were culture negative with or without positive serology. There were 75 diagnoses of meningitis based on cultures or NAAT examination of CSF either alone or with a positive blood sample; and 122 from blood samples (cultures or NAAT) alone. There were 2 other isolates from synovial fluid and 1 pericardial fluid tested by NAAT. For 1 NAAT diagnosis the source of the clinical sample was not disclosed. Thirteen cases were serologically positive where culture and NAAT were negative.

Antibiotic susceptibility surveillance of invasive meningococcal isolates

Penicillin

One hundred and twenty-four meningococcal isolates were available for determination of their susceptibility to penicillin and other antibiotics. Using defined criteria, 99 isolates (80%) were less sensitive to penicillin in the MIC range 0.06–0.5 mg/L and the remainder (20%) fully sensitive (MIC 0.03 mg/L or less). The proportion of less sensitive strains is more than that reported in 2009 (67%) and 2008 (72%) but similar to that reported in 2007 (79%).

Other antibiotics

All isolates were fully susceptible to ceftriaxone and by extrapolation to other third generation cephalosporins. One isolate had altered susceptibility (MIC, 0.06–0.5 mg/L) to ciprofloxacin (MIC 0.5 mg/L). There were no isolates with altered susceptibility to rifampicin.

Discussion

In 2010, there were 214 isolates laboratory confirmed by the NNN, representing 93% IMD notifications to NNDSS.[2] There has been a continued decrease in the number of notifications of IMD in Australia since 2004 and this has been reflected in a decrease in the number of laboratory confirmed cases of IMD by the NNN. However, the proportion of IMD notifications with laboratory confirmation has increased from 88% to 93% over this period. Fluctuations in the frequency of detection of cases continue between jurisdictions with

Table 5: Outcome data (survived, died) for laboratory confirmed cases of invasive meningococcal disease, 2010, by syndrome and serogroup

		Serogroup						
Disease type	Outcome	B	C	Y	W135	NG	ND	Total
Meningitis	Survived	16	0	0	2	0	1	19
	Died	0	0	0	0	0	0	0
	Total	16	0	0	2	0	1	19
Septicaemia	Survived	23	2	1	1	0	1	28
	Died	3	0	0	1	0	0	4
	Total	26	2	1	2	0	1	31
All cases	Survived	39	2	1	3	0	2	47
	Died	3	0	0	1	0	0	4
	Total	42	2	1	4	0	2	51

NG Not groupable.
ND Serogroup has not been determined.

Table 6: Anatomical source of samples positive for a laboratory confirmed case of invasive meningococcal disease, Australia, 2010

Specimen type	Meningococcal culture positive	NAAT positive*	Serology alone	Total
Blood	92	30	–	122
Cerebrospinal fluid +/– blood	30	45	–	75
Other†	2	2	–	4
Serum/serology	–	–	13	13
Total	124	77	13	214

* Nucleic acid amplification test (NAAT) positive in the absence of a positive meningococcal culture.
† Other samples: 2 isolates from joints, 1 NAAT from pericardial fluid and 1 NAAT diagnosis from an unknown source.
CSF Cerebrospinal fluid.

New South Wales recording the highest number of cases in 2009 (82) and 2010 (76), whereas Queensland recorded the highest number of cases in 2008 (83). There was been a decrease in the number of cases in Victoria from 61 in 2008 to 39 in 2009 and this further decreased in 2010 to 31 cases. The distribution of serogroup B (84%) and serogroup C (8%) is essentially the same as that reported for 2007–2009.

Of the 214 laboratory confirmed cases of IMD in 2010, cultures were obtained from sterile sites in 124 cases (58%), proportionally similar to the number of isolates for 2006–2009 (55%–61%). Non-culture based diagnoses were used to confirm the remaining 90 cases (42%) of IMD, again proportionally similar to the number of non-culture-based diagnoses in the period 2007–2009 (39%–45%). Attention is specifically drawn to earlier AMSP reports that explain differences between the number of clinically notified cases and laboratory confirmed cases.[11] It should also be noted that surveillance systems rarely capture all cases in any given period so that small differences in the number of cases should be expected.

Only 16 serogroup C infections were identified nationally in 2010. Serogroup B disease accounted for 84% of all infections where a serogroup was determined. No serogroup C cases were identified in South Australia, the Northern Territory or the Australian Capital Territory, with 8 cases in New South Wales, five in Queensland and small numbers present in the other states. Only low numbers of infections due to serogroups Y and W135 were encountered, and this is usual for Australia. A primary peak in IMD infection rates was once again evident in younger age groups with a secondary peak in adolescents and young adults. In 2010 there was an increase in the proportion of cases in the 45–64 years age group, primarily with serogoup B infections. The distribution of serogroup C disease was low across all age groups in 2010. As in previous years, there was a small number of serogroup C cases in those aged 25 years or more (Table 3), which may reflect the secondary benefit of herd immunity accruing to the wider community following vaccination of those age groups where disease was formerly highly concentrated.[12]

Phenotypic and genotypic data again found no evidence of substantial numbers of cases of IMD caused by *N. meningitidis* that have undergone genetic recombination, although sporadic instances of this occurrence have been detected in Australia. There were some concerns expressed that the documented capacity for genetic reconfiguration within meningococci may lead to the emergence of new and invasive subtypes following extensive vaccine use.[12] Analysis of meningococcal subtypes and any evidence for the expansion of 'new' subtypes will continue as part of the NNN program. Mortality data were assessable in only a low proportion of cases (24%) and must be interpreted with caution. Three of the 4 fatal cases of IMD were associated with serogroup B infection and one with serogroup W135. The NNN does not attempt collection of morbidity data associated with IMD.

The distribution of penicillin MICs in invasive isolates in 2010 showed that the proportion with decreased susceptibility to penicillins was 80%, which was higher than the proportion reported in 2009 (67%) and 2008 (72%), but similar to that observed in 2007 (79%). It is emphasised that this decreased susceptibility does not affect clinical outcomes and penicillins remain a suitable treatment for IMD in Australia. All isolates were susceptible to the third generation cephalosporins and to the 'clearance' antibiotics rifampicin and ciprofloxacin with the exception of 1 isolate from New South Wales, with decreased susceptibility to ciprofloxacin. Strains with decreased susceptibility to quinolone antibiotics have been the subject of on-going international interest following their first description from the Australian Meningococcal Surveillance Programme group in 2000.[13–16] There was 1 isolate with decreased susceptibility to quinolone antibiotics detected in 2010, compared with four in 2009, two in 2008, and one in 2007.

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Australian Gonococcal Surveillance Programme annual report, 2010

The Australian Gonococcal Surveillance Programme

Abstract

The Australian Gonococcal Surveillance Programme monitors antibiotic susceptibility testing of *Neisseria gonorrhoeae* isolated in all states and territories. In 2010 the *in vitro* susceptibility of 3,997 isolates of gonococci from public and private sector sources was determined by standardised methods. Varying antibiotic susceptibility patterns were again reported across jurisdictions and regions. Resistance to the penicillins nationally was 29% and, with the exception of the Northern Territory, ranged from 22% in Queensland to 42% in Victoria. Quinolone resistance, most at high minimal inhibitory concentration (MIC) levels, was 35% nationally (excepting the Northern Territory), ranging from 28% in Queensland to 44% in Victoria. Decreased susceptibility to ceftriaxone (MIC 0.06 mg/L or more), was found nationally in 4.8% of isolates. There has not been an isolate of *N. gonorrhoeae* with an MIC value greater than 0.125 mg/L reported in Australia. Nationally, all isolates remained sensitive to spectinomycin. Azithromycin surveillance was performed in New South Wales, Queensland, Western Australia, the Northern Territory and South Australia, and resistance was found in low numbers of gonococci with MIC values up to 16 mg/L. In larger urban centres the ratio of male to female cases was high, and rectal and pharyngeal isolates were common in men. In other centres, and in rural Australia, the male to female ratio of cases was lower, and most isolates were from the genital tract. *Commun Dis Intell* 2011;35(3):229–236.

Keywords: antimicrobial resistance; disease surveillance; gonococcal infection; *Neisseria gonorrhoeae*

Introduction

The World Health Organization (WHO) estimates that 88 million cases of gonorrhoea (*Neisseria gonorrhoeae* infection) occur annually, globally.[1] In Australia, the rate of *Neisseria gonorrhoeae* infections has increased from 35.8 per 100,000 in 2005 to 40.4 per 100,000 in 2010.[2] Around the world, the increasing prevalence of antimicrobial resistance (AMR) in *Neisseria gonorrhoeae* and its impact on treatment outcome is a major and growing concern[1,3] as antibiotic treatment is fundamental to disease control at the population level.[3]

Emergence of resistance to the penicillins, tetracyclines; macrolides and fluoroquinolone antibiotics has necessitated the removal of these agents from standard treatment regimens.[4] This has resulted in the replacement with extended-spectrum cephalosporin antibiotics as the recommended first line treatment for gonorrhoea in Australia and elsewhere.[5] Unusually, but importantly in Australia however, treatments based on the penicillins remain effective in many rural centres where extremely high disease rates persist.[4]

In large centres in urban Australia, AMR in *Neisseria gonorrhoeae* has long been influenced by the introduction of multi-resistant strains from overseas.[4] There are an increasing number of reports from overseas sources[6,7] of treatment failures with orally administered extended-spectrum cephalosporin (ESCs). In Australia, oral extended-spectrum cephalosporin antibiotics are not available, therefore the injectable form (ceftriaxone) is recommended for use in high doses.[5] No treatment failures have yet been reported following ceftriaxone treatment of genital-tract gonorrhoea. However there were 2 instances of failure of treatment of pharyngeal gonorrhoea after treatment with ceftriaxone 250 mg intramuscularly, reported in Sydney[8] where elimination of intercurrent genital-tract infection with the same organism was achieved. The gonococci involved both had raised minimal inhibitory concentrations (MIC values) for ceftriaxone.

Strategies for treating and controlling gonorrhoea are based on single dose regimens effecting a cure in a minimum of 95%, and the formulation of these regimens is reliant on data derived from continuous AMR monitoring of gonococci to the antibiotics in clinical use.[3,9] Recently, and following the reports of treatment failures with orally administered extended-spectrum cephalosporins,[6,7] calls have been made internationally for enhanced surveillance of all forms of gonococcal AMR in order to optimise gonococcal antibiotic treatment.[1,10]

Since 1981, the Australian Gonococcal Surveillance Programme (AGSP) has monitored the susceptibility of *N. gonorrhoeae* continuously, making it the longest, continually running national surveillance system for gonococcal AMR.[11] The emergence and spread of penicillin and quinolone resistant gonococci in major cities in Australia has been well documented.[4] This analysis of AMR in *N. gonorrhoeae* in Australia

was derived from data collated by the AGSP during the 2010 calendar year. It provides information regarding the gonococcal isolates showing resistance to multiple antibiotics including those with decreased susceptibility to ceftriaxone.[4,12]

Methods

Ongoing monitoring of AMR in gonococci in Australia is performed by the AGSP through a collaborative program conducted by reference laboratories in each state and territory. The AGSP is a component of the National Neisseria Network of Australia and comprises participating laboratories in each state and territory. This collaborative network of laboratories obtains isolates for examination from as wide a section of the community as possible, and both public and private sector laboratories refer isolates to regional testing centres. The increasing use of non-culture-based methods of diagnosis has the potential to reduce the size of the sample of isolates available for testing. Details of the number of organisms examined are thus provided in order to indicate the AGSP sample size.

Gonococci isolated in, and referred to, the participating laboratories are examined for antibiotic susceptibility to the penicillins; quinolones; spectinomycin and third generation cephalosporins; and for high-level resistance to the tetracyclines, by a standardised methodology previously described.[11,13] The AGSP also conducts a program-specific quality assurance program.[14]

Antibiotic susceptibility data from each jurisdiction are submitted quarterly to the coordinating laboratory, which collates the results and provides individual feedback to each participating laboratory. Additionally, the AGSP collects data on the gender of the patient, and the site of isolation of gonococcal strains. Where available, data on the geographic source of acquisition of antibiotic-resistant isolates are included in analyses.

Results

Number of isolates

There were 4,100 gonococcal isolates referred to, or else isolated in, AGSP laboratories in 2010, representing 41% of the 10,014 cases of gonococcal infection notified to the Australian Government Department of Health and Ageing in 2010,[2] proportionally essentially unchanged from 2009 (40%) and 2008 (42%).

The source and site of infection of these isolates are shown in Table 1. In 2010, 1,328 of the 4,100 gonococcal isolates (32%) were from New South Wales; 913 (22%) were from Victoria; 840 (21%) were from Queensland; 448 (11%) were from the Northern Territory; 352 (9%) were from Western Australia and 178 (4%) were from South Australia. There were a small number in the Australian Capital Territory (30; 0.7%) and Tasmania (11; 0.3%).

Isolate numbers in 2010 increased from those reported in 2009 in most jurisdictions: New South Wales (from 949), Victoria (from 786), Queensland (from 561), the Northern Territory (from 387), and Western Australia (from 318). There was a decrease in referred isolates from the Australian Capital Territory (from 38) and similar numbers were reported from South Australia (170) and Tasmania (11).

Table 1: Source and number of gonococcal isolates, Australia, 2010, by sex, site and region

		State or territory								
Gender	Site	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Male	Urethra	8	644	297	463	93	9	468	218	2,200
	Rectal	7	328	3	80	26	0	193	37	674
	Pharynx	6	184	0	37	20	0	137	23	407
	DGI	0	8	10	2	0	0	1	1	22
	Other/NS	4	31	2	12	11	0	12	6	78
	Total	25	1,195	312	594	150	9	811	285	3,381
Female	Cervix	4	113	127	228	24	1	83	62	642
	Rectal	0	2	0	1	0	0	3	1	7
	Pharynx	0	11	0	2	0	0	11	2	26
	DGI	0	3	8	10	0	0	1	2	24
	Other/NS	1	4	1	5	4	1	4	0	20
	Total	5	133	136	246	28	2	102	67	719
Total		30	1,328	448	840	178	11	913	352	4,100

DGI Disseminated gonococcal infection.

Source of isolates

There were 3,381 strains from men (82%) and 719 (18%) from women, with the male to female (M:F) ratio of 4.7:1, which was higher than the 4.4:1 in 2009 and the 3.7:1 ratio for 2008. The number of strains from men increased from 2,622 in 2009, and the number of isolates from women increased from 596 in 2009, but the proportions of isolates from males and females were the same as in 2009: men 81% and women 19%.

