

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 program¹ 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) techniques⁶ 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									NS	Total
		<1	1–4	5–9	10–14	15–19	20–24	25–44	45–64	65+		
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

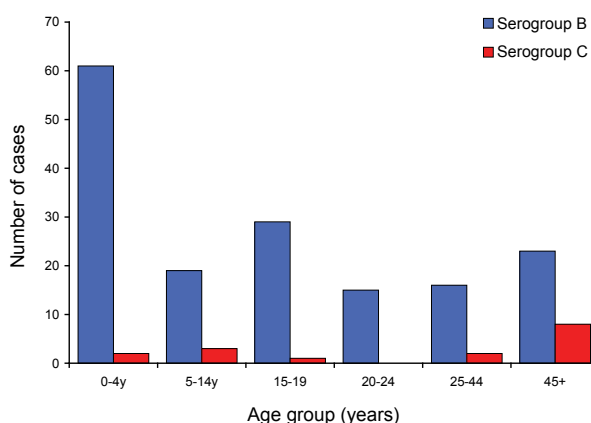
Table 3: A comparison of the number and proportion of serogroup B and serogroup C laboratory-confirmed cases, 2004 to 2010, by known age

Year	Serogroup	Age (years)									
		< 4		5–14		15–19		20–24		25+	
		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

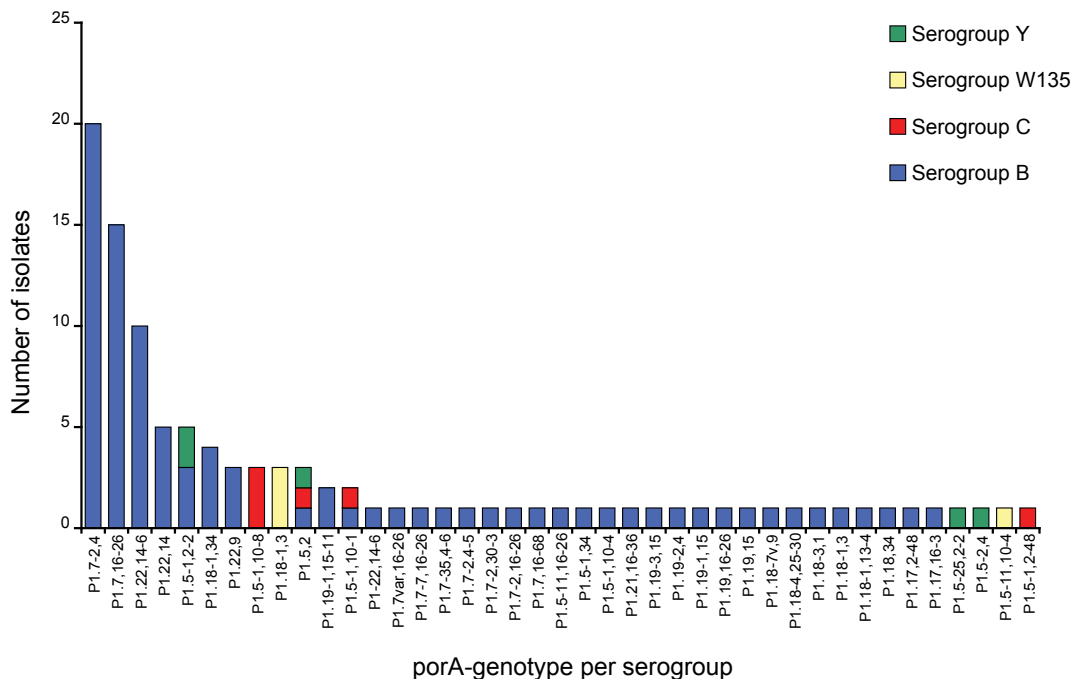
Outcome data for invasive meningococcal disease for laboratory confirmed cases

