Annual report of the Australian Meningococcal Surveillance Programme, 2009

The Australian Meningococcal Surveillance Programme

Abstract

In 2009 there were 233 laboratory-confirmed cases of invasive meningococcal disease (IMD) analysed by the National Neisseria Network, Australia, a nationwide network of reference laboratories. One hundred and thirty–five 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 98 cases were confirmed by non-culture-based methods (92 by nucleic acid amplification testing (NAAT) and six by serology) , and where possible serotyping was determined. Nationally, 194 (83%) laboratory-confirmed cases where a serogroup was determined were infected with serogroup B and 13 (5.6%) serogroup C meningococci. The national total of confirmed cases has remained relatively stable since 2006, but the number of cases may vary between jurisdictions each year. New South Wales had the highest number of recorded cases in 2009. 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 phenotypes circulating in Australia continue to be B:15:P1.7 and B:4:P1.4. Although serogroup C cases were low, phenotype C:2a:P1.5 again predominated in this group. No evidence of meningococcal capsular 'switching' was detected. Approximately two-thirds of all isolates showed decreased susceptibility to the penicillin group of antibiotics (MIC 0.06 to 0.5 mg/L). All isolates remained susceptible to ceftriaxone. Four isolates had reduced susceptibility to ciprofloxacin, and none to rifampicin. *Commun Dis Intell* 2010;34(3):291–302.

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 *N. 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. The NNN annual reports are published in *Communicable Diseases Intelligence*.[2]

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 1–19-year-olds that ran until May 2007) saw a significant and sustained reduction in the number of cases of IMD evident after 2004. 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 *N. meningitidis* isolates circulating locally. This report provides relevant details of cases of IMD confirmed by laboratory testing in Australia in 2009.

Methods

Isolate based invasive meningococcal disease cases

Case confirmation

Case confirmation was based upon isolation of, or positive nucleic acid amplification testing for *N. meningitidis* from a normally sterile site or by

positive serology and defined as IMD according to Public Health Laboratory Network criteria.[3] 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 total number of cases and particularly the number of cases of meningitis, e.g. where there was no lumbar puncture or else where lumbar puncture was delayed and the culture sterile, is underestimated. 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 National Institute for Public Health, The Netherlands. Increasingly, sequencing of products derived from amplification of the porin genes *porA* and *porB* and *FetA* has been used to supplement and supplant 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.[4]

sensitive	MIC ≤ 0.03 mg/L
less sensitive	MIC 0.06 – 0.5 mg/L
relatively resistant	MIC ≥ 1 mg/L

Strains with MIC values which 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 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 nucleic acid amplification testing (NAAT) and occasionally by serological techniques. NAAT testing is essentially by polymerase chain reaction (PCR) techniques[5] 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 Public Health Laboratory Service reference laboratory, United Kingdom, as assessed for Australian conditions.[6–9] 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 233 laboratory-confirmed cases of IMD in 2009 (Table 1) compared with 260 in 2008; 281 in 2007; 271 in 2006; 345 in 2005 and 361 in 2004. In 135 (58%) cases, a positive culture was obtained with or without a positive non-culture-based test and 98 (42%) cases were confirmed by a non-culture-based method alone. The highest number of laboratory-confirmed cases was from New South Wales (82 cases), which has

Table 1: Number of laboratory-confirmed cases of invasive meningococcal disease, Australia, 2009, by serogroup and state or territory

State or territory	Serogroup					Total
	B	C	Y	W135	NG	
ACT	3	0	0	0	0	3
NSW	60	7	4	4	7	82
NT	2	1	0	0	0	3
Qld	54	2	1	0	3	60
SA	21	0	2	0	0	23
Tas	2	0	0	0	0	2
Vic	34	1	1	0	3	39
WA	18	2	1	0	0	21
Australia	194	13	9	4	13	233

NG Non groupable

increased from 62 in 2008 but decreased from 101 in 2007. The total number of all laboratory-confirmed cases decreased in Queensland from 83 in 2008 (and 75 in 2007) to 60 in 2009. There was also a decrease in cases from Victoria (39 cases in 2009), which was lower than the 61 cases in 2008 and 59 cases in 2007. Small or no numerical differences were noted in other jurisdictions.