The number of referred isolates from females increased in 2010 in New South Wales (from 124) and Queensland (from 121), but were essentially unchanged from Victoria (from 101); Western Australia (from 75) and the Northern Territory (134 in 2009). Small increases were also noted from the Australian Capital Territory and Tasmania. There was a continuing decrease in referred isolates from females in South Australia (from 40 in 2009, and 104 in 2008).

There were 46 referred isolates from disseminated gonococcal infection; 22 in men (0.7% of all referred isolates from men), essentially unchanged from 2009: 23 isolates 0.9% of all referred isolates. In females there were 24 isolates in 2010 (3% of referred isolates, an increase from the 4 isolates (0.7%) referred from females in 2009). Although not all infected sites were identified, isolates from urine samples were regarded as genital tract isolates and most of the other unidentified isolates were probably from this source, although they were not specified.

Antibiotic susceptibility patterns

Three thousand nine hundred and ninety-seven of the 4,100 referred gonococcal isolates in 2010 (97%) remained viable for susceptibility testing. These were examined by the AGSP reference laboratories for susceptibility to penicillin (representing this group of antibiotics), ceftriaxone (representing later generation cephalosporins), ciprofloxacin (representing quinolone antibiotics), spectinomycin, and for high level resistance to tetracycline (TRNG). As in past years, the patterns of gonococcal antibiotic susceptibility differed between the various states and territories. For this reason data are presented by region as well as aggregated for Australia as a whole.

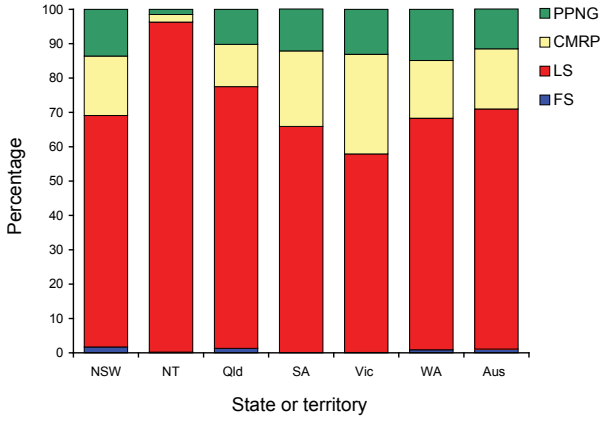
Penicillins

The categorisation of gonococci isolated in Australia in 2010 by penicillin MIC is shown in Figure 1. Infections unlikely to respond to treatment with the penicillin group of antibiotics (penicillin, ampicillin and amoxycillin with or without clavulanic acid), are caused by penicillinase-producing *N. gonorrhoeae* (PPNG) and /or *N. gonorrhoeae* that are chromosomally resistant to penicillin (CMRP). Resistance in the PPNG group results from the production of beta-lactamase, and in the CMRP group by the aggregation of chromosomally-controlled resistance mechanisms.[3] Chromosomal resistance is defined by a MIC to penicillin of 1 mg/L or more.[3,13] The MIC is the least amount of antibiotic that inhibits *in vitro* growth under defined conditions. Infections with gonococci classified as fully sensitive (MIC ≤ 0.03 mg/L) or less sensitive (MIC 0.06–0.5 mg/L) would be expected to respond to standard penicillin treatments, although response to treatment may vary at different anatomical sites.

Nationally, of those gonococci available for susceptibility testing 1,161 (29%) were penicillin resistant by one or more mechanisms in 2010, a decrease in the proportion of isolates resistant to this group of antibiotics recorded in 2009 (36%) and 2008 (44%). In 2010, there were 699 CMRP (17%) and 462 PPNG (12%) identified. In 2009 there were 22% CMRP, and 15% PPNG showing that the decrease in penicillin resistance nationally in 2010 was due to a decrease in the proportion of gonococci with both chromosomally-mediated resistance and penicillinase production, whereas in 2008 and 2009 the reduction was predominantly due to chromosomally-mediated resistance.

The proportion of penicillin-resistance of all gonococcal isolates was highest in Victoria 42% (CMRP 29%: PPNG 13%); South Australia 34% (CMRP 22%: PPNG 12%); Western Australia 32% (CMRP 17%: PPNG 15%); New South Wales 31% (CMRP 17%:PPNG 14%) and in Queensland 22% (CMRP 12%:PPNG 10%).

Figure 1: Penicillin resistance of gonococcal isolates, Australia, 2010, by region



FS	Fully sensitive to penicillin, MIC ≤ 0.03 mg/L
LS	Less sensitive to penicillin, MIC 0.06–0.5 mg/L
CMRP	Chromosomally mediated resistant to penicillin, MIC ≥ 1 mg/L
PPNG	Penicillinase-producing *Neisseria gonorrhoeae*

Proportions were lower than those reported in 2009 in Victoria and South Australia and New South Wales. In Western Australia and Queensland the proportions were essentially unchanged from 2009. There were 4 CMRP and 3 PPNG identified in the Australian Capital Territory; and 3 CMRP and 1 PPNG in Tasmania. In the Northern Territory, there were 15 PPNG, which was unchanged from 2009: 9 CMRP (3 from Alice Springs and 6 from Darwin) and 6 PPNG (all from Darwin) representing a total of 3.6% of strains that were penicillin-resistant in 2010 (4.2% in 2009, 3.9% in 2008, 4.1% in 2007, 4.6% in 2006).

Data on acquisition were available for 104 (23%) infections with PPNG. Sixty-two (13%) of these were acquired locally and 42 (9%) by overseas contact. These external contacts were principally in Western Pacific or South East Asian countries with those reported from Thailand; the Philippines; Indonesia and Vietnam the most numerous. Additionally, China, India, Singapore and, more widely the United Kingdom, were named as countries of acquisition.

Ceftriaxone

From 2001 onwards, low numbers of gonococcal isolates with raised ceftriaxone MIC values (in the range 0.06–0.125 mg/L, referred to as having decreased susceptibility) have been found in Australia. The proportion has increased incrementally with the data from recent years showing a rise from 0.6% in 2006, 0.8% in 2007, and 1.1% in 2008; to 2.0% in 2009. In 2010 an increase of isolates with decreased susceptibility to ceftriaxone was observed nationally: 191 of 3,997 (4.8%). There has not been an isolate of *N. gonorrhoeae* with an MIC value greater than 0.125 mg/L reported in Australia.

In South Australia 19 of 164 isolates (11.6%) had decreased susceptibility to ceftriaxone, and there were 52 of 908 (5.7%) from Victoria; 74 of 1,321 (5.6%) from New South Wales; 17 of 328 (5.2%) from Western Australia; and 26 of 823 (3.2%) from Queensland. There were 2 of 30 (6.7%) from the Australian Capital Territory; and 1 of 412 (0.2%) from the Northern Territory. There were no isolates with decreased susceptibility to ceftriaxone reported from Tasmania.

In 2010, there was a significant increase from 2009 in gonococci with decreased susceptibility to ceftriaxone in all jurisdictions with the exception of the Northern Territory and Tasmania, as shown in Table 2.

Table 2: Gonococcal isolates with decreased susceptibility to ceftriaxone,* Australia, 2010 and 2009, by state or territory

	2010		2009	
	n	%	n	%
Australian Capital Territory	2	6.7	2	5.3
New South Wales	74	5.6	16	1.7
Northern Territory	1	0.2	1	0.2
Queensland	26	3.2	10	1.8
South Australia	19	11.6	9	5.3
Tasmania	0	0.0	0	0.0
Victoria	52	5.7	17	2.2
Western Australia	17	5.2	9	3.1
Australia	191	4.8	64	2.0

* MIC value 0.06–0.125 mg/L

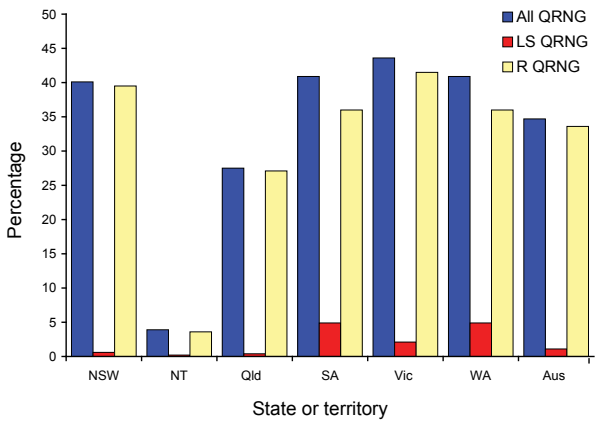
Spectinomycin

All isolates from all jurisdictions were again susceptible to this injectable antibiotic.

Quinolone antibiotics

Figure 2 shows the distribution of gonococci with altered susceptibility to quinolones nationally and by jurisdiction. Thus far resistance to the quinolone antibiotics in N. *gonorrhoeae* is mediated only by chromosomal mechanisms so that incremental increases in MIC values are observed. The AGSP uses ciprofloxacin as the representative quinolone and

Figure 2: Percentage of gonococcal isolates less sensitive to ciprofloxacin* or with higher level ciprofloxacin resistance† and all strains with altered quinolone susceptibility, Australia, 2010, by state or territory



* LS QRNG: MIC 0.06–0.5 mg/L

† R QRNG: MIC 1 mg/L or more

defines altered susceptibility as an MIC of 0.06 mg/L or more.[13] Treatment with currently recommended doses of 500 mg of ciprofloxacin is effective for strains with a lower level of resistance, viz. 0.06–0.5 mg/L, in about 90% of cases, but lower doses of the antibiotic will result in treatment failure more often. At higher levels of resistance i.e. a MIC of 1 mg/L or more, rates of failed treatment rise rapidly. At MIC levels of 4 mg/L or more treatment failure approaches 100%, even with higher ciprofloxacin doses.

Nationally in 2010, 1,385 of gonococci examined (35%) had some level of resistance to quinolones (QRNG), representing a further decrease in the proportion of quinolone resistance from 43% in 2009 and 54% in 2008. Most of the QRNG found in 2010 (1,342; 97%) had resistance at a higher level i.e. MICs ≥ 1 mg/L and the many of these had MIC levels of 8–64 mg/L.

In Victoria, 396 (44%) of all isolates examined were QRNG; with 67 (41%) in South Australia; 530 (40%) in New South Wales; 131, (40%) in Western Australia, and 226 (28%) in Queensland. In other jurisdictions, the number of QRNG remained low: in the Australian Capital Territory there were 18 (60%) QRNG isolated; 16 (4%) from the Northern Territory and 1 QRNG from Tasmania.

Information on country of acquisition of QRNG was available for 55 (4%) of the 1,385 cases. Forty-one of these (3%) were acquired locally and 14 (1%) from overseas from sources referred to under PPNG acquisition and with contacts additionally reported from the United States of America.

High-level tetracycline resistance

The spread of high-level tetracycline resistance in *N. gonorrhoeae* (TRNG) is examined as an epidemiological marker even though tetracyclines are not a recommended treatment for gonorrhoea and are rarely, if ever, used for treatment of gonorrhoea in Australia. Despite the lack of use of this antibiotic group, the proportion of TRNG detected continues to increase. In 2006, 12% of referred isolates were TRNG; increasing in 2007 (505 TRNG 16.6%) and again in 2008 (553 TRNG, 18%). In 2009, this increase continued with 650 (21%) TRNG detected, and this proportion was unchanged in 2010 (822 TRNG: 21%).

TRNG were present in all jurisdictions, with the highest proportion in the Northern Territory (111 TRNG, 27%); Western Australia (77 TRNG, 24%); New South Wales (310 TRNG 24%) and South Australia (34, 21%). Lower proportions of TRNG were present in Victoria (148, 16%) and Queensland (134, 16%). In the Australian Capital Territory there were 7 TRNG and 1 in Tasmania.

Discussion

World Health Organization recommendations for standardised treatment regimens for gonorrhoea are based on data from epidemiological surveys of both the distribution and extent of AMR in gonococci.[3] Antimicrobial resistance at a rate of 5% or more in gonococci sampled in a general population is the 'threshold' for removal of an antibiotic from treatment schedules and substitution with another, effective, agent.[3,15] Programs such as the AGSP seek to determine the proportion of antimicrobial resistance in gonococcal strains isolated in a defined patient population and relate these findings to the likely efficacy of current treatment schedules.[3,5,9,13,15] Surveillance strategies are dependent on quality AMR data, and the requirements for *in vitro* growth and AMR testing of the fastidious *N. gonorrhoeae* complicate this process. An important aspect of surveillance is to obtain and examine a sufficient and representative sample of isolates.[3,13,15] In 2010, the strains examined by the AGSP were sourced from the public and private health sectors, constituting a comprehensive sample that meets these requirements, in spite of the increasing use of nucleic acid amplification testing for diagnosis of gonorrhoea in Australia. The AGSP distributes reference panels for use in internal quality control practice and for External Quality Assurance Schemes,[14,16] which are necessary for validation of gonococcal AMR data.

The overall number of gonococcal strains examined by the AGSP in 2010 (3,997) was higher than the number examined in 2009 (3,220) and 2008 (3,192), but proportionally unchanged from approximately 40% of gonococcal case notifications in Australia. Isolate numbers in 2010 increased from those reported in 2009 in most jurisdictions excepting the Australian Capital Territory, and were unchanged for South Australia and Tasmania.

In 2010, 29% of gonococci nationally were resistant to the penicillins, and 35% to the quinolone antibiotics. These proportions were reduced from those reported nationally in 2009 (penicillin resistance, 36%; quinolone resistance, 43%), and in 2008 (penicillin resistance, 44%; quinolone resistance, 54%), where previously they have increased each year since 2003.[4] In 2010, there were decreased numbers of gonococci with both chromosomally-mediated resistance to penicillin and penicillinase production, whereas in 2008 and 2009 the reduction in penicillin resistance was primarily accounted for by the reduction of CMRP rates. Aggregated data have shown a predominant clone of CMRP coupled with high-level quinolone resistance circulating with increasing frequency annually since 2003.[4,12] It is possible that the

continued reduction in resistance to both penicillin and the quinolones in 2010 continues to reflect a 'clonal shift' in gonococcal isolates.

In 2010, the level in Australia of gonococci with high-level tetracycline resistance was low but stable despite low exposure to these antibiotics in Australia.[4] Evidence of the 'rural-urban divide',[4] in gonococcal resistance was maintained (Figures 1 and 2), underscoring the necessity for disaggregated information rather than pooled national data to define treatment regimens appropriate for the various jurisdictions. Remote areas in some jurisdictions with high disease rates continue to be able to use penicillin-based treatments, but effective use of this inexpensive and acceptable treatment is contingent on vigilant monitoring of resistance patterns.