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	<i>porB</i>	n	<i>porA</i>	n	<i>Fet A</i>	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	<i>porB</i>	n	<i>porA</i>	n	<i>Fet A</i>	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				
				P1.15	1			P1.17,2-48	1	F1-19	1				
				NST	1			P1.18-1,13-4	1	F5-12	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				
								P1.19-1,15-11	1	F5-1	1				
								P1.22,9	1	F5-12	1				
								P1.5-1,2-2	1	F3-3	1				
								P1.5-11,16-26	1	F3-6	1				
								P1.7,16-26	8	F3-3	6				
										F5-5	2				
								P1.7	1	P1.7,16-68	1	F3-3	1		
										P1.7-2,16-26	1	F3-3	1		
								P1.7	2	P1.7-2,4	7	F1-5	6		
								NST	1			F5-9	1		
												F1-5	1		
												P1.7-35,4-6	1	F1-5	1
												P1.7-7,16-26	1	F3-3	1
								ND	9	F1-5	2				
										F3-3	3				
										ND	3				
										F5-1	1				
	C	ND	1	P1.5	1			P1.5-1,10-1	1	F3-6	1				
									P1.5-1,10-8	3	F3-3	1			
				NST	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	<i>porB</i>	n	<i>porA</i>	n	<i>FetA</i>	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

Disease type	Outcome	Serogroup					Total	
		B	C	Y	W135	NG		ND
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 serogroup 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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References

1. National Neisseria Network. Meningococcal isolate surveillance Australia, 1994. *Commun Dis Intell* 1995;19:286–289.
2. Australian Government Department of Health and Ageing. National Notifiable Diseases Surveillance System data for meningococcal disease. Report number 4: Notifications of selected disease by state and territory and year. [online]. Accessed on 14 August 2011. Available from: http://www9.health.gov.au/cda/source/Rpt_4.cfm

3. Australian Meningococcal Surveillance Programme. Annual report of the Australian Meningococcal Surveillance Programme, 2009. *Commun Dis Intell* 2010;34(3):291–301.
4. Public Health Laboratory Network. Meningococcal infection laboratory case definition: Accessed August 2011. Available from: <http://www.health.gov.au/internet/main/publishing.nsf/Content/cda-phlncd-mening.htm>
5. Tapsall J. Antimicrobial testing and applications in the pathogenic *Neisseria*. In: Merlino J, ed. Antimicrobial susceptibility testing: methods and practices with an Australian perspective. Australian Society for Microbiology, Sydney, 2004. pp 175–188.
6. Porritt RJ, Mercer JL, Munro R. Detection and serogroup determination of *Neisseria meningitidis* in CSF by polymerase chain reaction (PCR). *Pathology* 2000;32(1):42–45.
7. Australian Meningococcal Surveillance Programme. Annual report of the Australian Meningococcal Surveillance Programme, 1999. *Commun Dis Intell* 2000;24(7):181–189.
8. Gray SJ, Borrow R, Kaczmarski EB. Meningococcal serology. In: Pollard AJ, Martin MCJ, eds. Meningococcal disease methods and protocols. Humana Press, Totawa, New Jersey, 2001 pp 61–87.
9. Robertson PW, Reinbott P, Duffy Y, Binotto E, Tapsall JW. Confirmation of invasive meningococcal disease by single point estimation of IgM antibody to outer membrane protein of *Neisseria meningitidis*. *Pathology* 2001;33(3):375–378.
10. Lahra MM, Robertson PW, Whybin R, Tapsall JW. Enhanced serological diagnosis of invasive meningococcal disease by determining anti-group C capsule IgM antibody by EIA. *Pathology* 2005;37(3):239–241.
11. The Australian Meningococcal Surveillance Programme. Annual report of the Australian Meningococcal Surveillance Programme, 2002. *Commun Dis Intell* 2003;27(2):196–208.
12. Maiden MC, Ibarz-Pavon AB, Urwin R, Gray SJ, Andrews NJ, Clarke SC, et al. Impact of meningococcal serogroup C conjugate vaccines on carriage and herd immunity. *J Infect Dis* 2008;197(5):737–743.
13. Shultz TR, Tapsall JW, White PA, Newton PJ. An invasive isolate of *Neisseria meningitidis* showing decreased susceptibility to quinolones. *Antimicrob Agents Chemother* 2000;44(4):1116.
14. Singhal S, Purnapatre KP, Kalia V, Dube S, Nair D, Deb M, et al. Ciprofloxacin-resistant *Neisseria meningitidis*, Delhi, India. *Emerg Infect Dis* 2007;13(10):1614–1616.
15. Centers for Disease control and Prevention. Emergence of fluoroquinolone-resistant *Neisseria meningitidis*—Minnesota and North Dakota, 2007–2008. *MMWR Morbid Mortal Wkly Rep* 2008;57(7):173–175.
16. Shultz TR, White PA, Tapsall JW. An *in-vitro* assessment of the further potential for development of quinolone resistance in *Neisseria meningitidis*. *Antimicrob Agent Chemother* 2005;49(5):1753–1760.