Seasonality

Forty-five (19%) cases occurred between 1 January and 31 March, 56 (24%) between 1 April and 30 June, 83 (36%) between 1 July and 30 September and 49 (21%) between 1 October and 31 December. A winter peak of meningococcal disease is usual and the above pattern was also present in 2007 and 2008.

Age distribution

Nationally, the peak incidence of meningococcal disease was again in those aged 4 years and under (Table 2). Those aged less than 1 year or in the 1–4 year age group together accounted for 78 (33%) cases of the total) in 2009. There were 94 (36%) cases confirmed in these age groups in 2008 and 100 (36%) in 2007. A secondary disease

Table 2: All laboratory-confirmed cases of invasive meningococcal disease, Australia, 2009, by age, state or territory and serogroups B and C

State or territory	Serogroup	Age group										Total
		<1	1–4	5–9	10–14	15–19	20–24	25–44	45–64	65+	NS	
ACT	B	2	1	0	0	0	0	0	0	0	0	3
	C	0	0	0	0	0	0	0	0	0	0	0
	Total	2	1	0	0	0	0	0	0	0	0	3
NSW	B	10	12	2	2	13	2	6	3	2	8	60
	C	0	1	0	1	1	0	1	1	0	2	7
	Total	10	13	2	3	14	2	7	4	2	10	67
NT	B	1	0	1	0	0	0	0	0	0	0	2
	C	0	0	0	0	0	0	1	0	0	0	1
	Total	1	0	1	0	0	0	1	0	0	0	3
Qld	B	7	13	6	2	7	5	10	4	0	0	54
	C	0	0	1	1	0	0	0	0	0	0	2
	Total	7	13	7	3	7	5	10	4	0	0	56
SA	B	2	3	0	1	9	2	3	1	0	0	21
	C	0	0	0	0	0	0	0	0	0	0	0
	Total	2	3	0	1	9	2	3	1	0	0	21
Tas	B	0	2	0	0	0	0	0	0	0	0	2
	C	0	0	0	0	0	0	0	0	0	0	0
	Total	0	2	0	0	0	0	0	0	0	0	0
Vic	B	4	7	1	2	7	5	5	1	2	0	34
	C	1	0	0	0	0	0	0	0	0	0	1
	Total	5	7	1	2	7	5	5	1	2	0	35
WA	B	4	4	1	3	2	0	2	1	1	0	18
	C	0	0	0	0	0	1	1	0	0	0	2
	Total	4	4	1	3	2	1	3	1	1	0	20
Australia	B	30	42	11	10	38	14	26	10	5	8	194
	C	1	1	1	2	1	1	3	1	0	2	13
	Total B+C	31	43	12	12	39	15	29	11	5	10	207
	Other	3	1	2	2	7	1	4	3	3	0	26
	Total	34	44	14	14	46	16	33	14	8	10	233
	% of all	14.6	18.5	6.0	6.0	19.7	6.9	14.2	6.0	3.4	4.3	

NS Not stated.

Totals include cases due to other serogroups (13) and cases where the serogroup was not determined (13).