Recent AGSP reports have drawn attention to the emergence and spread of gonococci in Australia that exhibit decreased susceptibility to the later generation cephalosporin antibiotics, also referred to as the extended spectrum cephalosporins. These gonococci have also been found in increasing numbers in the WHO Western Pacific Region.[17] In 'urban' Australia, the injectable agent ceftriaxone is now the standard treatment for gonorrhoea in public sector clinics, and is currently given by intramuscular injection in a dose of 500 mg. This dose is higher than the 250 mg dose that is more commonly used throughout the Western Pacific Region,[5] but 500 mg is the smallest volume vial currently available in Australia.

Decreased susceptibility to the ESCs has been accompanied by an increasing number of reports of treatment failures with the orally administered members of this group.[5–7] The decreased susceptibility to ESCs is quantified by the determination of the MIC value, and encompasses the range 0.06–0.125 mg/L. To date, there have been no strains of *N. gonorrhoeae* reported in Australia with an MIC value greater than 0.125 mg/L; nor substantiated reports of treatment failure in genital tract gonorrhoea following ceftriaxone therapy. However, concerns are escalating locally and globally as increasing proportions of strains with decreased susceptibility are reported. In Australia in 2010, the number of gonococcal isolates with decreased susceptibility to the ESCs (4.8%) more than doubled from 2009 (2%). This alarming increase underscores the need for continued high quality surveillance of AMR in *N. gonorrhoeae* with MIC values. Further, sentinel site surveillance in high risk populations is critically important to monitor instances of treatment failure. Sentinel site surveillance programs involve patient follow-up and test of cure (TOC) cultures after treatment of *N. gonorrhoeae* infections, in particular those in oropharyngeal sites. Sentinel site surveillance for AMR in *N. gonorrhoeae* is currently conducted in a very limited number of settings in Australia and needs to be expanded throughout all jurisdictions as a matter of priority.

There has been recent clarification of the mechanisms of resistance that are responsible for the MIC increases to ceftriaxone in gonococci. Attention has been paid particularly to the presence of 'mosaic' *penA* genes in gonococci with raised ESC MIC values. *penA* encodes penicillin binding protein 2 (PBP2), the major site of action of ceftriaxone and mosaic PBP2 are altered to reduce this activity. Additional gene polymorphisms that affect antibiotic access to the organism complement these PBP2 changes and further increase ESC MICs. Of recent interest has been an extension of a study from 2001 to 2005 on the dynamics of the spread of mosaic PBP2-containing gonococci (mPBP2-GC) in Australia. This initial investigation suggested that mPBP2-GC found locally were also present in Hong Kong (where they were associated with treatment failure with an oral ESC, ceftibuten),[7] and Japan.[6] Continuing studies in 2007 and 2008 showed that the subtypes of the mPBP2-GC present in Australia had altered markedly and that these strains had increased as a proportion of all gonococci tested.[18]

Also of relevance have been local studies that showed that other non-mosaic lesions in *penA* were also responsible for increases in ceftriaxone MIC values similar to those found in mosaic PBP2 containing gonococci.[19] These lesions were single nucleotide polymorphisms that represented mutations occurring in the *penA* of *N. gonorrhoeae*. This contrasted with the mosaic *penA* alteration, which results from acquisition of 'foreign' DNA by the gonococcus.[20] Despite these advances, not all the increases detected in ESC MIC levels can be explained by the molecular mechanisms described so far. This poses difficulties in developing reliable laboratory methods for the detection of ESC 'resistant' gonococci.

All gonococcal isolates tested in Australia in 2010, including those with altered cephalosporin susceptibility, were susceptible to spectinomycin. A low proportion of gonococci were also found to be resistant to azithromycin in 2010. Azithromycin has been suggested as a possible component of treatment for gonorrhoea that uses dual antibiotic treatment.[21] Resistance to azithromycin, widely used as an anti-chlamydial agent in conjunction with gonococcal treatment, has been reported with increasing frequency overseas. MIC levels in azithromycin-resistant gonococci have reached very high levels in Europe, but these strains have not been detected in Australia.

The emergence and spread of anti-microbial resistance in *N. gonorrhoeae* is a global public health issue, and evolving problems of emergence and

spread of resistance are complex and require attention to both disease control strategies and rational use of antibiotics.[10,22,23] Critically, both disease control strategies and the understanding of the global scope of AMR are informed by surveillance programs of AMR nationally and internationally. Continuing commitment and vigilance to surveillance of AMR in *N. gonorrhoeae* means that maintenance of culture-based systems will be required while this surveillance is still based on testing of gonococcal isolates.

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Antimicrobial susceptibility of *Staphylococcus aureus* isolated from hospital inpatients, 2009:

Report from the Australian Group on Antimicrobial Resistance

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

Abstract

In 2009, the Australian Group on Antimicrobial Resistance (AGAR) conducted a period-prevalence survey of clinical *Staphylococcus aureus* isolated from hospital inpatients. Thirty medical microbiology laboratories from each state and mainland territory participated. Specimens were collected more than 48 hours post-admission. Isolates were tested by Vitek2® (AST-P579 card) and by Etest for daptomycin. Nationally, the proportion of *S. aureus* that were MRSA was 33.6%, ranging from 27.3% in South Australia to 41.4% in New South Wales/Australian Capital Territory. Resistance to the non-β-lactam antimicrobials was common except for rifampicin, fusidic acid, daptomycin and high-level mupirocin. No resistance was detected for vancomycin, teicoplanin, quinupristin-dalfopristin or linezolid. Resistance in the methicillin susceptible *S. aureus* (MSSA) was rare apart from erythromycin (12%) and absent for vancomycin, teicoplanin, daptomycin, quinupristin-dalfopristin and linezolid. The proportion of methicillin resistant *S. aureus* (MRSA) has remained stable since the first AGAR inpatient survey in 2005 yet during the same time frame resistance to many antimicrobials, in particular tetracycline, trimethoprim-sulphamethoxazole and gentamicin, has significantly decreased. This suggests that non-multi-resistant community-associated MRSA (CA-MRSA) clones are becoming more common in the hospital setting and replacing the long-established multi-resistant clones such as ST239-III (Aus 2/3 EMRSA). Given hospital outbreaks of CA-MRSA are thought to be extremely rare it is most likely that patients colonised at admission with CA-MRSA have become infected with the colonising strain during their hospital stay. *Commun Dis Intell* 2011;35(3):237–243.

Keywords: antibiotic resistance, *Staphylococcus aureus*, nosocomial

Introduction

Staphylococcus aureus is a major pathogen both in the hospital environment and the wider community. It causes a wide variety of infections in man that are associated with considerable morbidity and significant mortality. Manifestations of *S. aureus* infection range from skin and soft tissue infections such as impetigo and furunculosis, to invasive infections such as osteomyelitis, necrotising pneumonia and infective endocarditis. Invasive infections are frequently associated with life-threatening bacteraemia infections. A study of 1,865 cases of *S. aureus* bacteraemia by the Australia New Zealand Cooperative on Staphylococcal Sepsis (ANZCOSS) has shown that all-cause 30-day mortality for *S. aureus* bacteraemia was 20.6%.[1] In Australia, as in most of the world, antimicrobial resistance in *S. aureus* is a major impediment to effective treatment. A subsequent ANZCOSS study of 3,430 bacteraemia cases showed that 30-day mortality varied significantly for isolates with different susceptibility patterns, with mortality increasing as resistance to the number of antimicrobials increased: mortality for methicillin susceptible *S. aureus* (MSSA) was 16.5%, for non-multi-resistant methicillin resistant *S. aureus* (MRSA) 19.4%, for ST22-IV-like MRSA (typically resistant to one or two non-β-lactam antimicrobials) 24.4% and for multi-resistant ST239-III-like MRSA 31.7%.[2]

Strategies exist to combat MRSA causing healthcare associated (HA) infections such as staff and patient screening, contact precautions, patient isolation and decolonisation of positive patients.[3] Although infection control strategies are expensive, the cost per MRSA infection is often more expensive: estimated to be €2, 730 in one Spanish hospital[4] and US$9,275 in a French intensive care unit (ICU).[5] Another effective option available to hospitals is to restrict the use of antimicrobials. A 70% reduction in cephalosporin usage resulted in a 30% reduction in MRSA cases in an Italian ICU despite being offset by increased fluoroquinolone use.[6] The United States of America successfully reduced the HA-MRSA infection rate from 1.4 to 0.6 episodes per 1,000 patient days after fluoroquinolone use was reduced by 34%.[7] An Australian cardiac surgical unit reported no cases of HA-MRSA surgical site infection (SSI) after changing antibiotic prophylaxis protocols from cefazolin to vancomycin and rifampicin. Prior to the intervention more than 50% of the SSIs in the unit were MRSA. The estimated cost saving was AUD$576,655 over the following 12 months based on the reduction of all SSIs.[8] Limited success in reducing MRSA transmission has been achieved through enhanced hand hygiene.[9,10] The Australian Group

for Antimicrobial Resistance (AGAR) has performed antimicrobial resistance period-prevalence surveys in Australia since 1986.[11] Presently, 30 laboratories from all states and mainland territories of Australia contribute to AGAR surveys. Hospital inpatient surveys have been conducted biennially since 2005, alternating with biennial community surveys.[12] The findings of the 2009 AGAR hospital inpatients survey are presented here and results compared to the two previous hospital inpatients surveys.

Methods

Thirty laboratories from all 6 states, the Australian Capital Territory and the Northern Territory participated in the *S. aureus* AGAR survey. From 1 July to 30 November 2009, each laboratory collected up to 100 consecutive *S. aureus* isolates from hospital inpatients (hospital stay > 48 hours at the time of specimen collection). Only 1 isolate per patient was tested. Each *S. aureus* isolate was judged to come from a potentially infected site; specimens received for the purpose of gathering surveillance data were excluded. Hospital laboratories collected only from one institution. The four private laboratories collected from any institution they serviced.

Species identification

S. aureus was identified by morphology and positive results of at least two of the following tests: slide coagulase test, tube coagulase test, appropriate growth on chromogenic agar and demonstration of deoxyribonuclease production. Additional tests such as fermentation of mannitol, growth on mannitol-salt agar or polymerase chain reaction for the presence of the *nuc* gene may have been performed for confirmation.

Susceptibility testing methodology

All isolates were tested using the Vitek2® AST-P579 card. All isolates with a penicillin minimum inhibitory concentration (MIC) of ≤ 0.125 mg/L were screened for the presence of β-lactamase using nitrocefin discs. The MIC to daptomycin was determined using Etest® strips (bioMerieux). Isolates with a daptomycin MIC > 1 mg/L were confirmed by broth microdilution. Results were interpreted for susceptibility according to Clinical Laboratory and Standards Institute breakpoints[13] except for mupirocin and fusidic acid.[14] Isolates with an MIC in the intermediate resistance category have been called resistant in this report.

Statistical analysis

The difference between proportions were tested using Chi-square test with alpha set at the 5% level and Fisher's exact test for 95% confidence limits (GraphPad® Prism Software). Relative risk and 95% confidence intervals (CI) were calculated using VassarStats (http://faculty.vassar.edu).

Results

To ensure institutional anonymity data were combined as follows: New South Wales with the Australian Capital Territory, Tasmania with Victoria, and Queensland with the Northern Territory (Table 1). There were 2,728 isolates included in the survey with the majority (75.6%) contributed by Victoria/Tasmania (26.5%), Queensland/Northern Territory (25.1%) and New South Wales/Australian Capital Territory (24.0%).

Table 1: Isolates by region

Region	Number of institutions	Total	%
NSW/ACT	8	655	24.0
Qld/NT	7	685	25.1
SA	3	282	10.3
Vic/Tas	8	723	26.5
WA	4	383	14.0
Total	30	2,728	100.0

Skin and soft tissue infection (SSTI) specimens contributed the majority (71.2%) of isolates followed by respiratory specimens (17.3%). Blood culture isolates contributed 6.1% of the total with significantly ($P < 0.0001$) more isolates causing non-invasive (91.9%) than invasive (8.1%) infections (Table 2).

The proportion of MRSA was 33.6% (95% CI 31.8%–35.4%) nationally (Table 3), with significantly different ($P < 0.0001$) proportions across Australia ranging from 27.3% (95% CI 22.2%–32.5%) in South Australia to 41.4% (95% CI 37.7%–45.2%) in New South Wales/Australian

Table 2: Source of isolates

Specimen source	n	%
Skin and soft tissue	1,942	71.2
Respiratory	473	17.3
Blood	167	6.1
Urine	93	3.4
Sterile body cavity	52	1.9
Cerebrospinal fluid	1	0.04
Total	2,728	100.0
Invasive	220	8.1
Non-invasive	2,508	91.9

Capital Territory. The proportion of non-invasive *S. aureus* that were MRSA (33.9%) was not significantly higher than for invasive isolates (30.0%) ($P = 0.241$). There were significant differences in the proportion of MRSA isolated in the 5 sources of infection ($P = 0.0002$) with MRSA isolated most commonly from urinary isolates (50.5% of the time) followed by respiratory specimens at 40.2% (Table 4).

The national proportion of MRSA in 2009 was 33.6%, which was not significantly different from the proportions identified in 2005 or 2007 (31.9% and 32.9% respectively, $P = 0.1823$) and the proportions were stable across all regions (Table 5).

Amongst the MRSA, resistance to the non-β-lactam antimicrobials was common except for fusidic acid, rifampicin, mupirocin and daptomycin where resistance was below 4% (Table 6 and Figure). Resistance was not detected for vancomycin, teicoplanin, quinupristin-dalfopristin or linezolid. Resistance levels varied between regions with New South Wales/Australian Capital Territory having the highest proportions for four of the top 5 antimicrobials for resistance. Compared with New South Wales/Australian Capital Territory, Western Australia had lower levels of resistance by 28 to 53 percentage points (PP) for erythromycin (28 PP), tetracycline (52 PP), trimethoprim-sulphamethoxazole (52 PP), ciprofloxacin (53 PP) and gentamicin (52 PP). For constitutive clindamycin resistance both South Australia and Western Australia had lower rates than the other states. Nearly half of MRSA (446/916, 48.7%) were multi-resistant (resistant to 3 or more non-β-lactams). The proportion of MRSA that

Table 3: Proportion of *Staphylococcus aureus* that were methicillin resistant, 2005 to 2009, by region and source

	All isolates			Invasive*			Non-invasive		
Region	n/N	%	95%CI	n/N	%	95%CI	n/N	%	95%CI
NSW/ACT	271/655	41.4	37.7–45.2	26/65	40.0	29.0–52.1	245/590	41.5	37.6–45.6
Qld/NT	210/685	30.7	27.3–34.2	14/49	28.6	17.9–42.4	196/636	30.8	27.4–34.5
SA	77/282	27.3	22.2–32.5	3/18	16.7	5.8–39.2	74/264	28.0	23.0–33.7
Vic/Tas	250/723	34.6	31.2–38.1	14/57	24.6	15.2–37.1	236/666	35.4	31.9–39.2
WA	108/383	28.2	23.9–32.9	9/31	29.0	16.1–46.6	99/352	28.1	23.7–33.0
Aus	916/2728	33.6	31.8–35.4	66/220	30.0	24.3–36.4	850/2508	33.9	32.1–35.87

* Blood/cerebrospinal fluid/sterile body cavity

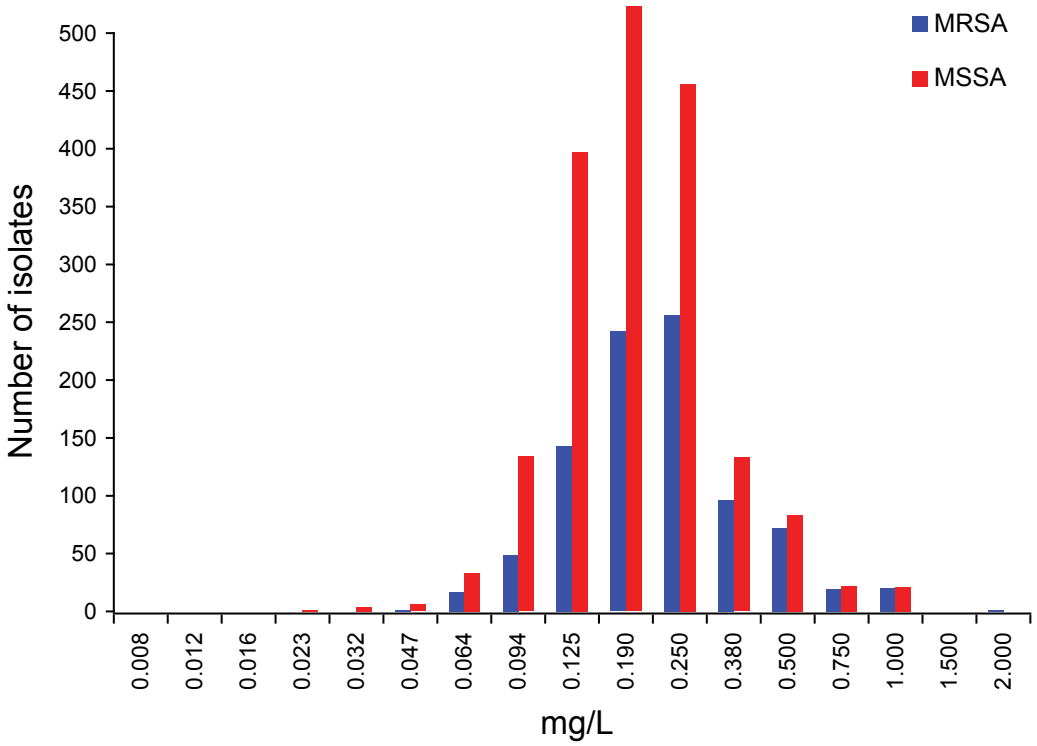
Table 4: Proportion of *Staphylococcus aureus* that were methicillin resistant, by specimen type

	All isolates		
Source of infection	n/N	%	95%CI
Skin and soft tissue	613/1,942	31.6	29.5–33.7
Respiratory	190/473	40.2	35.9–44.7
Blood/cerebrospinal fluid	57/168	33.9	27.2–41.4
Urine	47/93	50.5	40.6–60.5
Sterile body cavity	9/52	17.3	9.4–29.7

Table 5: Proportion of *Staphylococcus aureus* that were methicillin-resistant *Staphylococcus aureus*, 2005 to 2009

	Methicillin-resistant *Staphylococcus aureus*					
	NSW/ACT	Qld/NT	SA	Vic/Tas	WA	Aus
2005	43.4	26.7	24.7	31.6	22.5	31.9
2007	41.3	31.0	27.2	33.3	19.0	32.9
2009	41.4	30.7	27.3	34.6	28.2	33.6
X^2 for trend	0.6683	2.565	0.5669	1.419	3.452	1.779
P	0.4136	0.1093	0.4515	0.2336	0.0632	0.1823

Figure: Daptomycin minimum inhibitory concentration (susceptible MIC ≤ 1 mg/L)



were multi-resistant ranged from 11.1% in Western Australia to 59.4% in New South Wales/Australian Capital Territory (data not shown).

Some significant improvements in resistance to the non-β-lactams have occurred since the first AGAR hospital inpatients survey in 2005. Nationally, resistance has decreased to erythromycin (80.0% in 2005 to 70.9% in 2009, $P < 0.0001$), clindamycin (44.2% to 35.0%, $P < 0.0001$), tetracycline (59.4% to 45.1%, $P < 0.0001$), trimethoprim-sulphamethoxazole (60.3% to 41.6%, $P < 0.0001$), ciprofloxacin (76.8% to 71.2%, $P = 0.0052$), gentamicin (60.6% to 43.7%, $P < 0.0001$) and rifampicin (5.2% to 3.3%, $P = 0.048$) while resistance has remained stable to fusidic acid (4.3% to 3.1%, $P = 0.1621$) and high-level mupirocin (0.6% to 0.7%, $P = 0.979$). The national decreases in resistance may primarily be the result of significant regional decreases in New South Wales/Australian Capital Territory and Victoria/Tasmania particularly for erythromycin, tetracycline, trimethoprim-

Table 6: MSRA: Number and proportion resistant to the non-β-lactam antimicrobials, Australia, by region

	NSW/ACT (n=271)		Qld/NT (n=210)		SA n=77)		Vic/Tas (n=250)		WA (n=108)		Aus (n=916)		Difference across regions	
Drug	n	%	n	%	n	%	n	%	n	%	n	%	X^2	*P*
Erythromycin	212	78.2	141	67.1	56	72.7	186	74.4	54	50.0	649	70.9	32.93	<0.0001
Clindamycin*	138	50.9	79	37.6	9	11.7	84	33.6	11	10.2	321	35.0	78.63	<0.0001
Tetracycline	150	55.4	105	50.0	36	46.8	119	47.6	3	2.8	413	45.1	92.39	<0.0001
Trimethoprim-sulphamethoxazole	147	54.2	90	42.9	28	36.4	114	45.6	2	1.9	381	41.6	90.72	<0.0001
Ciprofloxacin	225	83.0	127	60.5	53	68.8	215	86.0	32	29.6	652	71.2	148.1	<0.0001
Gentamicin	150	55.4	103	49.1	33	42.9	111	44.4	3	2.8	400	43.7	90.99	<0.0001
Fusidic acid	4	1.5	11	5.2	3	3.9	7	2.8	3	2.8	28	3.1	5.924	0.2049
Rifampicin	2	0.7	16	7.6	0	0.0	10	4.0	2	1.9	30	3.3	19.21	0.0007
Mupirocin†	2	0.7	2	1.0	0	0.0	1	0.4	1	0.9	6	0.7	0.6890	0.9527

* Constitutive resistance

† High-level resistance

Table 8: MSSA: Number and proportion resistant to penicillin and the non-β-lactam antimicrobials, Australian, by region

Drug	NSW/ACT (n=384)		Qld/NT (n=475)		SA (n=205)		Vic/Tas (n=473)		WA (n=275)		Aus (n=1812)		Difference across regions	
	n	%	n	%	n	%	n	%	n	%	n	%	X^2	*P*
Penicillin	330	85.9	411	86.5	180	87.8	421	89.0	230	83.6	1,572	86.8	4.856	0.3024
Erythromycin	50	13.0	68	14.3	18	8.8	52	11.0	30	10.9	218	12.0	5.553	0.2351
Clindamycin*	12	3.1	5	1.1	2	1.0	5	1.1	1	0.4	25	1.4	11.66	0.0200
Tetracycline	22	5.7	4	0.8	8	3.9	8	1.7	9	3.3	51	2.8	21.96	0.0002
Trimethoprim-sulphamethoxazole	14	3.6	5	1.1	5	2.4	11	2.3	0	0.0	35	1.9	13.98	0.0074
Ciprofloxacin	12	3.1	4	0.8	4	2.0	10	2.1	10	3.6	40	2.2	8.282	0.0818
Gentamicin	10	2.6	2	0.4	1	0.5	9	1.9	0	0.0	22	1.2	14.83	0.0051
Fusidic acid	11	2.9	19	4.0	9	4.4	16	3.4	12	4.4	67	3.7	1.621	0.8050
Rifampicin	1	0.3	1	0.2	0	0.0	1	0.2	0	0.0	3	0.2	1.123	0.8906
Mupirocin†	0	0.0	7	1.5	0	0.0	3	0.6	1	0.4	11	0.6	9.763	0.0446

* Constitutive resistance

† High-level resistance

sulphamethoxazole and gentamicin. Significant falls in rifampicin resistance occurred in Queensland/ Northern Territory and South Australia.

In 2009, as in past AGAR hospital isolates surveys, increasing age was a risk factor for methicillin resistance (Table 7). Of 2,728 *S. aureus* isolates, 916 were MSRA (34%). Inpatients 41 years and older were 1.6 times more likely (RR 1.6, 95% CI 1.4–1.9) to have an MRSA not MSSA infection compared with younger patients.

Resistance to the non-β-lactams amongst methicillin susceptible *S. aureus* (MSSA) was rare apart from erythromycin (12.0% nationally) (Table 8). Resistance was not detected for vancomycin, teicoplanin, quinupristin-dalfopristin, daptomycin or linezolid. Multi-resistance was uncommon in MSSA (31/1812, 1.7%).

Nationally, there were no significant changes in the trends for resistance for MSSA in any of the antimicrobials tested. In Victoria/Tasmania, there was a significant increase in resistance in penicillin by 7 PP between 2005 and 2009 (82.0% and 89.0% respectively, $P = 0.0022$). Changes occurred in resistance patterns for tetracycline with a 3 PP decrease in resistance from 2005 and 2009 in Victoria/ Tasmania (5.1% and 1.7% respectively, $P = 0.0051$) and an increase by 3 PP for tetracycline resistance in Western Australia (0.0% to 3.3% respectively, $P = 0.0045$).

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

Age (years)	MRSA		Total tested
	n	%	
0–1	18	2.0	184
2–16	20	2.2	95
17–40	108	11.8	360
41–61	214	23.4	621
62–101	556	60.7	1,468
Total	916	100.0	2,728

Discussion

This survey demonstrates that MRSA remains a significant burden in Australian hospitals. However, the trend data generated may have some limitations. The mix of laboratories has altered over time with one fewer New South Wales and one fewer South Australian laboratory participating in the 2009 survey compared with the 2005 survey. However, an analysis of results of the 28 laboratories that participated in all surveys gave similar results with no changes to the statistical significance of the antimicrobial resistance trends in MRSA or MSSA either regionally or nationally.

For 2009, the national proportion of *S. aureus* that were MRSA was 33.6%, which was similar to the proportion in 2005 (31.9%, $P = 0.19$) and 2007 (32.9%, $P = 0.18$). Yet, differences between regions were significant with New South Wales/Australian Capital Territory having a higher proportion than other regions. Approximately a third of blood/CSF and skin and soft tissue *S. aureus* infections were methicillin resistant. The proportion for respiratory and urine specimens was higher with half of all

S. aureus isolated from urines having methicillin resistance. The overall proportion of MRSA in invasive (mainly bacteraemia) isolates was similar to that of non-invasive isolates (30.0% and 33.9% respectively, $P = 0.2724$). The high proportion of MRSA in invasive isolates is of concern as MRSA bacteraemia is associated with increased mortality compared with MSSA.[15,16] Direct comparison with prevalence in other countries is difficult due to methodological differences. For example, the European surveillance system reports the proportion of MRSA in bacteraemia isolates in both inpatients and outpatients. Amongst 198 continuous contributing laboratories in 22 European countries the proportion of MRSA compared with MSSA significantly decreased from 2002 to 2009. Targeted MRSA public health initiatives in several countries was cited as a possible cause of this decline. The overall proportion of MRSA in Europe in 2009 varied markedly from less than 1% in Iceland and Norway to 58% in Malta.[17] The Netherlands and the Scandinavian countries have been consistently able to keep MRSA at very low levels in their hospitals over long periods.

Amongst the MRSA in this study, more that 70% were resistant to erythromycin and ciprofloxacin, and more than 40% were resistant to tetracycline, trimethoprim-sulphamethoxazole and gentamicin. Regional differences were again common and this was due to the different MRSA clones circulating in Australia. In the 1980s and 1990s multi-resistant strains (later typed as ST239-III or Aus2/3 EMRSA) became epidemic in the eastern Australian states with some spread to hospitals in South Australia, the Northern Territory and Tasmania.[18]

In 1982, a state-wide MRSA policy was introduced in Western Australia with the aim of preventing these strains from becoming established in Western Australia hospitals. As a result, MRSA with tetracycline, trimethoprim-sulphamethoxazole and gentamicin resistance (characteristic of ST239-III) are rare in Western Australia—less than 3% in this survey. Erythromycin and ciprofloxacin resistance was more widespread in this survey with at least 30% of MRSA with this profile in any region. Erythromycin and ciprofloxacin resistance is common in ST239-III strains but is also characteristic of ST22-IV (EMRSA-15). ST22-IV is a common healthcare-associated MRSA (HA-MRSA) in Australia and is found in all regions.[19,20] Resistance was not detected for vancomycin, teicoplanin, quinupristin-dalfopristin or linezolid. Compared with previous AGAR hospital inpatient surveys in 2005 and 2007, the proportion of MRSA resistant to erythromycin, clindamycin, tetracycline, trimethoprim-sulphamethoxazole, ciprofloxacin, gentamicin and rifampicin has decreased nationally, lead by significant decreases in New South Wales/Australian Capital Territory and Victoria/Tasmania, whilst the proportion of *S. aureus* that are MRSA has remained stable in all regions and nationally. This finding suggests that non-multi-resistant community strains of MRSA are becoming more common in Australian hospitals at the expense of the long-established multi-resistant ST239-III.