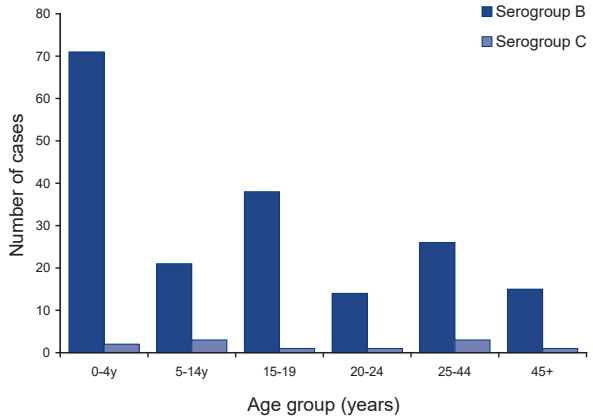
peak is also usual in the adolescent/young adult age group (15–24 years). The total of 46 (20%) of all confirmed cases in those aged 15–9 years in 2009 was a little less than the 50 cases (19%) in 2008 and 56 (20%) cases in this age group in 2007. Those aged 15–24 years accounted for 62 (27%) cases compared with 71 (27%) cases in 2008 and 87 (31%) cases in 2007.

Serogroup data

The serogroup of the meningococci causing disease was determined in 220 of the 233 laboratory-confirmed cases of IMD. Of these 220 cases where a serogroup was determined, 194 (88%) were serogroup B and 13 (5.9%) were serogroup C. This distribution was unchanged from 2008 where 219 (88%) were serogroup B and 15 (6%) were serogroup C and was much the same in 2007, where 223 (85%) were serogroup B and 17 (6.5%) were serogroup C. In 2009, an additional 9 (3.9%) cases were of serogroup Y and 4 (1.7%) of serogroup W135. With the continuing low numbers of serogroup C infections, serogroup B meningococci predominated in all age groups (Figure) and jurisdictional differences in serogroup distribution were not evident. The 13 serogroup C cases of IMD were distributed in 5 jurisdictions: New South Wales (7), Queensland (2), Western Australia (2) Victoria and the Northern Territory (1 each). Four of the 13 cases of serogroup C disease in 2009 were aged 25 years or more; 3 cases were in the 5–14 age group; 2 cases were reported in those aged 4 years or less, a single case in the 15–19 year age group and one in the 20–24 year age group. The age of the patient was not specified for 2 cases.

Table 3 shows a national comparison of the number and proportion of serogroup B and C cases by age from 2004 to 2009. In those aged 14 years or less, there was a decrease in total case numbers and in serogroup B cases in 2009, from 2008. Serogroup C case numbers were low in these age groups across this period. In the 15–19 year age group, the number of serogroup B cases has again decreased, but in the 20-24 years group the proportion of serogroup B cases increased as serogroup C cases declined. In older (25 years or more) age groups in 2009, there was a decrease in the number and proportion of both serogroup B and serogroup C cases when compared with 2008. This reduction follows an increase in the number and proportion of serogroup B cases from 2007 to 2008 when the number and proportion of serogroup C cases was unchanged.

Figure: Number of serogroup B and C cases of invasive meningococcal disease confirmed by all methods, Australia, 2009, by age



Phenotypes of invasive meningococcal isolates

Serogroup B meningococci are typically of heterogeneous phenotypes. In 2009, 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, Western Australia and the Northern Territory (Darwin) isolates. The serogroup B and C serotypes/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 53 isolates were serotyped, 44 were serogroup B and 9 were serogroup C. Of those that were serogroup B, 14 were serogroup B serotype 15, and 11 of the 14 were serosubtype P1.7, which has been circulating in Australia for many years. Nine were serotype 4 and five of these were serosubtype P1.4, which has been circulating in New Zealand at high rates for many years. Nine were non-typeable.

Nine serogroup C strains were phenotyped (including 2 from Queensland) and all were serotype 2a. This phenotype has predominated in serogroup C meningococci in Australia for many years. Of these nine, eight were phenotype C: 2a:P1.5 and 1 strain was non-subtypeable. There is continuing interest in the 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 has been of particular interest. This phenotype has figured prominently in Victorian data in former years. For example, in 2003 there were 29, 21 in 2004, and in 2005 8 serogroup C isolates of this serotype/serosubtype were detected nationally. No isolates with this phenotype were seen in 2009.