Given reports of outbreaks of CA-MRSA in Australian hospitals are thought to be rare,[21,22] it is likely that many infections in hospital inpatients are caused by the patients' own colonising strains acquired prior to admission. It appears that current infection control procedures are successful in preventing their spread. Although at present in Australia there is no evidence of increasing resistance in local CA-MRSA,[23] data from the United States of America show that previously non-multi-resistant CA-MRSA can acquire multiple resistances over time.[24] With community clones such as the Queensland clone (ST93-IV), South Western Pacific (ST30-IV) and WA 1 (ST1-IV) well established in Australia,[12,25] it is important to monitor susceptibility patterns to MRSA over time as this information will guide therapeutic practices. In addition to this threat, virulent multi-resistant overseas CA-MRSA have recently been isolated in Australia[26] and only time will tell if these difficult to treat clones become established in the Australian community or healthcare institutions.

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Australian Paediatric Surveillance Unit annual report, 2010

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Background

National active surveillance of rare diseases of childhood, including infectious and vaccine preventable diseases, genetic disorders, childhood injuries and mental health conditions is conducted by the Australian Paediatric Surveillance Unit (APSU). The study of communicable and vaccine preventable diseases is supported in part by the Department of Health and Ageing (DoHA). In addition to conducting ongoing surveillance, the APSU has demonstrated readiness to respond rapidly to emerging diseases, epidemics and pandemics having severe impacts in children.[1] In 2010, the APSU conducted national surveillance for rare infections or vaccine preventable conditions resulting in significant impacts on the child and family. The conditions included: acute flaccid paralysis (AFP), acute rheumatic fever (ARF), congenital cytomegalovirus infection (cCMV), congenital rubella, perinatal exposure to HIV, neonatal herpes simplex virus (HSV) infection, congenital and neonatal varicella, severe complications of varicella, and severe complications of influenza infection. Surveillance for intussusception (IS) was conducted in response to the introduction of new rotavirus vaccines in Australia as IS was initially linked with the receipt of older rotavirus vaccines.[2] The APSU, together with the National Centre for Immunisation Research and Surveillance (NCIRS) coordinates the Paediatric Active Enhanced Disease Surveillance (PAEDS) system. PAEDS is a hospital based surveillance system including investigators from four tertiary paediatric hospitals in four states, and complements surveillance conducted by the APSU for AFP and IS.[3]

Methods

The APSU study protocols and case definitions are developed in collaboration with groups of investigators who have expertise in each of the conditions under surveillance. Detailed protocols, case definitions and contact details of the expert investigators for each condition are available at www.apsu.org.au The APSU sends monthly report cards listing the conditions under surveillance to approximately 1,300 paediatricians and other child health clinicians around Australia. Report cards are returned whether the clinician has a case to report or not, and the rate of returned report cards provides a measure of participation.[1,4] In 2010, approximately 85% of clinicians chose to receive and respond to the APSU report card via email. All reported cases are followed up by a questionnaire requesting de-identified data on the child's demographics, clinical presentation, treatment and short-term outcome. During surveillance for severe complications of influenza, clinicians were asked to return all questionnaires by fax as soon as children were identified, ensuring timely data collection. All questionnaires are reviewed by the study investigators before classification according to case definition criteria.

It is estimated that 92% of all paediatricians who have graduated with a Fellowship of the Royal Australasian College of Physicians (FRACP) and who are in active clinical practice in Australia, participate in the APSU. Lists of clinicians are updated to include new FRACP graduates and the APSU clinician database is constantly updated as clinicians change contact details, move out of clinical practice or retire. Despite high response rates to the report cards (average 96% per annum since 2000) complete case ascertainment is unlikely.[5] This is particularly relevant in remote communities where children have limited access to paediatricians or when hospital admission is brief and the child may not be seen by a paediatrician. The APSU encourages the use of complementary data sources where available, and reporting by a range of specialists to maximize case identification.[1,4] Reported rates for conditions ascertained through the APSU therefore represent a minimum estimate of the incidence of these conditions in the relevant Australian child populations as reported by the Australian Bureau of Statistics.[6]

The PAEDS[3] system developed by the APSU and the NCIRS complements data collected for several conditions, including AFP and IS. PAEDS enhances the surveillance effort, especially where hospital stays are minimal, where biological samples are required, and where a detailed history might be needed from parents or caregivers.

Results

In 2010, 1,330 clinicians participated in APSU surveillance. Consistent with previously reported high rates of participation, the report card return rate was 95%. Enhanced data about diagnosis, clinical management and short-term outcome were

available for more than 90% of all notified cases. The reported rate per 100,000 per annum of the relevant child population for each condition was calculated for 2010 as was an overall annual rate for the whole study period (Table).

All data are provided after review by the expert investigators responsible for each condition and are accurate as at May 2011. However, it is possible that some notifications may be reclassified at a later date, additional clinical data for existing notifications, or additional late notifications may be received and this will have an effect on final case counts reported over the last 12 months.

Acute flaccid paralysis

The introduction of the PAEDS hospital-based surveillance system has strengthened Australia's AFP surveillance for case ascertainment and has contributed to Australia achieving the World Health Organization (WHO) surveillance target of a non-polio AFP rate of ≥1 per 100,000 children

Table: Confirmed cases identified in 2010 and for the total study period, and reported rates per 100,000 of the relevant child population

Condition	Date study commenced	Questionnaire response rate (%)	Number of confirmed cases 2010	Reported rate for 2010 (per 100,000)	Number of confirmed cases for total study period*	Reported rate for total study period (per 100,000 per annum)†
Acute flaccid paralysis	Mar 1995	100	41*	1.0‡	598*	0.9‡
Congenital cytomegalovirus	Jan 1999	93	31	10.5§	191	5.8§
Acute rheumatic fever	Oct 2007	96	39	0.9‡	151	1.1‡
Congenital rubella (with defects)	May 1993	No notifications	Nil	Nil	50	0.1‖
Perinatal exposure to HIV**	May 1993	97	52	17.6§	437	9.2§
HIV infection**			5	1.7	77	1.6
Neonatal herpes simplex virus infection	Jan 1997	100	4	1.3§	121	3.2§
Congenital varicella	May 2006	No notifications	Nil	Nil	2	0.2§
Neonatal varicella	May 2006	50¶	1	0.3§	15	1.3§
Severe complications of varicella	May 2006	92	9	0.2‡	45	0.2‡
Intussusception	May 2007 to May 2010	88	9	2.5**	163	5.2**
Severe complications of Influenza	Influenza season each year since 2008††	90	25	1.8‡	179	1.8‡

* Includes all cases of acute flaccid paralysis (AFP) reported via the Australian Paediatric Surveillance Unit or the Paediatric Active Enhanced Disease Surveillance. All have been classified by the Polio Expert Panel as 'non-polio AFP' according to World Health Organization criteria.

† Includes 3 cases due to perinatal exposure and 2 cases due to exposure during medical procedures abroad.

‡ Based on population of children aged < 15 years.

§ Based on number of births.

‖ Based on population of children aged < 16 years.

¶ Two notifications only; one questionnaire received.

** Based on number of children aged ≤ 24 months.

†† Influenza surveillance was undertaken each year since 2008 during the influenza season, July to September except in the pandemic year (2009) when surveillance occurred from June to October.

All the figures were correct at the time of submission. As additional information becomes available cases may be reclassified for the current year and for previous years.

<15 years in 2010 for the third year in a row. After review of all cases by the Polio Expert Panel, the most common diagnoses of non-polio AFP was Guillain-Barré syndrome (36%) and acute disseminated encephalomyopathy (12%). Faecal specimens (2 within 14 days of onset of paralysis) were taken for 34% of cases however, only 22% were adequate for analysis by the National Polio Reference Laboratory. This is well below the WHO target, which is set at 80% of all reported cases—a target that is seldom achieved in developed countries. The importation of a type 1 wild poliovirus in an adult into Australia in 2007[7] and the continued detection of cases of wild polio internationally highlight the need for continued national surveillance to keep Australia polio-free.

Acute rheumatic fever

Between October 2007 and December 2010, 151 confirmed cases of ARF were reported from all states and territories of Australia, except for Tasmania and the Australian Capital Territory, suggesting the need for a national approach to the control of ARF and rheumatic heart disease. The majority of children identified as Aboriginal or Torres Strait Islander (87%), however there were 10 Caucasian children and 8 children of Pacific Islander ethnicity. A similar breakdown of ethnicity was noted in an audit of a tertiary children's hospital in Sydney.[8] Approximately 66% of all children resided in small rural towns or remote areas, with the other 34% resided in urban or suburban areas. The most common presenting symptoms in children diagnosed with ARF included carditis, polyarthritis and fever. All children were prescribed long-term prophylactic treatment with Benzathine Penicillin G to prevent recurrences and progression to rheumatic heart disease (RHD). The newly established organisation, Rheumatic Heart Disease Australia (http://www.rhdaustralia.org.au/) will further develop a national approach to ARF and RHD control in Australia.

Congenital cytomegalovirus infection

cCMV is the most common infectious cause of congenital malformations in Australia.[9] APSU data show that cCMV infection is not associated with maternal illness in approximately one third of cases, and should be considered regardless of maternal history.[9] There were 191 cases confirmed by the end of 2010, however cCMV remains under-diagnosed. Polymerase chain reaction analysis of newborn screening cards may retrospectively identify additional cases to further describe the impact of cCMV. Universal neonatal hearing screening programs may also help identify new cases. Despite most infants (90%) being symptomatic very few are treated with antivirals (8%).[10] This study continues to inform the ongoing debate about the need for routine screening of mothers and infants.

Congenital rubella (with defects)

There were no notifications of congenital rubella in 2010 supporting the effectiveness of the rubella vaccination program. In 2008 and 2009 there were 3 notifications of congenital rubella to the APSU. At the time of publication in 2010, detailed clinical data were not yet available,[11] however it is now known that two of these notifications were prevalent rather than incident cases. The other child, notified in 2008 was confirmed as an incident case of congenital rubella. The child was born in Australia to an immigrant woman from India whose vaccination history was unclear. The risk of congenital rubella remains, particularly among immigrant women born in countries with poorly developed vaccination programs, justifying continued surveillance.[12] Such women should have serological testing for rubella after arrival in Australia, and vaccination when appropriate. Travel to rubella endemic counties in the first trimester by women with no prior rubella immunity poses a risk of congenital rubella to the foetus.

Perinatal exposure to HIV and HIV infection

In 2010 there were 52 cases of perinatal exposure to HIV, three of which acquired HIV infection perinatally. In addition, there were 2 children with HIV infection who were exposed to contaminated blood products overseas. HIV infection was diagnosed prior to, or at the birth of the child in 46 (85.5%) of the mothers, enabling use of interventions including use of antiretroviral treatment (n=45), and avoidance of breast-feeding (n=43). HIV infection among children remains a rare occurrence in Australia, however, the number of reported cases of perinatal exposure has increased and may possibly be attributed to the availability of interventions to minimise the risk of mother-to-child transmission among women who know their HIV status prior to pregnancy. Of concern is the small number of mothers whose HIV infection was not known until after the birth of their child, precluding the use of interventions to minimise the risk of mother-to-child HIV transmission.[13,14]

Neonatal herpes simplex virus infection

Significant numbers of cases of neonatal HSV continue to be confirmed, with preponderance in female infants. The incidence over the last 14 years has remained steady, however the survival of infants has improved. This may be due to changes to recommendations for treatment with the use of higher doses of antiviral agents recommended since 2003.[15] Furthermore, the method of diagnosis has changed over the last 14 years with a move to more sensitive molecular techniques, which may potentially lead to earlier accurate diagnosis enabling earlier treatment. This study has also demonstrated

a change in the HSV strain causing infection in recent years, with an increasing number of cases due to HSV-1, whereas previously HSV-2 was almost exclusively detected in cases of neonatal HSV in Australia.[16] There is a need for ongoing surveillance to describe potential relationships between early detection and treatment with outcome.

Intussusception

There were 162 cases reported to the APSU during the period May 2007 to May 2010. Adequate information about immunisation status was available for only 80 cases, 14 of whom received a rotavirus vaccine within 14 days of developing IS. As the number of reports through the APSU was very small, the data were combined with cases collected via the PAEDS system. The combined data showed that there was a slightly elevated risk of IS following the 1st dose of a rotavirus vaccine, among young infants aged 1–2 months.[2]

Severe complications of varicella infection

In 2010, 9 children hospitalised with severe complications of varicella were reported. The complications included septic shock, focal purulent collection, and ataxia. Median stay in hospital was 8 days and 6 children were admitted to a paediatric Intensive Care Unit. All of the reported children were unvaccinated and most of the infecting contacts were close family members or other children in the school and pre-school setting.

Congenital and neonatal varicella

There was only 1 case of neonatal varicella reported in 2010. The incidence rate of neonatal varicella for the current surveillance study period (2006–2010) was 1.3 per 100,000 live births per annum—a considerable reduction when compared with the previous surveillance study conducted by the APSU during 1995–97, when the incidence was estimated at 5.8 per 100,000 live births per annum.[16] No cases of congenital varicella have been reported since 2008, supporting the effectiveness of the varicella vaccination program, which began at the end of 2005.

Severe complications of influenza

In 2010, 25 children hospitalised with severe complications of influenza were reported to the APSU and most (64%) had influenza A H1N1 2009. In contrast, in 2009, 100 children with severe complications were reported to the APSU; 77% had pandemic influenza H1N1 2009, but H3N2 was also detected. A range of complications were reported with x-ray confirmed pneumonia most common during both years. However, in 2009 serious complications such as encephalitis and rhabdomyolysis were more common than in 2010. Although a smaller number of children were reported in 2010 compared with 2009, a similar proportion was admitted to a paediatric Intensive Care Unit (44% in 2010 compared with 38% in 2009). There were 2 deaths in 2010 compared with 7 deaths in 2009. Vaccination for seasonal influenza was uncommon in 2010 even in the 2 children with pre-existing chronic disorders and eligible for free vaccination.[17]

Conclusions and future directions

APSU data contribute significantly to the national surveillance effort, providing valuable information for clinicians, policymakers and the community.[10,11,16] The APSU is often the only source of national data that includes clinical and/or laboratory details, and data on both inpatients and outpatients.[10,11]

After demonstrating the feasibility of the APSU to respond rapidly to an outbreak of influenza in 2007, the APSU has conducted surveillance for influenza in 2008, 2009, and 2010, providing a unique and detailed dataset on severe complications of influenza in children and a publication describing the impact of the severe complications of influenza from 2008 to 2010 is in preparation. The APSU will once again conduct surveillance for the severe complications of influenza from June to September in 2011.

Surveillance for juvenile respiratory papillomatosis will commence in the second half of 2011. Respiratory papillomatosis is a rare but devastating condition in children aged less than 12 years, and is thought to be perinatally transmitted.[17] Juvenile respiratory papillomatosis is difficult to treat, recurrences are common, and may lead to airway obstruction. The Human Papillomavirus (HPV) vaccine, which protects against HPV6 and HPV11, is currently nationally recommended and it is hoped that the rates of juvenile papillomatosis among young children will reduce with increased vaccination rates.

The APSU continues to provide useful data and clinical and public health insights relating to infectious diseases in Australian children. Ongoing surveillance through the PAEDS system will continue to complement the work of the APSU, and both APSU and PAEDS provide a platform for the rapid response to potential emerging infectious diseases threatening Australian children.

APSU currently conducts surveillance for other rare conditions of childhood (www.apsu.org.au) and is also involved in the study of impacts of rare diseases on families, clinicians and health services. This endeavour will be further supported by an Australian Research Council Linkage Grant (LP110200277).

The APSU has advocated for the development and adoption of a coordinated national plan for rare diseases in Australia and drafted a rationale for such a plan with the support of the Australian Research Alliance for Children and Youth. The APSU collaborated with the Department of Population Genomics, Western Australian Department of Health to organise the *Awakening Australia to Rare Diseases Symposium,* a first national symposium on rare diseases in Australia, held in April 2011 and attended by people affected by rare diseases, patient support groups, researchers, clinicians, government representatives and industry. A significant outcome of the symposium was the establishment of the National Rare Diseases Organising Committee, which will advocate for the further development and adoption of a national plan for rare diseases.

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Communicable diseases surveillance

Tables

National Notifiable Diseases Surveillance System

A summary of diseases currently being reported by each jurisdiction is provided in Table 1. There were 53,422 notifications to the National Notifiable Diseases Surveillance System (NNDSS) with a notification received date between 1 April and 30 June 2011 (Table 2). The notification rate of diseases per 100,000 population for each state or territory is presented in Table 3.

Table 1: Reporting of notifiable diseases by jurisdiction

Disease	Data received from:
Bloodborne diseases	
Hepatitis (NEC)	All jurisdictions
Hepatitis B (newly acquired)	All jurisdictions
Hepatitis B (unspecified)	All jurisdictions
Hepatitis C (newly acquired)	All jurisdictions except Queensland
Hepatitis C (unspecified)	All jurisdictions
Hepatitis D	All jurisdictions
Gastrointestinal diseases	
Botulism	All jurisdictions
Campylobacteriosis	All jurisdictions except New South Wales
Cryptosporidiosis	All jurisdictions
Haemolytic uraemic syndrome	All jurisdictions
Hepatitis A	All jurisdictions
Hepatitis E	All jurisdictions
Listeriosis	All jurisdictions
STEC, VTEC*	All jurisdictions
Salmonellosis	All jurisdictions
Shigellosis	All jurisdictions
Typhoid	All jurisdictions
Quarantinable diseases	
Cholera	All jurisdictions
Highly pathogenic avian influenza in humans	All jurisdictions
Plague	All jurisdictions
Rabies	All jurisdictions
Severe acute respiratory syndrome	All jurisdictions
Smallpox	All jurisdictions
Viral haemorrhagic fever	All jurisdictions
Yellow fever	All jurisdictions
Sexually transmissible infections	
Chlamydial infection	All jurisdictions
Donovanosis	All jurisdictions
Gonococcal infection	All jurisdictions
Syphilis <2 years duration	All jurisdictions
Syphilis >2 years or unspecified duration	All jurisdictions except South Australia
Syphilis - congenital	All jurisdictions

Table 1: Reporting of notifiable diseases by jurisdiction, *continued*

Disease	Data received from:
Vaccine preventable diseases	
Diphtheria	All jurisdictions
Haemophilus influenzae type b	All jurisdictions
Influenza (laboratory confirmed)	All jurisdictions
Measles	All jurisdictions
Mumps	All jurisdictions
Pertussis	All jurisdictions
Pneumococcal disease (invasive)	All jurisdictions
Poliomyelitis	All jurisdictions
Rubella	All jurisdictions
Rubella - congenital	All jurisdictions
Tetanus	All jurisdictions
Varicella zoster (chickenpox)	All jurisdictions except New South Wales
Varicella zoster (shingles)	All jurisdictions except New South Wales
Varicella zoster (unspecified)	All jurisdictions except New South Wales
Vectorborne diseases	
Arbovirus infection (NEC)	All jurisdictions
Barmah Forest virus infection	All jurisdictions
Dengue virus infection	All jurisdictions
Japanese encephalitis virus infection	All jurisdictions
Kunjin virus infection	All jurisdictions
Malaria	All jurisdictions
Murray Valley encephalitis virus infection	All jurisdictions
Ross River virus infection	All jurisdictions
Zoonoses	
Anthrax	All jurisdictions
Australian bat lyssavirus	All jurisdictions
Brucellosis	All jurisdictions
Leptospirosis	All jurisdictions
Lyssavirus (NEC)	All jurisdictions
Ornithosis	All jurisdictions
Q fever	All jurisdictions
Tularaemia	All jurisdictions
Other bacterial infections	
Legionellosis	All jurisdictions
Leprosy	All jurisdictions
Meningococcal infection	All jurisdictions
Tuberculosis	All jurisdictions

* Infections with Shiga-like toxin (verotoxin) producing *Escherichia coli* (STEC/VTEC).

NEC Not elsewhere classified.

Table 2: Notifications of diseases received by state and territory health authorities, 1 April to 30 June 2011, by date of diagnosis

Disease	State or territory								Total 2nd quarter 2011	Total 1st quarter 2011	Total 2nd quarter 2010	Last 5 years mean 2nd quarter	Ratio	Year to date 2011	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Bloodborne diseases															
Hepatitis (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.2
Hepatitis B (newly acquired)*	1	7	0	11	1	6	10	6	42	48	56	69.8	0.6	90	135.8
Hepatitis B (unspecified)†	16	597	51	239	80	18	484	172	1,657	1,742	1,842	1,662.2	1.0	3,388	3,379.8
Hepatitis C (newly acquired)*‡	2	9	0	NN	9	10	5	34	69	108	101	97.4	0.7	177	196.0
Hepatitis C (unspecified)†	49	772	52	607	102	47	600	249	2,478	2,597	2,995	2,818.2	0.9	5,056	5,816.8
Hepatitis D	0	0	0	0	0	0	7	2	9	11	7	9.6	0.9	20	18.8
Gastrointestinal diseases															
Botulism	0	1	0	0	0	0	0	0	1	0	0	0.0	0.0	1	0.4
Campylobacteriosis§	94	NN	47	1,253	451	180	1,514	485	4,024	4,939	3,347	3,586.0	1.1	8,895	7,898.8
Cryptosporidiosis	0	106	9	120	48	8	74	106	471	638	399	717.6	0.7	1,104	2,073.4
Haemolytic uraemic syndrome	0	1	0	0	0	0	0	0	1	4	1	3.4	0.3	5	8.6
Hepatitis A	0	13	0	8	1	0	6	3	31	45	49	81.4	0.4	75	162.8
Hepatitis E	0	7	0	1	0	0	0	0	8	16	11	8.4	1.0	24	19.4
Listeriosis	0	6	0	2	2	0	8	1	19	19	13	12.2	1.6	38	38.6
STEC, VTEC‖	0	3	1	2	6	1	1	2	16	19	12	17.0	0.9	35	50.8
Salmonellosis	13	688	90	686	211	33	574	264	2,559	4,807	2,893	2,262.0	1.1	7,329	5,647.2
Shigellosis	2	27	12	15	7	0	18	19	100	164	125	149.6	0.7	262	340.0
Typhoid	0	6	1	2	1	1	11	2	24	57	29	22.8	1.1	81	55.0
Quarantinable diseases															
Cholera	0	0	0	4	0	0	0	0	4	1	0	0.4	10.0	5	1.2
Highly pathogenic avian influenza in humans	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Plague	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rabies	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Severe acute respiratory syndrome	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Smallpox	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Viral haemorrhagic fever	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Yellow fever	0	0	0	2	0	0	0	0	2	0	0	0.0	0.0	2	0.0

Table 2 *continued*: Notifications of diseases received by state and territory health authorities, 1 April to 30 June 2011, by date of diagnosis

Disease	State or territory								Total 2nd quarter 2011	Total 1st quarter 2011	Total 2nd quarter 2010	Last 5 years mean 2nd quarter	Ratio	Year to date 2011	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Sexually transmissible infections															
Chlamydial infection¶,**	318	5,091	652	4,684	1,276	412	4,714	3,063	20,210	20,824	18,844	15,094.2	1.3	40,927	30,071.6
Donovanosis	0	0	0	0	0	0	0	0	0	0	0	1.2	0.0	0	1.8
Gonococcal infection**	34	663	513	757	122	6	554	443	3,092	2,966	2,570	2,288.2	1.4	6,037	4,474.4
Syphilis < 2 years duration**	2	97	8	66	17	0	61	41	292	368	291	321.4	0.9	657	628.8
Syphilis > 2 years or unspecified duration**	5	58	20	43	NDP	3	152	14	295	325	316	333.4	0.9	619	666.8
Syphilis – congenital**	0	0	0	0	0	0	0	0	0	4	1	2.2	0.0	4	3.6
Vaccine preventable diseases															
Diphtheria	0	0	1	3	0	0	0	0	4	0	0	0.0	0.0	4	0.0
Haemophilus influenzae type b	0	2	1	1	0	0	1	0	5	3	6	6.2	0.8	8	10.2
Influenza (laboratory confirmed)	38	900	34	1,674	931	77	332	122	4,108	2,674	786	3,537.6	1.2	6,756	3,918.6
Measles	0	11	2	5	0	0	8	4	30	80	15	29.8	1.0	109	59.0
Mumps	0	16	0	8	3	1	5	5	38	39	26	62.2	0.6	77	123.4
Pertussis	203	2,893	84	1,971	564	31	1,868	399	8,013	10,740	5,400	3,707.0	2.2	18,652	7,449.0
Pneumococcal disease (invasive)	7	145	33	105	45	5	138	70	548	224	422	408.8	1.3	769	612.2
Poliomyelitis	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Rubella	0	5	0	2	1	0	1	6	15	22	11	12.0	1.3	37	20.8
Rubella – congenital	0	0	0	0	0	0	0	0	0	0	0	0.4	0.0	0	0.4
Tetanus	0	0	0	1	0	0	0	1	2	1	0	0.2	10.0	3	1.8
Varicella zoster (chickenpox)††	2	NN	20	51	84	9	151	83	400	376	351	318.0	1.3	773	612.6
Varicella zoster (shingles)††	4	NN	46	9	404	44	199	190	896	1,013	696	500.2	1.8	1,903	1,037.4
Varicella zoster (unspecified)††	22	NN	0	926	12	21	551	254	1,786	1,794	1,660	1,216.6	1.5	3,564	2,497.6
Vectorborne diseases															
Arbovirus infection (NEC)	0	0	0	4	0	0	2	0	6	4	0	3.6	1.7	10	10.2
Barmah Forest virus infection	0	95	24	174	23	1	34	39	390	841	382	488.2	0.8	1,228	1,092.4
Dengue virus infection	5	18	3	21	2	1	17	53	120	361	264	138.6	0.9	479	434.2
Japanese encephalitis virus infection	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Kunjin virus infection‡‡	0	0	1	0	0	0	0	0	1	0	1	0.6	1.7	1	1.4
Malaria	2	17	3	30	1	2	19	14	88	120	94	143.4	0.6	207	285.6
Murray Valley encephalitis virus infection‡‡	0	1	0	0	0	0	0	6	7	8	0	0.8	8.8	15	1.4
Ross River virus infection	2	164	42	331	85	1	187	172	984	3,071	1,873	1,394.2	0.7	4,033	3,516.4

Table 2 *continued*: Notifications of diseases received by state and territory health authorities, 1 April to 30 June 2011, by date of diagnosis

Disease	State or territory								Total 2nd quarter 2011	Total 1st quarter 2011	Total 2nd quarter 2010	Last 5 years mean 2nd quarter	Ratio	Year to date 2011	Last 5 years YTD mean
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA							
Zoonoses															
Anthrax	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.6
Australian bat lyssavirus	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Brucellosis	0	1	0	8	0	0	1	1	11	12	4	7.8	1.4	23	17.4
Leptospirosis	0	13	0	32	1	0	4	1	51	124	44	41.2	1.2	175	87.2
Lyssavirus (NEC)	0	0	0	0	0	0	0	0	0	0	0	0.0	0.0	0	0.0
Ornithosis	0	4	0	0	0	0	12	1	17	24	9	24.8	0.7	40	48.6
Q fever	1	26	1	37	2	0	4	1	72	82	98	90.4	0.8	154	189.6
Tularaemia	0	0	0	0	0	0	0	0	0	1	0	0.0	0.0	1	0.0
Other bacterial infections															
Legionellosis	1	32	0	11	10	1	20	25	100	81	83	83.6	1.2	180	156.8
Leprosy	0	1	0	0	0	0	2	0	3	0	2	2.2	1.4	3	4.8
Meningococcal infection§§	1	13	0	15	4	3	16	5	57	57	56	61.6	0.9	114	112.6
Tuberculosis	4	89	7	63	17	3	58	25	266	292	286	264.8	1.0	555	552.4
Total	828	12,598	1,758	13,984	4,523	925	12,423	6,383	53,422	61,746	46,471			114,704	

* Newly acquired hepatitis includes cases where the infection was determined to be acquired within 24 months prior to diagnosis.

† Unspecified hepatitis and syphilis includes cases where the duration of infection could not be determined.

‡ In Queensland, includes incident hepatitis cases.

§ Not reported for New South Wales where it is only notifiable as 'foodborne disease' or 'gastroenteritis in an institution'.

|| Infections with Shiga-like toxin (verotoxin) producing *Escherichia coli* (STEC/VTEC).

¶ Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral, throat and eye samples, except for South Australia, which reports only genital tract specimens. The Northern Territory and Western Australia, exclude ocular infections.

** In the national case definitions for chlamydial, gonococcal and syphilis infections the mode of transmission cannot be inferred from the site of infection. Transmission (especially in children) may be by a non-sexual mode (e.g. perinatal infections, epidemic gonococcal conjunctivitis).

†† Ratio of current quarter total to the mean of last 5 years for the same quarter. Ratios for varicella zoster (chickenpox), varicella zoster (shingles) and varicella zoster (unspecified) are based on 4 years of data.

‡‡ In the Australian Capital Territory, Murray Valley encephalitis virus infection and Kunjin virus infection are combined under Murray Valley encephalitis virus infection.