Table 3: Comparison of the number and proportion of serogroup B and serogroup C laboratory-confirmed cases, 2004 to 2009, by known age

Year	Serogroup	Age (years)									
		< 4		5–14		15–19		20–24		25+	
		n	%	n	%	n	%	n	%	n	%
2009	B	72	92.2	21	75.0	38	82.6	14	87.5	41	74.5
	C	2	2.6	3	10.7	1	2.2	1	6.3	4	7.3
	All*	77		28		46		16		55	
2008	B	82	89.1	23	95.8	42	91.3	15	83.3	57	85.1
	C	4	4.4	0	0.0	1	2.2	2	11.1	8	11.4
	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	61.3
	C	2	2.0	3	12.0	4	8.2	7	23.0	10	16.1
	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.

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 has been used in an increasing number of jurisdictions in place of serotyping, based on the use of monoclonal antibodies. In 2009, some jurisdictions have moved to the use of genotyping (Victoria, Queensland, South Australia (including Alice Springs) and Tasmania). There was a heterogeneity of typing data across jurisdictions with predominance of a few phenotypes or genotypes as shown in Table 4.

In New South Wales there was a cluster of 3 cases (1 culture-positive and non-phenotypeable and 2 NAAT positive) where genotyping was performed with the results indistinguishable for all 3 cases (Table 4).

Outcome data for invasive meningococcal disease for laboratory-confirmed cases

Outcome data (survived or died) were available for 69 (30%) of the 233 laboratory-confirmed cases (Table 5). Three deaths were recorded in this group (1.3%), all of which were attributable to septicaemia and with serogroup B infection. Outcome data were available for 58 of 194 cases with serogroup B infection. No deaths were recorded for the remainder of 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 had positive PCR and/or serology; those shown as PCR positive were culture negative with or without positive serology. There were 77 diagnoses of meningitis based on cultures or PCR examination of CSF either alone or with a positive blood sample; and 144 from blood samples (cultures or PCR) alone. There were 3 other isolates from synovial fluid and in 2 cases the source of the clinical sample was not disclosed. There were 6 cases that were serologically positive where culture and PCR were negative.

Table 4: Phenotypes (serotype, sero-subtype) and genotypes: *porB* variable region type, *porA* variable region type, and *FetA* type of isolates or DNA extracts from cases of invasive meningococcal disease infection, 2009, by state or territory

State or territory	Serogroup	Phenotype				Genotype					
		Sero-type	n	Sero-subtype	n	*porB*	n	*porA*	n	*FetA*	n
ACT	B	15	1	P1.7	1						
		NT	2	P1.14	1						
				P1.9	1						
NSW	B	15	12	P1.7	9						
				P1.15	1						
				NST	2						
		4	8	P1.4	5						
				P1.15	1						
				P1.14	1						
				NST	1						
		1	3	P1.14	1						
				P1.6,	1						
				NST	1						
		NT	4	NST	4						
	*Cluster	NT	1	NST	1	B,C,7,14b	3	P1.19-1;15-11	3	F5-1	3
		14	1	P1.6.3	1						
	C	2a	5	P1.5	4						
				NST	1						
NT	B	4	1	P1.14	1			P1.22-1,14	1		
	C							P1.5-1,10-8	1		
Qld	B							P1.22,14	6	F1-7	1
										F1-68	1
										F5-5	1
										F1-31	1
										ND	2
								P1.19,13-1	3	F5-1	3
								P1.18-1,34	2	F1-5	2
								P1.22-21,14	2	F5-5	2
								P1.1,16-26	1		

Table 4: Phenotypes (serotype, sero-subtype) and genotypes: *porB* variable region type, *porA* variable region type, and *FetA* type of isolates or DNA extracts from cases of invasive meningococcal disease infection, 2009, by state or territory, *continued*

State or territory	Serogroup	Phenotype				Genotype					
		Sero-type	n	Sero-subtype	n	*porB*	n	*porA*	n	*FetA*	n
Qld, cont'd								P1.19-1,10-8	1		
								P1.19-1,15	1	F5-1	
								P1.22,14-6	1	F1-5	1
								P1.4,16-26	1	ND	
								P1.5-1,10-4	1	F1-5	
								P1.7,16-106	1	F3-3	
								P1.7-2,4		F3-6	1
	C	2a	2	P1.5	2			P1.5-1,10-8	2		
SA	B							P1.7-2,4	9	F1-5	7
								P1.18-1,3	2	F5-1	1
								P1.18-1,34	1	F1-5	1
								P1.19-1, 15-11	1	F5-1	1
								P1.17, 16-93	1	F5-5	1
								P1.7, 16	1	F3-3	1
								P1.22, 14	1	F5-9	1
								P1.5-1,10-8	1		
Tas	B	ND		ND							
Vic	B					A,A,A,Ba	4	P1.7,16-26	4		
						4,D,7,14a	3	P1.7-2,4	3		
						4,C,7,14a	2	P1.18-1,3	2		
						19,Ac,7a,1	2	P1.18-1,34	2		
						19,Ab,7var,Aa	1	P1.17,16-3	1		
						19,Ac,7a,1	2	P1.22,14	2		
						19,Db,7c,14	1	P1.22,14-6	1		
						19,Dvar,7b,14	1	P1.22,14	1		
						4,C,7,14a	1	P1.7-2,13-2	1		
						B,C,7,14b	1	P1.5-1,2-2	1		
						ND	2	P1.19-1,15-11	2		
						ND	2	P1.22,14	2		

Table 4: Phenotypes (serotype, sero-subtype) and genotypes: *porB* variable region type, *porA* variable region type, and *FetA* type of isolates or DNA extracts from cases of invasive meningococcal disease infection, 2009, by state or territory, *continued*

State or territory	Serogroup	Phenotype				Genotype					
		Sero-type	n	Sero-subtype	n	*porB*	n	*porA*	n	*FetA*	n
Vic, cont'd						ND	2	P1.7,16-26	2		
						ND	1	P1.7-2,4	1		
						new,D(var),7b,B(var)	1	P1.22,9	1		
						new,D(var),7c,B(var)	1	P1.18-1,34	1		
	C							P1.5-1,10-8			
WA	B	14	2	P1.7	1						
				NST	1						
		15	1	P1.7	1						
		NT	8	NST	4						
				P1.14	1						
				P1.9	2						
				P1.5	1						
	C	2a	2	P1.5	2						

* Cluster of 3 cases (1 culture positive and non phenotypeable; and 2 NAAT positive) where genotyping was performed.

NT Not typeable

NST Not subtypable

ND Not determined.

Antibiotic susceptibility surveillance of invasive meningococcal isolates

Penicillin

All 135 isolates from culture confirmed cases of IMD in 2009 were available for determination of their susceptibility to penicillin and other antibiotics. Using defined criteria, 91 (67%) isolates were less sensitive to penicillin in the MIC range 0.06 to 0.5 mg/L and the remainder (23%) fully sensitive (MIC 0.03 mg/L or less). The proportion of less sensitive strains is lower than that reported in 2008 (72%) and 2007 (79%).

Other antibiotics

All isolates were fully susceptible to ceftriaxone and by extrapolation to other 3rd generation cephalosporins. Four isolates had altered susceptibility (MIC, 0.06–0.5 mg/L) to ciprofloxacin (MIC, 0.25 mg/L), three from Victoria and one from Western Australia. There were no isolates with altered susceptibility to rifampicin.