§§ Only invasive meningococcal disease is nationally notifiable. However, New South Wales, the Australian Capital Territory and South Australia also report conjunctival cases.

NN Not notifiable.

NEC Not elsewhere classified.

NDP No data provided.

Table 3: Notification rates of diseases, 1 April to 30 June 2011, by state or territory. (Annualised rate per 100,000 population)

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Bloodborne diseases									
Hepatitis (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis B (newly acquired)*	1.1	0.4	0.0	1.0	0.2	4.7	0.7	1.0	0.8
Hepatitis B (unspecified)†	17.8	33.0	88.8	21.2	19.5	14.2	34.9	30.0	29.7
Hepatitis C (newly acquired)*	2.2	0.5	0.0	NN	2.2	7.9	0.4	5.9	1.5
Hepatitis C (unspecified)†,‡	54.6	42.7	90.6	53.8	24.8	37.0	43.3	43.4	44.4
Hepatitis D	0.0	0.0	0.0	0.0	0.0	0.0	0.5	0.3	0.2
Gastrointestinal diseases									
Botulism	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Campylobacteriosis§	104.8	NN	81.9	111.0	109.7	141.8	109.2	84.5	106.6
Cryptosporidiosis	0.0	5.9	15.7	10.6	11.7	6.3	5.3	18.5	8.4
Haemolytic uraemic syndrome	0.0	0.1	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Hepatitis A	0.0	0.7	0.0	0.7	0.2	0.0	0.4	0.5	0.6
Hepatitis E	0.0	0.4	0.0	0.1	0.0	0.0	0.0	0.0	0.1
Listeriosis	0.0	0.3	0.0	0.2	0.5	0.0	0.6	0.2	0.3
STEC,VTEC‖	0.0	0.2	1.7	0.2	1.5	0.8	0.1	0.3	0.3
Salmonellosis	14.5	38.0	156.7	60.8	51.3	26.0	41.4	46.0	45.8
Shigellosis	2.2	1.5	20.9	1.3	1.7	0.0	1.3	3.3	1.8
Typhoid fever	0.0	0.3	1.7	0.2	0.2	0.8	0.8	0.3	0.4
Quarantinable diseases									
Cholera	0.0	0.0	0.0	0.4	0.0	0.0	0.0	0.0	0.1
Human pathogenic avian influenza in humans	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Plague	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rabies	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Severe acute respiratory syndrome	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Smallpox	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Viral haemorrhagic fever	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Yellow fever	0.0	0.0	0.0	0.2	0.0	0.0	0.0	0.0	0.0
Sexually transmitted infections									
Chlamydial infection¶,**	354.4	281.3	1,135.5	414.8	310.3	324.6	339.9	533.5	361.8
Donovanosis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Gonococcal infection**	37.9	36.6	893.4	67.0	29.7	4.7	39.9	77.2	55.4
Syphilis < 2 years duration**	2.2	5.4	13.9	5.8	4.1	0.0	4.4	7.1	5.2
Syphilis > 2 years or unspecified duration†,**	5.6	3.2	34.8	3.8	NDP	2.4	11.0	2.4	5.7
Syphilis – congenital**	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Vaccine preventable diseases									
Diphtheria	0.0	0.0	1.7	0.3	0.0	0.0	0.0	0.0	0.1
Haemophilus influenzae type b	0.0	0.1	1.7	0.1	0.0	0.0	0.1	0.0	0.1
Influenza (laboratory confirmed)	42.4	49.7	59.2	148.3	226.4	60.7	23.9	21.3	73.5
Measles	0.0	0.6	3.5	0.4	0.0	0.0	0.6	0.7	0.5
Mumps	0.0	0.9	0.0	0.7	0.7	0.8	0.4	0.9	0.7
Pertussis	226.3	159.9	146.3	174.6	137.2	24.4	134.7	69.5	143.5
Pneumococcal disease (invasive)	7.8	8.0	57.5	9.3	10.9	3.9	10.0	12.2	9.8
Poliomyelitis	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Rubella	0.0	0.3	0.0	0.2	0.2	0.0	0.1	1.0	0.3
Rubella – congenital	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Tetanus	0.0	0.0	0.0	0.1	0.0	0.0	0.0	0.2	0.0

Table 3 *continued*: Notification rates of diseases, 1 April to 30 June 2011, by state or territory. (Annualised rate per 100,000 population)

Disease	State or territory								
	ACT	NSW	NT	Qld	SA	Tas	Vic	WA	Aust
Varicella zoster (chickenpox)	2.2	NN	34.8	4.5	20.4	7.1	10.9	14.5	10.6
Varicella zoster (shingles)	4.5	NN	80.1	0.8	98.3	34.7	14.3	33.1	23.7
Varicella zoster (unspecified)	24.5	NN	0.0	82.0	2.9	16.5	39.7	44.2	47.3
Vectorborne diseases									
Arbovirus infection (NEC)	0.0	0.0	0.0	0.4	0.0	0.0	0.1	0.0	0.1
Barmah Forest virus infection	0.0	5.2	41.8	15.4	5.6	0.8	2.5	6.8	7.0
Dengue virus infection	5.6	1.0	5.2	1.9	0.5	0.8	1.2	9.2	2.1
Japanese encephalitis virus infection	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Kunjin virus infection††	0.0	0.0	1.7	0.0	0.0	0.0	0.0	0.0	0.0
Malaria	2.2	0.9	5.2	2.7	0.2	1.6	1.4	2.4	1.6
Murray Valley encephalitis virus infection††	0.0	0.1	0.0	0.0	0.0	0.0	0.0	1.0	0.1
Ross River virus infection	2.2	9.1	73.1	29.3	20.7	0.8	13.5	30.0	17.6
Zoonoses									
Anthrax	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Australia bat lyssavirus	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Brucellosis	0.0	0.1	0.0	0.7	0.0	0.0	0.1	0.2	0.2
Leptospirosis	0.0	0.7	0.0	2.8	0.2	0.0	0.3	0.2	0.9
Lyssavirus (NEC)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Ornithosis	0.0	0.2	0.0	0.0	0.0	0.0	0.9	0.2	0.3
Q fever	1.1	1.4	1.7	3.3	0.5	0.0	0.3	0.2	1.3
Tularaemia	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0
Other bacterial diseases									
Legionellosis	1.1	1.8	0.0	1.0	2.4	0.8	1.4	4.4	1.8
Leprosy	0.0	0.1	0.0	0.0	0.0	0.0	0.1	0.0	0.1
Meningococcal infection‡‡	1.1	0.7	0.0	1.3	1.0	2.4	1.2	0.9	1.0
Tuberculosis	4.5	4.9	12.2	5.6	4.1	2.4	4.2	4.4	4.8

* Newly acquired hepatitis includes cases where the infection was determined to be acquired within 24 months prior to diagnosis.

† Unspecified hepatitis and syphilis includes cases where the duration of infection could not be determined.

‡ In Queensland, includes incident hepatitis C cases.

§ Notified as 'foodborne disease' or 'gastroenteritis in an institution' in New South Wales.

|| Infection with Shiga toxin/verotoxin-producing *Escherichia coli* (STEC/VTEC).

¶ Includes *Chlamydia trachomatis* identified from cervical, rectal, urine, urethral, throat and eye samples, except for South Australia, which reports only genital tract specimens; the Northern Territory and Western Australia exclude ocular infections.

** In the national case definitions for chlamydial, gonococcal and syphilis infections the mode of transmission cannot be inferred from the site of infection. Transmission (especially in children) may be by a non-sexual mode (e.g. perinatal infections, epidemic gonococcal conjunctivitis).

†† In the Australian Capital Territory, Murray Valley encephalitis virus infection and Kunjin virus infection are combined under Murray Valley encephalitis virus infection.

‡‡ Only invasive meningococcal disease is nationally notifiable. However, New South Wales, the Australian Capital Territory and South Australia also report conjunctival cases.

NEC Not elsewhere classified.

NN Not notifiable.

NDP No data provided.

Additional reports

Gonococcal surveillance

(Dr Monica M Lahra, The Prince of Wales Hospital, Randwick, NSW, 2031 for the Australian Gonococcal Surveillance Programme)

The Australian Gonococcal Surveillance Programme (AGSP) reference laboratories in the various states and territories report data on sensitivity to an agreed 'core' group of antimicrobial agents quarterly. The antibiotics currently routinely surveyed are penicillin, ceftriaxone, ciprofloxacin and spectinomycin, all of which are administered as single dose regimens and currently used in Australia to treat gonorrhoea. When in vitro resistance to a recommended agent is demonstrated in 5 per cent or more of isolates from a general population, it is usual to remove that agent from the list of recommended treatment.[1] Additional data are also provided on other antibiotics from time to time. At present all laboratories also test isolates for the presence of high level (plasmid-mediated) resistance to the tetracyclines, known as TRNG. Tetracyclines are however, not a recommended therapy for gonorrhoea in Australia. Comparability of data is achieved by means of a standardised system of testing and a program-specific quality assurance process. Because of the substantial geographic differences in susceptibility patterns in Australia, regional as well as aggregated data are presented. For more information see Commun Dis Intell *2011;35(1):56–57.*

Reporting period 1 January to 31 March 2011

The AGSP laboratories received a total of 1,059 isolates in the 1st quarter of 2011, of which 1,034 underwent susceptibility testing. This number was similar to the 1,056 isolates referred in this period in 2010. Approximately 30% of this total was from New South Wales; 21% from Victoria; 19% from Queensland; 11% from the Northern Territory; 10% from Western Australia; 7% from South Australia and 2% from the Australian Capital Territory. A small number of isolates were also received from Tasmania.

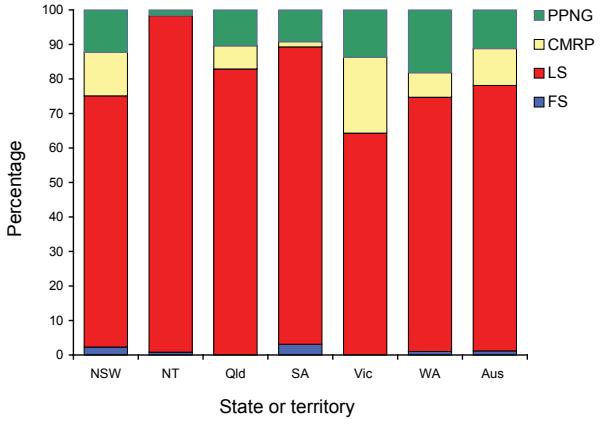
Penicillin

In this quarter, 227 (22%) of all isolates examined were penicillin-resistant by one or more mechanisms. One hundred and sixteen (11%) were penicillinase producing *Neisseria gonorrhoea* (PPNG); and 111 (11%) had chromosomally mediated resistance to penicillin (CMRP). There has been a continuing decrease in the proportion of penicillin-resistant gonococci by any mechanism over the past few years (2010: 32%; 2009: 39%; 2008: 45%; and 2007: 39%). Whilst nationally the proportion of PPNG has remained stable at 11%–13% over the period 2007–2010, the proportion of gonococci with CMRP has decreased in the corresponding quarter from 28% in 2007 to 32% in 2008; 26% in 2009 to 20% in 2010 and to 11% in this quarter of 2011.

The proportion of all strains resistant to the penicillins by any mechanism ranged from 3.3% in the Northern Territory to 36% in Victoria. In Victoria there were 50 CMRP (22%) and 31 PPNG (14%); in New South Wales there were 39 CMRP (13%) and 38 PPNG (13%); in Queensland there were 13 CMRP (7%) and 20 PPNG (10%), and in Western Australia there were 7 CMRP (7%) and 18 PPNG (18%). No CMRP, but 2 PPNG strains were found in the Northern Territory: one acquired in South East Asia (Thailand); and the geographic acquisition of the other was unknown. There was 1 CMRP and no PPNG in the Australian Capital Territory and no CMRP and 1 PPNG reported from Tasmania.

The proportions of gonococci fully sensitive (MIC ≤ 0.03 mg/L); less sensitive (MIC 0.06–0.5 mg/L); CMRP (MIC ≥ 1 mg/L) and PPNG aggregated for Australia and by state and territory are shown in Figure 1. A high proportion of those strains classified as PPNG or CMRP fail to respond to treatment with penicillins (penicillin, amoxicillin, ampicillin) and early generation cephalosporins.

Figure 1: Categorisation of gonococci isolated in Australia, 1 January to 31 March 2011, by penicillin susceptibility and state or territory



FS	Fully sensitive to penicillin, MIC ≤0.03 mg/L.
LS	Less sensitive to penicillin, MIC 0.06–0.5 mg/L.
CMRP	Chromosomally mediated resistant to penicillin, MIC ≥1 mg/L.
PPNG	Penicillinase producing *Neisseria gonorrhoeae*.

Of note, was the marked decrease in penicillin-resistant strains in South Australia in this quarter, to 7 (11%) from 25 (46%) reported in this same quarter in 2010. This decrease comprising 1 CMRP (1.5%) and 6 PPNG (9%), was coupled by an increase in the number and proportion, 56 (86%) of gonococci in the penicillin less sensitive category (MIC range 0.06–0.5 mg/L).

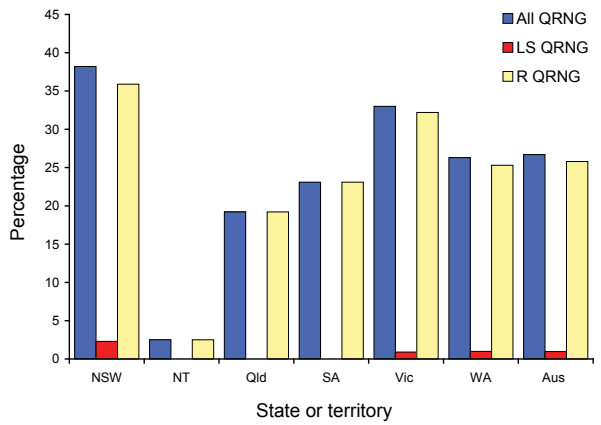
Quinolones

Quinolone resistant *N. gonorrhoeae* (QRNG) are defined as those isolates with an MIC to ciprofloxacin equal to or greater than 0.06 mg/L. QRNG are further subdivided into less sensitive (ciprofloxacin MICs 0.06–0.5 mg/L) or resistant (MIC ≥ 1 mg/L) groups.