Discussion

In 2009, there were 233 cases analysed by the NNN representing a decrease in numbers from previous years. The total number of laboratory-confirmed cases of IMD nationally was relatively stable from 2006 to 2008 (range 260–281) after recording 345 cases in 2005. However; there have been fluctuations in the frequency of detection of cases between jurisdictions over this period with New South Wales recording the highest number of cases in 2009 (82), whereas Queensland recorded the highest number of cases in 2008 (83). There was also a decrease in the number of cases in Victoria from 61 in 2008 to 39 in 2009. These changes in case distribution were essentially attributable to altered numbers of serogroup B cases in 2009 and once again little change was detected in serogroup C numbers. Cultures were obtained from sterile sites in 135 (58%) cases, the lowest number of isolates detected over the duration of the program since it commenced in 1994; however this is proportionally similar to the number of isolates received in recent years: 2008 (149: 57%) 2007 (154: 55%) and 2006

Table 5: Outcome data (survived, died) for laboratory-confirmed cases of invasive meningococcal disease, 2009, by syndrome and serogroup

Disease type	Outcome	Serogroup					Total
		B	C	Y	W135	NG*	
Meningitis	Survived	17	0	1	0	1	19
	Died	0	0	0	0	0	0
	Total	17	0	1	0	1	19
Septicaemia	Survived	38	2	3	2	2	47
	Died	3	0	0	0	0	3
	Total	41	2	3	2	2	50
All cases	Survived	55	2	4	2	3	66
	Died	3	0	0	0	0	3
	Total	58	2	4	2	3	69

NG Not groupable

Table 6: Anatomical source of samples positive for a laboratory-confirmed case of invasive meningococcal disease, Australia, 2009

Specimen type	Isolate of meningococci	PCR positive*	Serology alone	Total
Blood	100	44	–	144
CSF +/– blood	32	45	–	77
Other†	3	3	–	6
Serum/serology	–	–	6	6
Total	135	92	6	233

* Polymerase chain reaction (PCR) positive in the absence of a positive culture.

† Other samples: 3 isolates from joints, 1 PCR from joint and 2 PCR diagnoses from an unknown source.

CSF Cerebrospinal fluid.

(166: 61%). Non-culture-based diagnoses were used to confirm a further 91 (39%) cases of IMD in 2009, compared with 111 (43%) in 2008 and 127 (45%) in 2007. Attention is specifically drawn to earlier Australian Meningococcal Surveillance Programme (AMSP) reports that explain differences between the numbers of clinically notified cases and laboratory-confirmed cases.[10] It should also be noted that surveillance systems rarely capture all cases in any given period so that small differences in numbers of cases should be expected.

Only 13 serogroup C infections were identified nationally in 2009 so that serogroup B disease accounted for 88% of all infections where a serogroup was determined. No serogroup C cases were identified in South Australia, the Australian Capital Territory or Tasmania while there were 7 cases in New South Wales and small numbers present in the other jurisdictions. Only low numbers of infections due to serogroups Y and W135 were encountered, which is usual for Australia. A primary peak in IMD infection rates was again evident in younger age groups, with a secondary peak in adolescents and young adults. The distribution of serogroup C disease was low across all age groups in 2009. 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.[11]

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.[11] Analysis of meningococcal subtypes and any evidence of the expansion of 'new' subtypes will continue as part of the NNN Programme. Mortality data were assessable in only a low proportion of cases and must be interpreted with caution. All of the small number of fatal cases of IMD were associated with serogroup B infections. The NNN does not attempt collection of morbidity data associated with IMD.

The distribution of penicillin MIC values from invasive isolates in 2009 showed that the proportion with decreased susceptibility to penicillins was 67%, a little less than that observed in 2008 (72%) and 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 3rd generation cephalosporins and to the 'clearance' antibiotics rifampicin and ciprofloxacin with the exception of 4 isolates with decreased susceptibility ciprofloxacin: three from Queensland and one from Western Australia. The group of strains with decreased susceptibility to quinolone antibiotics is the subject of on-going international interest following their first description from the AMSP group in 2000.[12–15] There were 2 isolates in AMSP data with decreased susceptibility to quinolone antibiotics detected in 2008, and one in 2007.

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