There were 276 (27%) QRNG detected in the 1st quarter of 2011. All but 10 had ciprofloxacin MICs of 1 mg/L or more and 189 (68% of QRNG) had ciprofloxacin MICs of 4 mg/L or more. The total number (276) and proportion (27%) of QRNG in this quarter nationally was lower than recent quarters. In the equivalent period in 2010 there were 385 QRNG (38%), 2009 (397 QRNG: 46%) and 2008 (415 QRNG: 35%).

The distribution of quinolone resistant isolates of *N. gonorrhoeae* in Australia by jurisdiction is shown in Figure 2. The highest proportion of QRNG was found in New South Wales where there were 118 QRNG representing 38% of all isolates. There were 75 QRNG isolates (33%) in Victoria; 26 (26%) in Western Australia; 15 (23%) in South Australia and 37 (19%) in Queensland.

Figure 2: The distribution of quinolone resistant isolates of *Neisseria gonorrhoeae* in Australia, 1 January to 31 March 2011, by state or territory



LS QRNG Ciprofloxacin MICs 0.06–0.5 mg/L.
R QRNG Ciprofloxacin MICs ≥ 1 mg/L.

This parallels the decrease in penicillin resistance also noted in all jurisdiction in this quarter, with the exception of Victoria where penicillin resistance remained similar. Three QRNG were detected in the Northern Territory, and one each in the Australian Capital Territory and Tasmania.

Ceftriaxone

Twenty-eight gonococcal isolates (2.7%) with decreased susceptibility to ceftriaxone (MIC range 0.06–0.12 mg/L) were detected nationally, which was less than half of the proportion (6.1%) detected in the same quarter in 2010. There were 14 isolates with decreased susceptibility to ceftriaxone in New South Wales, eight in Victoria, four in Queensland, and one in each of South Australia and the Northern Territory. There were no isolates with decreased susceptibility to ceftriaxone detected in Western Australia, the Australian Capital Territory or Tasmania. The decrease in the proportion of isolates with decreased susceptibility to ceftriaxone (MIC ≥ 0.06 mg/L) corresponds with the decrease in CMRP-resistant gonococci and QRNG also reported in the 1st quarter of 2011. It is possible that the decreased number of isolates with decreased susceptibility to ceftriaxone together with a decrease in CMRP and QRNG reflects a clonal shift from that which was evident in 2010.

Spectinomycin

All isolates were susceptible to this injectable agent. This antibiotic is no longer available in Australia.

Tetracycline

The following data relate to a form of high-level plasmid mediated resistance to the tetracyclines, and gonococcal isolates possessing this plasmid are known as tetracycline resistant N*eisseria* gonorrhoea (TRNG). Nationally, the number (217) and proportion (21%) of TRNG detected in the 1st quarter of 2011 was unchanged from that reported in the same quarter of 2010 (203 TRNG, 20%). TRNG were found in all states and territories, and proportions ranged from 12% in Victoria to 30% of isolates in Western Australia. In the Northern Territory, the number of TRNG approximately doubled in this quarter of 2011 (33 TRNG: 28%) compared with the same quarter in 2010 (16 TRNG: 18%).

Reference

1. Management of sexually transmitted diseases. World Health Organization 1997; Document WHO/GPA/TEM94.1 Rev.1 p 37.

Reporting period 1 April to 30 June 2011

The AGSP laboratories received a total of 1,109 isolates in the 2nd quarter of 2011, an increase from the 1,027 isolates seen in the corresponding period in 2010. Of these, 1,078 (97%) remained viable for susceptibility testing. There were 310 (29%) from New South Wales, 287 (27%) from Victoria, 186 (17%) from Queensland, 136 (13%) from the Northern Territory, 102 (9%) from Western Australia and 36 (3%) from South Australia. There were 20 isolates from the Australian Capital Territory (1.9%) and 1 isolate from Tasmania. The number of isolates examined in the 2nd quarter in Victoria, the Northern Territory, Western Australia and the Australian Capital Territory had increased, and there was a decline in numbers examined in New South Wales, Queensland, and South Australia.

Penicillin

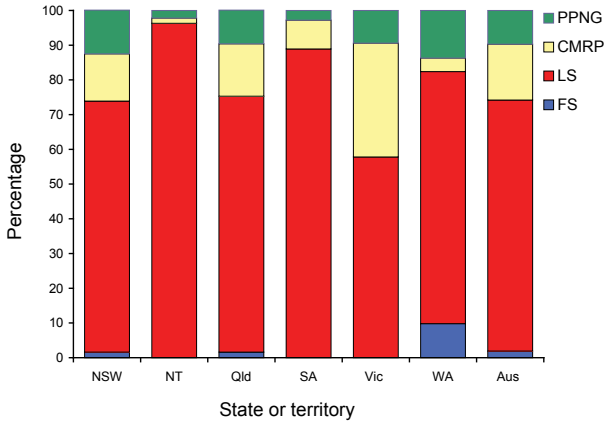
In the 2nd quarter of 2011, 279 isolates (26%) examined were penicillin-resistant by one or more mechanisms, which was proportionally lower than the 30% reported in the same quarter in 2010. One hundred and seventy-four (16%) were chromosomally resistant to penicillin (CMRP), and 105 (10%) were penicillinase-producing *N. gonorrhoeae* (PPNG). In the same quarter in 2010, the proportion of both CMRP and PPNG was higher (19% and 11% respectively).

The proportion of all strains resistant to the penicillins by any mechanism ranged widely across all jurisdictions: Northern Territory 3.7%; South Australia 11%; Western Australia 18%; Queensland 25%; New South Wales 26%; and Victoria 42%. There were 20 isolates from the Australian Capital Territory for this quarter and three were penicillin-resistant. Of note, there was a decline in the proportion of penicillin resistance in all jurisdictions from the same quarter in 2010, with the exception of Queensland and the Northern Territory. Penicillin resistance increased in Queensland from 20% to 25% in 2011 and in the Northern Territory from 2.3% to 3.7% in 2011.

Figure 1 shows the proportion of gonococci fully sensitive (MIC ≤ 0.03 mg/L); less sensitive (MIC 0.06–0.5 mg/L); CMRP (MIC ≥ 1 mg/L), as well as the PPNG data aggregated for Australia, and by state and territory. A high proportion of strains classified as PPNG or CMRP fail to respond to treatment with penicillins (penicillin, amoxycillin, ampicillin) and early generation cephalosporins.

Penicillin resistance due to CMRP predominated in Victoria (CMRP 33%: PPNG 9%); Queensland (CMRP 15%: PPNG 10%); New South Wales (CMRP 14%: PPNG 13%) and South Australia (CMRP 8%: PPNG 3%). However, in Western Australia PPNG were more prominent (PPNG 14%: CMRP 4%). There were 2 CMRP and 3 PPNG detected in the Northern Territory, and in the Australian Capital Territory there was 1 CMRP and 2 PPNG. One PPNG isolate was detected in Tasmania.

Figure 1: Categorisation of gonococci isolated in Australia, 1 April to 30 June 2011, by penicillin susceptibility and state or territory



FS	Fully sensitive to penicillin, MIC ≤0.03 mg/L.
LS	Less sensitive to penicillin, MIC 0.06–0.5 mg/L.
CMRP	Chromosomally mediated resistant to penicillin, MIC ≥1 mg/L.
PPNG	Penicillinase producing *Neisseria gonorrhoeae*.

Quinolones

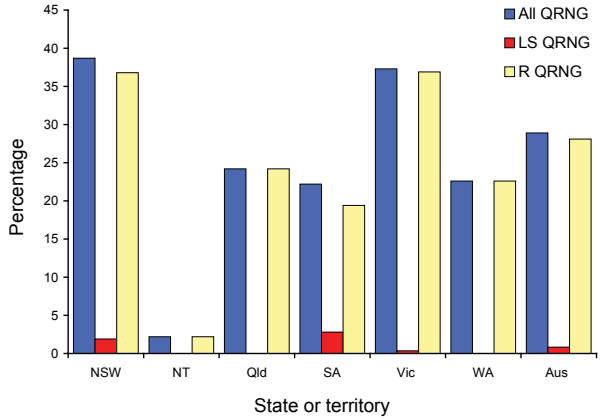
Quinolone-resistant *N. gonorrhoeae* (QRNG) are defined as those isolates with an MIC to ciprofloxacin equal to or greater than 0.06 mg/L. QRNG are further subdivided into less sensitive (ciprofloxacin MICs 0.06–0.5 mg/L) or resistant (MIC ≥ 1 mg/L) groups.

There were a total of 311 (QRNG) in this quarter for 2011, representing 29% of all gonococci tested nationally. The proportion of QRNG continues to decline when compared with the corresponding quarter in 2010: 38%; 2009: 44%; and 2008: 59%. The majority of QRNG in the current period exhibit higher-level resistance (ciprofloxacin MICs ≥ 1 mg/L).

As shown in Figure 2, QRNG were detected in all states and territories with the highest proportions in New South Wales, where there were 120 QRNG (39% of all isolates) and Victoria: 107 QRNG (37% of all isolates) In Queensland there were 45 QRNG (24%); 23 (23%) in Western Australia; and 8 (22% of all isolates) in South Australia. There were 5 QRNG

isolates from the Australian Capital Territory; 3 (2.2%) from the Northern Territory and none reported from Tasmania.

Figure 2: The distribution of quinolone resistant isolates of *Neisseria gonorrhoeae* in Australia, 1 April to 30 June 2011, by state or territory



LS QRNG Ciprofloxacin MICs 0.06–0.5 mg/L.
R QRNG Ciprofloxacin MICs ≥ 1 mg/L.

Ceftriaxone

Thirty-nine gonococcal isolates (3.6%) with decreased susceptibility to ceftriaxone (MIC range 0.06–0.12 mg/L) were detected nationally, a decrease when compared with the same quarter in 2010 (55 isolates: 5.4%). There were 17 (5.5%) isolates with decreased susceptibility to ceftriaxone in New South Wales, 14 (4.9%) in Victoria, 5 (2.7%) in Queensland and 1 isolate (1%) reported from Western Australia. There were 2 isolates in the Australian Capital Territory and no gonococci with decreased susceptibility to ceftriaxone reported from South Australia, the Northern Territory or Tasmania.

Although a decrease in the number and proportion of these gonococci showing decreased susceptibility to ceftriaxone when compared to the same quarter in 2010, there was an increase when compared with the previous quarter in 2011, where 28 (2.7%) had MICs in the range 0.06–0.12 mg/L.

Decreased susceptibility to ceftriaxone (MIC range 0.06–0.12 mg/L) is of increasing concern globally. To better monitor this, the number and proportion of isolates with a raised MIC = 0.03 mg/L are also reported.

In this quarter, data for ceftriaxone MIC = 0.03 mg/L was contributed by all jurisdictions. There were 60 (5.6%) in Victoria; 37 (3.4%) in New South Wales; 13 (1.2%) in Queensland; 3 (0.3%) in South Australia; and 2 (0.2%) in Western Australia. One was reported from the Australian Capital Territory, and none from the Northern Territory or Tasmania.

Spectinomycin

All isolates were susceptible to this injectable agent.

Tetracycline

There were 199 tetracycline resistant *N. gonorrhoeae* (TRNG) detected nationally (19%), which was lower than the number and proportion reported in same quarter in 2010 (218 TRNG: 22%). The highest proportions of TRNG in any jurisdiction were reported from New South Wales: (68 TRNG: 6.3%); the Northern Territory: (40 TRNG: 3.7%); Western Australia: (33 TRNG: 3.1%); Queensland: (28 TRNG: 2.6%) and Victoria (24 TRNG: 2.2%). The number and proportion in the other jurisdictions were South Australia (4 TRNG: 0.4%); the Australian Capital Territory (2 TRNG), and none from Tasmania.

Reference

1. Management of sexually transmitted diseases. World Health Organization 1997; Document WHO/GPA/TEM94.1 Rev.1 p 37.

Meningococcal surveillance

(Dr Monica M Lahra, The Prince of Wales Hospital, Randwick, NSW, 2031 for the Australian Gonococcal Surveillance Programme)

The reference laboratories of the Australian Meningococcal Surveillance Programme report data on the number of cases confirmed by laboratory testing using culture and by non-culture based techniques. Culture positive cases, where Neisseria meningitidis *is grown from a normally sterile site or skin lesions, and non-culture based diagnoses, derived from results of nucleic acid amplification assays (NAA) and serological techniques, are defined as invasive meningococcal disease (IMD) according to Public Health Laboratory Network definitions. Data contained in quarterly reports are restricted to a description of the numbers of cases by jurisdiction and serogroup, where known. Some minor corrections to data in the Table may be made in subsequent reports if additional data are received. A full analysis of laboratory confirmed cases of IMD in each calendar year is contained in the annual reports of the Programme is published in Communicable Diseases Intelligence. For more information see* Commun Dis Intell *2011;35(1):57.*

Laboratory confirmed cases of invasive meningococcal disease for the period 1 April to 30 June 2011 are included in this issue of Communicable Diseases Intelligence *(Table).*

Table: Number of laboratory confirmed cases of invasive meningococcal disease, Australia, 1 April to 30 June 2011, by serogroup and state or territory

State or territory	Year	Serogroup													
		A		B		C		Y		W135		ND		All	
		Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD	Q2	YTD
Australian Capital Territory	11	0	0	3	6	0	0	0	0	0	0	0	0	3	6
	10	0	0	1	1	0	0	0	0	0	0	0	0	1	1
New South Wales	11	0	0	5	15	0	0	2	5	0	1	0	3	7	24
	10	0	0	8	21	2	2	0	0	1	2	1	2	12	27
Northern Territory	11	0	0	0	0	0	0	0	0	0	0	0	0	0	0
	10	0	0	0	0	0	0	0	0	0	0	0	0	0	0
Queensland	11	0	0	12	20	2	3	1	2	0	0	0	0	15	25
	10	0	0	17	23	1	1	0	0	1	1	0	0	19	25
South Australia	11	0	0	1	4	1	1	0	0	1	2	0	0	3	7
	10	0	0	6	10	0	0	0	1	0	0	0	0	6	11
Tasmania	11	0	0	2	2	0	1	0	0	1	2	0	0	3	5
	10	0	0	0	1	0	0	0	0	0	0	0	1	0	2
Victoria	11	0	0	14	24	0	0	0	0	0	0	0	0	14	24
	10	0	0	7	10	0	0	1	2	2	3	0	0	10	15
Western Australia	11	0	0	4	8	0	0	1	1	0	0	0	0	5	9
	10	0	0	2	5	0	1	1	1	0	0	0	0	3	7
Total	11			41	79	3	5	4	8	2	5	0	3	50	100
	10	0	0	41	71	3	4	2	4	4	6	1	3	51	88